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PREFACE.

So wide is the range of Modern Practice that an author who would cover it in a single volume, and a reader who would grasp it intelligently, must approach their respective tasks with the aid of *thorough system.* In the following pages an effort has been made to deal with the subject in this manner, from its main divisions, which follow the most rational classification, down to the subordinate paragraphs. Careful use has been made of types of various prominence to facilitate the finding of a topic and the appreciation of its importance. With the vast mass of material, an attempt has been made so to arrange the facts that the reader may grasp the process of reasoning.

Passing from these mechanical details to the characteristics which the author has endeavored to impress upon the book, it may be noted that, as far as possible, the causative pathology has been blended with the consecutive clinical features of disease, that reasons have been given for facts, that exceptions have been subordinated to what is usually found at the bedside, and that the allurements of typical clinical pictures and dogmatic generalizations have been avoided because they hold neither in practice nor at the postmortem table.

The author also conceives that a book on Practice should be *well-directed*, which implies that it should deal adequately with scientific theories and princip[illegible] that it should recognize that the final object of its existence is th[illegible] of knowledge *to the cure or alleviation of disease.* Accordi[illegible] amount of space has been devoted [illegible]ment, to the [illegible]ation of drugs and to numerous [illegible] prescrip[illegible] the student to use or improve [illegible] physiological [illegible] has been [illegible] upon care- [illegible] the writer [illegible] symp[illegible] disease are [illegible] those [illegible] hibit[illegible] ure.

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A TREATISE

ON THE

PRINCIPLES AND PRACTICE OF MEDICINE.

BY

ARTHUR R. EDWARDS, A.M., M.D.,

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Passing from these mechanical details to the characteristics which the author has endeavored to impress upon the book, it may be noted that, as far as possible, the causative pathology has been blended with the consecutive clinical features of disease, that reasons have been given for facts, that exceptions have been subordinated to what is usually found at the bedside, and that the allurements of typical clinical pictures and dogmatic generalizations have been avoided because they hold neither in practice nor at the postmortem table.

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The reader will also find a large number of tables giving the differential diagnosis of diseases likely to be confused, or in many instances, of entire subjects, such as those of the liver and kidney, the chief eruptive diseases and those of the typhoid group. They are practical though necessarily schematic. Nowadays the border-lines of surgery and medicine overlap, and the practitioner, internist and surgeon must

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be familiar with the province of each other. For this reason, as fa the scope of the treatise admits, surgical indications and results introduced.

The writer wishes to acknowledge the extremely careful atte bestowed by the Publishers upon every literary and typograp detail, and the assistance of Dr. F. G. Dyas and Dr. Milton Ma in the proof reading.

A. R.

CHICAGO, 1907.

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SECTION I.

THE SPECIFIC INFECTIONS.

TYPHOID FEVER.

The name (*Typhos*, *Stupor*) refers to the clouded mentality, and dates from Hippocrates. The disease was first recognized by Louis of Paris (1829), and was first described as a separate disease by Gerhart and Pennock (1837).

Definition.—Typhoid fever is a general infection, characterized—

1. *Etiologically* by the Bacillus typhosus.
2. *Anatomically* by hyperplasia and ulceration of the lymphatic structures of the intestine, hyperplasia of the spleen and mesenteric glands, and by parenchymatous change in other organs.
3. *Clinically* by a characteristic fever, roseolous eruption, enlargement of the spleen, slow pulse, a peculiar serum reaction, typhoid bacilli in the blood, and often by intestinal symptoms.

Etiology.—The *typhoid bacillus* was first seen by Eberth and Koch (1880), and first cultivated by Gaffky (1884).

1. **Characters.**—It has short, thick, rounded ends, is three times as long as wide, in length is one-third the diameter of a red blood-corpuscle, and its numbers are usually, but not always, proportionate to the degree of infection. It is polymorphous, sometimes thread-like, varies in different media, contains no spores (though it is maintained that a spore is found at one pole), is flagellated, and actively motile. It grows best at body heat, and is a facultative anaërobe growing on various solid and fluid media, especially on agar, gelatin, and, characteristically, on the potato. It does not produce acid, ferment lactose, produce indol, or coagulate milk. It stains with aniline dyes, but not by Gram's method; is not inoculable, but produces the typhotoxin.

Until recently, animal inoculations have been negative. It is sai that typical lesions have been produced in the chimpanzee.

2. **Location.**—It is found in the ulcers, in the lymphatics of the mesen tery, in the spleen, in the intestines at autopsy (chiefly in the uppe small intestine), in the fæces, in the blood, in the internal viscera, i the urine, in the roseolæ, less frequently in the lungs, muscles, uterus œsophagus and tongue, and in various secondary foci, as in pleuris pneumonia (sputum), endocarditis, meningitis, parotitis, osteomyeliti and abscesses. According to Lynch, it has also been isolated sixtee times from the blood of the fœtus in maternal typhoid infections. I curious cases typhoid bacilli may occur in the stools of individuals, par ticularly children, not suffering from typhoid fever.

3. **Tenacity.**—Its tenacity of life is great, the germ persisting for year in bone lesions and enduring from three weeks to three months afte death. It lives below zero, from two to twenty-two weeks. It resist drying, except in thin layers, for months, and lives some time in wate seldom longer than three weeks, although its multiplication is pre vented by saprophytes. It is killed in a few hours by direct sunligh The germ may live for months in ice, sour milk, fæces, or in the up per, but not in the deeper, layers of the soil. It may live a year in soile clothes, which explains contagion in washerwomen and nurses.

4. **Entrance.**—It resists gastric juice fairly well, yet its chief an probably sole *atrium* is the digestive tract, the infection being carrie in water (its main medium), milk, butter, ice, vegetables, or oyster As shown in the Spanish-American and Boer wars, dried fæces con taining the bacilli may be carried by flies and sand. Alice Hamilto found the bacilli in flies in the Hull House region of Chicago. Cock roaches and other insects may also convey infection.

5. **Exit.**—The *germ leaves the body* chiefly in the fæces and urin which are dangerous even far into convalescence, and which are th chief means of dissemination, the disease being less often conveye directly from one individual to another, although direct infection occu in hospitals and camps. Lenz found that certain persons after typhoid attack become "chronic carriers of typhoid bacilli," the bacil remaining in their dejecta for one or even fifteen years.

From a practical standpoint, the direct etiological diagnosis is somewhat limited value, since (*a*) differentiation from the colon bacill requires an expert bacteriologist; (*b*) the bacillus does not appe early nor invariably in the stools, where the diagnosis is difficult; an (*c*) since direct splenic puncture for diagnosis alone is no more justi able than in malaria. The bacillus itself is found in the blood ear in the first week. In the second week the serum usually gives th distinctive Widal reaction.

Predisposing Etiology.—1. **Reduced pyhsiological resistance,** from overwork or mental depression, is a frequent predisposing caus yet typhoid very often occurs in the robust. It is claimed that th weak or emaciated are immune,—*e. g.*, those with syphilis, tuberc losis, cancer, endocarditis, anæmia, or convalescents from other di eases. Some immunity is claimed in pregnancy, lactation, and the puerperium. Alimentary affections, according to Bouchard, such as decreased hydrochloric acid formation, or dilated stomach, favor infection. Most cases occur in time of war, or among the poorer classes, or among recent residents in typhoid localities.

2. **Season.**—For reasons not known most cases develop in the late summer and fall ("autumnal fever").

3. **Age.**—The years from fifteen to twenty-five include most cases (56 per cent.), yet fœtal cases are recorded, and a few cases occur in the first year of life. Their number increases from the first to the fifth year. Between the fifth and fifteenth years as many are affected as after thirty-five. The disease is relatively rare after fifty.

4. **Geographical Distribution.**—Typhoid is endemic rather than epidemic. Although it is the most common continued fever in temperate climates, it is a world-wide disease and sustains a most intimate relation to the water supply, the disposal of sewage, the density of population, and personal hygiene.

Immunity.—Congenital immunity is not proved. Immunity has no relation to the severity of the disease, though it is usually conferred after one attack, but is not life-long nor as frequent as in the case of scarlatina. In the Hamburg epidemic (1887), $2\frac{4}{10}$ per cent. of the cases were second attacks; and in Eichhorst's series there were over 4 per cent. of second attacks. These second attacks equal or even exceed the original in severity.

Symptoms.—The general clinical picture offers more variability than that of any other infection. Typhoid is not an intestinal disease, but a general infection. Its forms are:

1. The typical enteric, frequently with diarrhœa, tympany, hemorrhage, and perforation.
2. The septicæmic, without any, or with very slight, anatomical intestinal lesions, though responding to the Widal test and accompanied by bacillæmia.
3. Other localizations than enteric; *e. g.*, in the lung, larynx, bone, etc.
4. Mixed infections—malaria, streptococcus, colon bacillus, etc.
5. Paratyphoid, with close clinical resemblance to typhoid but with organisms differing culturally and in their agglutinating reactions. (See DIAGNOSIS).

The *incubation* lasts one to two weeks, with symptoms more vague than in other acute infections,—*e. g.*, depression, pains in the head, back, or limbs, dulness of hearing, disturbed sleep, or perhaps chilliness, night-sweats, anorexia, epigastric oppression, constipation, or even diarrhœa. The incubation lasted only three days in a girl who took a pure typhoid culture with suicidal intent.

The *first week* corresponds anatomically to the intestinal catarrh and the beginning of medullary infiltration of Peyer's plaques. The disease dates from the fever, which each evening is from 1° to $1\frac{1}{2}$° higher than on the previous day, until a temperature of 103° to 104° is reached. The pulse is full, dicrotic, and rapid, but not high in proportion to the fever. The tongue is coated white. Thirst, dry cough, enlarged spleen, eruption of rose spots, and typhoid bacilli in the blood, are found. The abdomen shows slight distention and tenderness over the ileocæcal region or epigastrium. Delirium is infrequent, and headache, constipation, dulness and apathy are usually present.

The *second week* corresponds to the end of medullary infiltration and the beginning of eschar-formation. The fever is higher and usually continuous. The morning remission is less. The pulse is full, faster, and less dicrotic. The blood responds to the Widal test. The tongue is dry and glazed, as are also the lips, pharynx, and mouth, to which the mucus adheres. The voice is weak. The bronchitis increases, and the urine is febrile. Abdominal symptoms are often aggravated. Meteorism appears. Yellow pea-soup dejecta, which are involuntary in severe cases, occur. The face is apathetic, its lines obliterated, the mouth half open, and the upper lip retracted, showing sordes. Roseolæ usually develop during this week. Nervous symptoms increase with higher fever; delirium follows; the patient becomes less querulous; and euphoria from narcosis by the toxins is the rule, while pain suggests complications. The patient may begin to recover, or may die at this stage from hemorrhage or nervous toxæmia.

The *third week* is the stage of ulceration. The fever gradually declines with marked morning remissions and unaccountable variations. The pulse is smaller, and faster (100 to 120), and loses its dicrotism; it is sometimes slower. The tongue clears and the roseolæ disappear, being replaced by miliaria from sweating. In exceptional cases diarrhœa may be seen for the first time in this week. Emaciation is marked. When the fever falls, the patient complains of hunger, weakness, and pain. Death may occur in the typhoid state from heart weakness, pulmonary inflammation, paralysis of the nerve-centres, hemorrhage, or perforation.

The *fourth week* (cicatrization) usually marks convalescence, as the average duration of typhoid is from three to six weeks; though in severe cases the symptoms assigned to the third week may continue. Convalescence may be uneventful, or marred by slight temperature, rapid pulse, palpitation, nausea, vomiting, muscular weakness, exaggerated reflexes, etc.

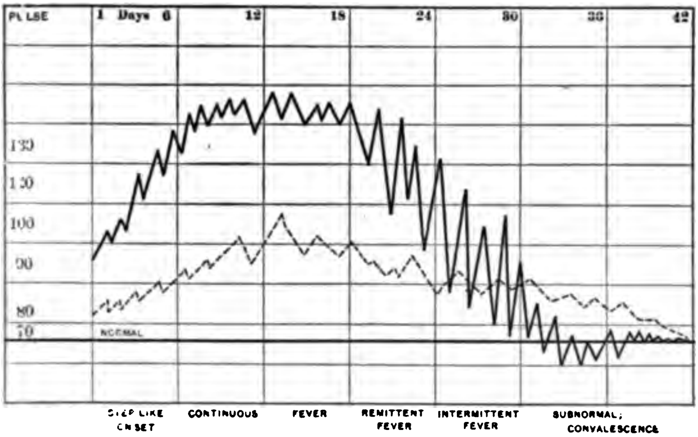
Symptoms in Detail.—To avoid repetition, and to explain anatomically the clinical signs and symptoms, the typhoid pathology will be incorporated under the appropriate organs, and complications and sequels will be classified with the more common typhoid manifestations.

1. **Fever.**—Fever is an expression of toxæmia, and is a cardinal symptom of typhoid. It is subacute. It lasts more than fourteen days (averaging three to four weeks), usually comes on without chill, and resolves by lysis. It is the best studied chapter in the disease since the time of Wunderlich, who in dubious cases diagnosticated

typhoid from the fever chart alone. In its incipiency there is usually a gradual rise for three to five days. Wunderlich placed great stress upon this, though the rise is sometimes sudden or accompanied by a chill. Chomel (1834) described cases with sudden onset and De Mussy (1884) spoke of an explosive onset; a sudden onset is observed in 10 per cent. of typhoid cases. The writer saw, with Dr. Korssell, a child whose temperature rose suddenly to 106° on the second day.

In the *first week*, corresponding to the catarrh and medullary infiltration, the evening temperature is $\frac{1}{10}$° to 1° more than in the morning, and the ascension is ladder-like. When there is an initial chill, every other possible disease should first be excluded, yet Osler observed chills in 24 per cent. of his cases. Chills occur: (*a*) at the onset, especially in children, and in light forms; (*b*) throughout the course, and accompanied by sweats, the *sudoral type* of the French; (*c*) in complications—pneumonia, pleurisy, otitis media; (*d*) after antipyretics, tubbing, or (*e*) in defervescence, sometimes from secondary sepsis.

Fig. 1.



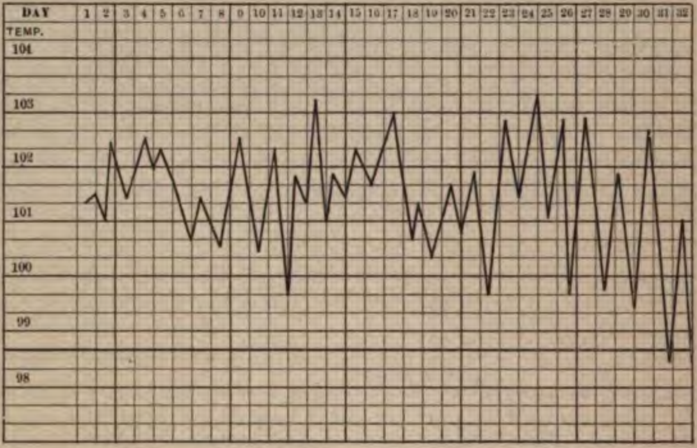
Typical typhoid-fever curve.—The heavy line marks the temperature curve, and the broken line the pulse curve.

In the *second week*, corresponding to the end of infiltration and to the formation of the slough, the fever is continuous from one-half to three weeks, Curschmann distinguishing two types: (i) one and one-half weeks fastigium, and (ii) three weeks fastigium. Especially high temperature suggests complications; if the fever becomes very high, the course is apt to be severe. Sometimes a pseudo-collapse may occur, particularly in subjects between twenty-five and thirty years of age.

In the *third week* (ulceration), the fever is remittent, the amphibolic stage of Wunderlich, and the *stadium hecticum* of Traube. The cause of fever variations is not known.

The *fourth week* (cicatrization) is the stage of "steep curves," lasting from three to eight days. When a sudden drop occurs, recurrences and relapses are not infrequent. A critical fall of the temperature sometimes follows. Jaccoud observed a brusque defervescence in 29 per cent. of his cases. The fever may be intermittent or remittent throughout the disease, especially in children, the aged, and in severe cases. Irregularity and variations, especially when protracted, cause difficulty in diagnosis, suggesting, perhaps, miliary tuberculosis or sepsis.

FIG. 2.



Typhoid fever; irregular fever curve throughout the course.'

Convalescence.—In its first week the fever-curve is usually subnormal; when not so, some irregularity must be looked for. Later there is a gradual rise to normal.

In the *typus inversus*, seen chiefly in children and the aged, the morning temperature is higher than the evening record. Afebrile typhoid is exceptional. Modification of temperature may be caused by visits from friends, excitement, entrance to the hospital, diet, hemorrhage, abortion, peritonitis, and other complications.

2. The Splenic Tumor.—The splenic tumor is a cardinal symptom occuring early and frequently, persisting in exacerbations and relapses. In importance and size, it ranks second to the splenic enlargement of malaria and sepsis. The cause of this tumor is the bacillus or its toxins (not the fever) which produce splenic hyperæmia and hyperplasia—changes closely analogous to those in the intestinal lymph-structures. Massive endothelial proliferation is said by Mallory to occur in the splenic, lymphatic, and medullary vessels. In the first week the spleen is hyperæmic and swollen to two or even three times its normal size. Its markings are indistinct. In the second week it is more pulpy and darker. By the fourth week it has usually disappeared, but as long as we find the splenic tumor, the disease has not ended.

Frequency.—(*a*) Anatomically the splenic tumor is present in 98.4 per cent. of the cases. Its rare absence is explained by thickening of the capsule, old induration or infarction of the organ, or extreme emaciation. (*b*) Clinically, it is present in 75 to 80 per cent. (Curschmann) or 90 per cent. (Leube). The percentage varies with the skill of the observer, the method of examination, the epidemic, and age of the subject (the sign being often absent in children, in adults older than forty-five years, or after hemorrhage). It should be palpated with the patient in the right diagonal position. This is difficult in pain, tympany, and in an anomalous position of the colon. The author has twice seen the spleen ruptured by deep or rough palpation. Percussion of the spleen is worthless. It should be remembered that the spleen may be ectopic. The examiner should stand or sit on the right side of the patient, palpating with the right hand pressing evenly on the abdomen, and the left hand hooked beneath and lifting up the lower left ribs. Relatively rare splenic complications are abscess, infarcts and rupture.

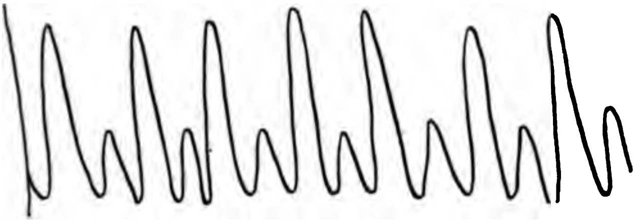
3. Dermal Symptoms.—(*a*) ROSE SPOTS (roseola typhosa, *tâche rosée lenticulaire* of Louis) constitute the third cardinal symptom. They are round spots, varying from the size of a pin-head to that of a lentil (lenticular), circumscribed, rose-colored, slightly elevated to the touch, purely hyperæmic, and therefore disappearing on pressure and reappearing on its release, not often fusing, and later becoming darker, but leaving no mark, with sometimes a vesicle at their apex, and rarely desquamating, except in a few infantile or juvenile cases. They are rarely hemorrhagic, and therefore the commonly used term, petechia, is inappropriate. Being purely hyperæmic, they are not found at autopsy. They scarcely ever pustulate. They may be seen as early as the fourth day, especially in women and children, but usually appear from the seventh to the tenth day. They occur in crops, during two weeks, each crop lasting from three to five days. Occasionally, they outlive the fever. Their location is on the lower breast and abdomen, though they sometimes appear twenty-four hours earlier on the back, because of its warmth. They rarely occur on the neck or face, and generally are more frequent on the proximal than on the distal parts of the extremities. If there are many on the lower parts of the limbs, they are probably not roseolæ. They number five to ten, or even thirty. They are more abundant in women, less so in children and the aged, and seldom so abundant as to resemble measles closely. Their frequency varies somewhat with the epidemic. Eichhorst found them in more than 1,200 cases, Leube in 80 per cent., Osler in 93 per cent., and Murchison in 33 per cent.

Typhoid bacilli have been found in the roseolæ in 50 to 80 per cent. of the cases and may establish a doubtful diagnosis, but, of course, rarely an early one; Polacco found the bacilli in 100 per cent. and Krause in 94 per cent. of their cases. In other diseases, as miliary tuberculosis, meningitis, tuberculosis, and pneumonia, they are very rarely encountered.

(*b*) OTHER CUTANEOUS MANIFESTATIONS.—Other cutaneous manifestations are: urticaria; erythema, even of the nodose or exudativ forms; pelioma typhosum, or the *tâche bleuâtre*—pale blue spots measuring 4 to 10 millimeters, due to pediculosis, and therefore seen in part nearest the pubic or axillary hair, etc.; erysipelas, phlegmon and furuncles (increased by hydrotherapy); alopecia after the typhoid attack rarely followed by permanent baldness. The typhoid odor is must or semi-cadaveric. Miliaria are frequent, though less than in sepsi and more common than in measles or scarlatina. They usually appea late in the third or fourth week. Herpes is rare, occurring in less tha 1 per cent. of the cases. The author has observed it only ten times i upwards of 1,800 cases. It is far less frequent than in pneumoni malaria, typhus, meningitis, and influenza. Decubitus occurs in 1 pe cent., mostly in severe and hospital cases. Noma is a rare complication. In 204 cases of gangrene collected by Keen, 50 per cent. occurre in the leg, 25 per cent. in the face, neck, and trunk, and the balanc in other locations. McFarland and Hare have reported a number c cases in Philadelphia. Sweats and chills occur in the "typhus sudoralis," especially in the latter half of the fever course, described i Naples by Borelli and in Paris by Jaccoud. Œdema is due mos frequently to phlebitis, sometimes to anæmia, and rarely to nephriti The skin sometimes shows atrophic striæ like those of pregnanc Desquamation may occur, particularly, in children.

4. Circulation.—(*a*) THE SLOW PULSE.—The slow pulse is the fourt cardinal symptom. In uncomplicated cases it follows the temperatur in the early stages, but is lower than the fever would justify, especiall in men under forty years; *e.g.*, pulse 90 with the fever 103°. It is rathe faster in women, in children, and in the nervous. Its slowness is du to an inhibitory action of the toxins on the medulla or the heart, a fa not recognized either by the older or by some of the modern writer The author has seen four typhoid cases in which the pulse ranged fro fifty-six to sixty-four, with the fever above 103° and the respiratio above forty. Slowness is of good import. A fast pulse early in th typhoid course is a poor prognostic, especially in men. Irregularit or crossing of the pulse and temperature-curves, is also ominou The pulse may become faster from heart or lung inflammation, perfor tive peritonitis, hemorrhage, and severe intoxication. It may al run up on visiting days, in women, children, emaciated and nervou individuals, or in the stage of great temperature variations. At first little tense in volume, it soon becomes *dicrotic*, which, with slownes suggests typhoid. Dicrotism, evidenced by a double shock, occurs i 80 per cent. of cases, but is not prognostic. It is usual in adults, absent in children and often in adults at the fastigium in very seve cases. Since it indicates lack of arterial tone, it is also absent in arteri sclerosis. In Osler's Clinic, the blood-pressure was found to be 115 125 millimeters of mercury in the systole, and 85 to 100 millimeters i diastole. In the first stage of convalescence, when there is subnorm temperature, the pulse is usually normal in rate, or slow, which is e plained by disturbed conductivity in the heart muscle. Myocarditis is possibly the cause in some cases. In the second stage it is usually higher than normal, especially after the patient gets out of bed, after eating, or a movement of the bowels. On the whole, bradycardia in convalescence is more common than tachycardia.

FIG. 3.



Pulse tracing in typhoid, showing dicrotism.

(*b*) **HEART MUSCLE.**—In the *heart muscle* early changes are rare. Parenchymatous degeneration, evidenced by albuminous granules, nuclear swelling, and pigmentation, are found later, the heart as a whole being flabby, soft, pale, and even waxy. Fatty and hyaline degeneration, vacuole formation, segmentary myocarditis, and a tendency toward muscle regeneration, are the usual findings in fatal cases. *Interstitial myocarditis*, like that of diphtheria or scarlatina, occurs in over 50 per cent. of typhoid cases (Rhomberg). It usually regresses without functional or organic disturbance, and chronic myocarditis rarely develops. Its clinical signs may appear at the end of the second week, and last into the fourth week, or it may develop first in convalescence. The symptoms are gradual dilatation, weak tones (especially the first tone at the apex), accentuation of the second pulmonic, a systolic bruit, and a fast, irregular pulse with lessened tension but without dicrotism. Tachycardia, without myocarditis, may result from the toxins acting on the medulla oblongata. Recovery is usual, even if the myocarditis lasts two or three months.

Dilatation of the right heart may be observed in severe cases. Less frequent are the following complications: Cardiac thrombosis and embolism; pericarditis ([one in 1,000 autopsies], mostly fibrinous, and mostly in complicating sepsis); endocarditis ([one in 1,000], mostly mural over myocarditic patches), sometimes due to Eberth's bacillus; collapse, with its usual signs, which may cause death from heart degeneration, or from toxic vasomotor paralysis (as in diphtheria, pneumonia, or pyocyaneus infections). Myocarditis is the most common cause of collapse, while pulmonary embolism, uræmia, cerebral hemorrhage, etc., are far less common. Sometimes no clear cause is discovered at autopsy. Collapse develops mostly in strong men (80 per cent. of Dewèvre's 140 cases) and most often during convalescence. (See PROGNOSIS.)

(*c*) **VESSELS.**—In the vessels, arteritis typhosa, most often found in the femoral artery, is rare, W. W. Keen collecting only forty-four cases. It is a specific inflammation caused by the typhoid bacillus.

Spontaneous gangrene may result. Arterial embolism from maranti thrombi is rare. Phlebitis typhosa occurs in 1 per cent. (to 2.6 per cent Thayer) of the cases, being considered marantic or infective (Eberth' bacillus or pus cocci), when pain and fever are present; it occur chiefly in males, is unilateral and is usually left-sided, since the le vein has a slower current, being crossed by the right iliac arter (Liebermeister). The symptoms are pain, coldness, œdema, and sometimes a tender, palpable cord, which should be manipulated mos carefully, if at all, lest embolism be caused. Thrombosis usually occur in the saphenous vein where it empties into the femoral, sometime in the popliteal vein, and rarely in the arms. It may extend from th leg to the cava, when the other leg may be involved. There is som danger of detachment, pulmonary embolism, and sudden death; bu relative recovery is usual after two or three months.

(*d*) THE BLOOD.—The *red blood-cells* average 4,000,000. Thei reduction is always slow, and anæmia is especially frequent du ing the third week or in relapses. An apparent increase of the re disks, analogous to that observed in cholera, may result from loss c fluids, as from diarrhœa (Hayem). The hæmoglobin decreases prc portionately to the red cells, or slightly in excess of the oligocythæmi especially in convalescence. Marked post-febrile anæmia is not common. The *leukocytes* decrease from 8,000 to 3,000, or even less, bu average 5,000 per cubic millimeter. Occasionally, large cells containin numbers of red corpuscles are seen. Leukopenia is more common ir typhoid than in any other febrile condition and helps to differentiat it from pneumonia, sepsis, meningitis, etc. It is possibly due to destruction of the lymphatic elements by the endothelial phagocytes. Increase of the white cells occurs only in complicating inflammations o in convalescence. The polymorphonuclear neutrophiles are absolutel and relatively decreased to below 50 or 60 per cent., while they ar increased in other infections. The large mononuclear and transitiona forms are relatively increased. The eosinophiles are greatly decreased their return is a good prognostic (Türck).

The Gruber-Widal serum test is based on Pfeiffer's experiments o immunity in cholera (1884). Pfeiffer and Kolle not only produce immunity, but found that bacteriological inoculations in animals became granular and gradually dissolved. Metschnikoff and Borde found the same reaction in the test-tube with the blood-serum of animals. The "agglutination" and "immobilization" of typhoid bacill brought in contact with a typhoid serum, was found by Durham an Gruber, and also independently by Pfeiffer and Kolle. Gruber pointe out the value of the test in proving the existence of a previous typhoid while Widal applied the laboratory facts at the bedside, and found th reaction during the disease. The agglutinating substances may pas from the maternal blood to that of the fœtus, or from the mother's mill to the infant's blood; they may be obtained from the serum of a bliste and sometimes from the urine, saliva, semen, tears, or cerebrospinal fluid

TECHNIQUE.—A bouillon culture, not more than twelve to eightee hours old, is used, taking forty parts of the bouillon and one of the blood (dilution of 1 to 40). The test may be made macroscopically in the test-tube, or on the microscope slide, where immobilization, clumping, agglutination, and final solution of the bacilli, may be observed, sometimes in a very short time—an hour is the usual time-limit for the test. Ficker and Ruediger have recently employed dead cultures with equally accurate results, and this method is certainly more convenient, safe, and capable of wider use than the use of living bacilli, especially in country practice. (Plate I.)

Significance.—The Gruber-Widal reaction may be found thirty years after a typhoid attack. Occasionally it is found in rheumatism, sepsis, miliary tuberculosis, meningitis, pneumonia, influenza, endocarditis, or icterus, and it is sometimes absent in typhoid. While present in 95 to 97 per cent. of typhoid cases, it often develops late; *e. g.*, on the seventeenth or the fortieth day. Cabot found the reaction in 97 per cent. of 5,978 collected typhoid cases. In Osler's series it appeared in 93 per cent. before the eighth day. When it appears in the first week, it is due to a "silently developing" infection (Widal); *i. e.*, the typhoid is more advanced than we appreciate. Leube considers the test significant only when it is absent in the early stages and develops later, care being necessary in all other cases whether the reaction be positive or negative. The author has long since ceased to exclude typhoid absolutely when no reaction is obtained, and he maintains the importance of the classic symptom-complex even without the serum reaction. Undue importance is sometimes attached to this as well as to many other laboratory tests, invaluable though they may be in connection with the physical findings. In severe typhoid the test may be absent, when early cultures (in 85 to 94 per cent. of the cases) may reveal typhoid bacilli in the blood. Kasel and Mann believe the reaction is not one of infection, but one of immunity, agglutination becoming most marked toward the end of the disease.

Typhoid bacilli have been *cultivated from the blood* in a large percentage of cases (even 88 and 94 per cent.), and undoubtedly are present in the blood of every case. In an analysis of 535 cases, Rosenberger found bacilli reported in 80 per cent.

5. Nervous Symptoms.—Typhoid was once called "nervous fever" (*febris nervosa, nervenfieber*). The nervous system may be conspicuously involved even in the incubation stage.

(*a*) HEADACHE.—Headache is almost invariable, being most often frontal or occipital, constricting, throbbing, or a pressure sensation. It usually disappears in the second week, and, if it lasts or develops late, it may indicate meningitis. Insomnia may be both early and lasting. Stupor and sleep must be distinguished from one another, since a patient may be stuporous yet sleepless.

(*b*) THE TYPHOID STATE.—The typhoid state begins in the second week (*v. s.*) with apathy and delirium, which is usually mild. The patient may attempt to get out of bed, and, in alcoholics, delirium tremens may be simulated or actually develop. In mild cases delirium

lasts into the third week. In very irritable subjects, as in women a
children, or in overwhelming intoxication, we may have photophobi
stupor, coma, loud breathing with an open mouth, absence of t
pharyngeal reflex, involuntaries, waving of the hands (floctitation
leaping of the tendons (*subsultus tendinum*), picking of the bedcloth
(*carphologia*), trismus, contractures, grinding of the teeth, rigid nec
and convulsions (in children especially), which symptoms are not d
to the fever, since they all may occur with low temperature, but
toxæmia. In coma-vigil the patient is semi-comatose subjectively, b
the open, unseeing eyes give a "watchful" appearance objectivel
Anatomically a "wet-brain" is found. Murchison observed convu
sions but six times in 2,960 cases. They may develop in children,
severe intoxication, or from such cerebral complications as meningiti
encephalitis, thrombosis, or embolism.

(*c*) NERVOUS COMPLICATIONS.—Typhoid may begin as mani
The author has seen five typhoid patients taken to the detention ho
pital in the third week, or even in convalescence, under a mistak
diagnosis. It may be a later though generally a rare symptom.

Psychoses are generally post-febrile, are of the depressive varie
such as melancholia, and usually give a favorable prognosis, thou
alienists assert that one-third to one-fifth do not recover. Eds
(1905) found that two-thirds of juvenile cases recover. Their cau
is the exhausting inanition of protracted toxæmia, or sometimes
hereditary predisposition. Typhoid may cure a previous insanit

Meningitis is usually cerebrospinal and occurs in 0.5 per cent. of t
cases, mostly in women and children, generally as a complication a
late in the typhoid course during the period of "steep curves." Me
ingitis may be due to the pneumococcus, the pyogenic cocci, or
Eberth's bacilli, which have been found on lumbar puncture in ov
twenty-six cases (Cole), of which half were serous and half suppur
tive. Mastoiditis as well as tuberculosis may cause the meningit
there being four recorded cases of the tuberculous variety. Actu
meningitis must not be confounded with pseudomeningitis or "meni
gismus," in which toxæmia produces irritative meningeal sympto
as above enumerated.

In the *brain*, œdema, degenerated and pigmented ganglionic cel
and nuclear multiplication, are the most common findings at t
autopsy. Hemorrhage, embolism, arterial and sinus thrombos
and abscess (sepsis), are rare pathological complications. Aphasi
monoplegia, hemiplegia, and paralysis of the cerebral nerves, are ra
clinical complications. The author has twice noted hemiplegia dev
oping suddenly in convalescence, probably embolic from detached hea
clots. In both cases the patients were allowed to leave the bed t
soon. Hawkins has noted seventeen cases of hemiplegia in typhoi

In the *spine*, myelitis, multiple sclerosis, infantile paralysis, a
Landry's paralysis (Eberth's bacillus), are the most common compli
tions or sequels. The knee-jerks are sometimes increased, especia
in emaciated subjects.

Neuroses may complicate convalescence, such as chorea, hysteria, neurasthenia, catalepsy, Basedow's disease, tetany, paralysis agitans, diabetes insipidus.

In the *nerves*, neuritis in the single or multiple form (*q.v.*) with anæsthesia, paralysis, and atrophy, may follow typhoid as it does other infections. Neuralgia occurs late. In the feet it constitutes the "tender toes" of Hanford, and is seemingly increased by bath treatment. It always results in recovery.

6. **Special Senses.**—The eye is rarely involved. Muscle paralysis (neuritis) and various inflammations—such as simple or phlyctenular conjunctivitis, iritis, panophthalmitis, uveitis, and cataract—are very uncommon. In convalescence, mydriasis and paralysis of accommodation often result from exhaustion. Amaurosis, either functional or lasting, from disk-disease, has been observed, as well as retinal hemorrhages, optic neuritis, choked disk, optic atrophy, orbital hemorrhage, and venous thrombosis. Uhtoft, in 253 cases of optic neuritis, found 17 cases following typhoid.

Involvement of the *ear* includes functional toxæmic disturbance of hearing, abscess, and otitis media (2 to 14 per cent.). Otitis is less frequent than in scarlatina or measles. Mastoid disease and meningitis are very exceptional.

7. **Muscles, Thyroid Gland, and Bones.**—The muscles anatomically are dry, of a smoked-meat appearance, and may be the seat of hemorrhages, rupture, granulo-fatty and waxy degenerations, and abscess, due to Eberth's bacilli and pus cocci. Myositis may explain some cases of muscular tenderness with cramps.

In the *thyroid gland*, a strumitis typhosa, with suppuration or regression, has been observed. It is due to the typhoid bacillus. Recovery is the rule.

Though *bone disease* was observed in 237 cases by Keen, it is much more frequent than the figures indicate. Periostitis is more common than bone disease proper. These complications are post-typhoid and are due to the typhoid bacillus (75 per cent.), or pus cocci (25 per cent.). They attack the tibia (38 per cent.), the ribs (13 per cent.), femur, ulna, temporal bones, etc., and occur chiefly in young individuals. "Their most striking features are chronicity, indolence, and a remarkable tendency to recurrence" (Osler). Osteomyelitis may resemble syphilitic or tuberculous osteopathies. Typhoid bacilli have been found seven and ten years after typhoid in osteomyelitic fistulæ. Longcope has found marrow changes which resemble lymphoid hyperplasia, and Fraenkel isolated typhoid bacilli in the vertebral marrow in ordinary cases of typhoid.

Arthritis may be typhoid or gonorrhœal, rheumatic, septic, etc. Keen has noted eighty-four cases. It may be poly- or mono-articular, serous or purulent. The hip is most frequently affected. The "typhoid spine," of Gibney, is characterized by pain, tenderness, and stiffness in the back. Local trouble, such as spondylitis (six recorded cases), or perispondylitis, is sometimes found, but the so-called typhoid

spine is often caused by hysteria, which is known to follow vario
acute infections. The prognosis is good.

8. Digestive Tract.—The upper lip is retracted and bleeds easil
Sordes of dried saliva, food, and epithelium, are observed on the tee
and gums. Though the tongue shows nothing absolutely characte
istic, Leube and others attach special importance to its appearanc
At first it is white on the dorsum, moist, flabby, and therefore indent
by the teeth, and its edges are red. It may be moist throughout t
disease. In the second week it becomes dry (from the fever, lack
saliva, and mouth-breathing), small and fissured; it bleeds, and
brown or even black coating replaces the white. In the third we
or later, a triangular patch clears at the tip of the tongue. Typho
bacilli are not uncommon on the tongue. Acute glossitis is a seve
complication. Aphthæ are rare, but may occur throughout the dig
tive tract.

Angina may usher in the disease, or, occurring in the first wee
lead to a wrong diagnosis. The throat is swollen, granular, and erode
The angina is usually of the ordinary type, but may be specific (Wagr
and French), due to the typhoid bacillus. The phlegmonous ty
is very dangerous. An angina cachectica of white plaques or epitl
lium and bacteria is occasionally observed.

Parotitis, occurring in ½ to 1 per cent. of the cases, has become l
frequent since special care of the mouth has lessened infection ascer
ing Steno's duct. It is usually one-sided, is due to the typhoid bacill
or pyogenic cocci, and is usually seen on the fifteenth day. It indica
a poor prognosis, especially when due to metastasis, and may rea
the skull, meninges, or brain. Two personal cases, caused by t
typhoid bacillus, recovered.

Specific typhoid lesions are rarely observed in the *stomach*, althou
Leube records an instance with hæmatemesis. Early and severe *e*
gastric pain rather contraindicates typhoid. Incipient *nausea* a
vomiting are unusual, occurring mostly in women or children.
observed at the height of the disease these symptoms suggest men
gitis, overfeeding, or perforation, and (in convalescence) dietary err

Anorexia is observed at the onset and during the fastigium, thou
great hunger develops in convalescence.

Ulceration of the œsophagus is very rare; the cicatrized ulcer n
produce later stenosis (twelve cases were collected by J. E. Thon
son, 1904).

The *liver* in the early stages is anatomically hyperæmic, large a
firm; later its lobular markings become indistinct from cloudy sw
ing and fatty change. The organ is softer, and Wagner's lymp
mata (and some focal necrosis) occur. Clinically, some tenderness
not unusual and at times may lead to an incorrect diagnosis of li
abscess or cholecystitis.

Icterus is very rare (0.5 per cent.), and its absence is of diagno
value, as the duodenal catarrh common in other infections is usu
absent. Icterus may occur from gall-stones, cholecystitis, abscess,

acute yellow atrophy. The author has but once seen icterus in typhoid fever. Single liver abscess is rare, as are suppurative pylephlebitis and suppurative cholangitis. Sheldon (1903) noted twelve cases of multiple liver abscess.

Bernheim maintains that typhoid favors the *formation of gall-stones.* They may develop months, years, or decades later.

Cholecystitis has been reported by Chiari and others; thirty cases with perforation are on record. Tenderness, pain, muscular rigidity, and enlarged gall-bladder, are found in most cases. Typhoid bacilli are found in the gall-bladder in nearly all autopsies, although seldom having produced symptoms, and are held to be the main cause of re-infections. They may also invade the bile tracts without producing or following typhoid. The bacilli in the biliary tract more probably occur from hæmatogenous than ascending infection.

Bretonneau first noted specific changes in the *intestines:* (*a*) *Hyperæmia, catarrh,* and beginning medullary infiltration in the lymphoid structures during the first week, most changes being observed in the lower ileum, whence the name ileo-typhoid. (*b*) *Medullary infiltration* or *hyperplasia,* during the second week, occurring first in Peyer's patches, and then in the follicles. The patches are oval and parallel with the long axis of the gut; they lie opposite to the mesenteric attachment, are 3 to 5 (8) millimeters high, and are at first red, then paler from compression of the blood-vessels. The infiltration is hard or soft. It may be uneven and reach beyond the plaque. The solitary follicles, even to the sigmoid flexure (colotyphoid), may be principally enlarged. These changes are not absolutely distinctive of typhoid, but are more significant in adults than in children, in whom they are frequent in digestive diseases, the exanthemata, and diphtheria. Mallory believes that the lymphoid swelling is wholly explained by the proliferation of the endothelial cells of the vessels to act as phagocytes; when they degenerate, fibrinous thrombi develop. Medullary infiltration need not advance to ulceration. Regression may occur and the lymph-cells degenerate and disappear. The lymphatic structures may then assume a reticulated, or "shaven beard," appearance. (*c*) *Ulceration* occurs in the third week, from anæmic necrosis, slough formation, and exfoliation of small particles. The ulcers thus formed by the typhoid bacilli are usually easily seen without opening the gut. They have an irregular, oval outline, sharp, steep, often undermined edges, with a red floor generally composed of the muscular coat and often extending deeper than the eschar. The lower ileum is most affected and may present almost no normal surface in extreme cases. The solitary follicles, both in the large and small gut, ulcerate at their apices. Baer has studied the localization of the typhoid ulcer and states that it may occur in any part of the digestive tract except the mouth. The pharynx is affected in 2 per cent. of the cases, the œsophagus in 4 per cent., the stomach in 2 per cent.; 97 per cent. of the ulcers develop in the small intestine, 89 per cent. in the ileum, and 2 per cent. in the appendix; 33 per cent. in the large intestine,—and there only, in 6 per cent. (*d*) *Cicatrization* occurs by formation of granulation tissue and growth inward of the epithelium. The patch is depressed and slightly pigmented. The typhoid cicatrix does not narrow the intestinal lumen.

There may be few or no anatomical changes in the intestine. This is analogous to diphtheria without membrane, and the exanthemata without eruption. Slight bowel lesions are usually present in most of the cases reported "without intestinal lesions."

The *mesenteric glands* are swollen, contain typhoid bacilli, are altered like the intestinal lymphatics, and may be palpable. The swelling occurs mostly in the second week. The swollen glands may regress, soften, become tuberculous, or suppurate, sometimes causing peritonitis, or simulating perforation, as in a case of the author's. Only six cases are recorded of suppuration in the mesenteric glands. Le Conte reports a recovery after operation. Other lymph-glands may be similarly involved.

No parallelism exists between the frequency, constancy, or severity of the clinical symptoms, and the degree of anatomical involvement *e. g.*, in ambulant cases there may be perforation or hemorrhage, o typical lethal cases may occur without intestinal change.

Meteorism is rather less common than usually described. It occur mostly in the duodenum or jejunum above the ulceration, or some times involves the colon. It is less frequent and less intense wit early treatment and careful dieting. If much tympany develops unde these circumstances, the prognosis is poor, since meteorism is due t toxæmic paresis of the intestinal musculature rather than to loca ulceration, catarrh, or serous infiltration. The heart and lungs ma be crowded upward and dyspnœa result. It may occur with hemor rhage or perforation.

A thickened ileum may be palpated. Abdominal *tenderness an pain* occurred in 60 per cent. of Osler's series, where particular atten tion was paid to these points. They are due to: (*a*) local bowel con ditions, as gas, constipation, inflammation in the ileocæcal region, o perforative and peritonitic complications; (*b*) to splenic or hepati tumefactions; (*c*) pleurisy, distended bladder, phlebitis, etc.; (*d* muscle changes or cutaneous hyperæsthesia, so frequently observe in fever of any variety.

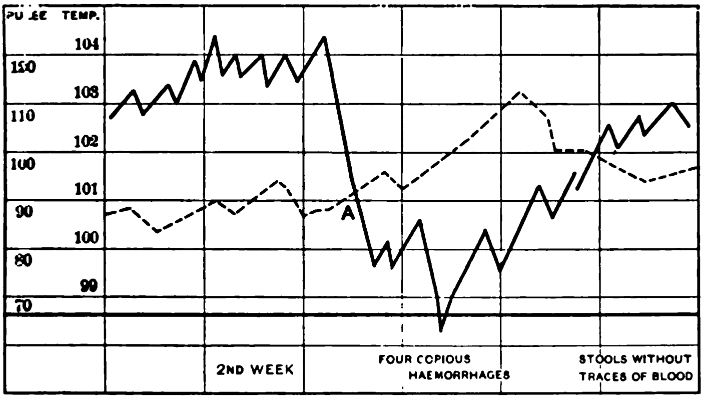
Typhoid *appendicitis* may simulate ordinary appendicitis, which differentiated only by the blood-count, sero-reaction, or, most surel by the clinical evolution; *e. g.*, the fever spots, etc., when due t typhoid, precede local pain and tenderness. Gurgling has no diag nostic value whatever in typhoid, although it occurs in most cases.

The *stools* present no pathognomonic chemical nor physical cha acteristics other than the bacillus typhosus, which is often abse during the first week, and also in the later course. The dejecta a thin, offensive, pea-soup-like, alkaline, and resemble the contents the small intestine (Addison). Because the fæces are poor in mucu two layers form on standing, an upper cloudy, and a lower, yello gray, granular and flocculent layer. The solids are 4 per cent. The stools contain red corpuscles and triple phosphates, the latter more abundantly than in any other disease. Shreds or sloughs may also be found.

Diarrhœa in the febrile stage occurs in 25 to 33 per cent. of the cases, although some state that it is observed in most patients. Three to six stools are the average. If this is exceeded, the type of the disease is severe; prodromal diarrhœa rarely occurs. Diarrhœa is a measure of the degree of toxæmia or of colonic involvement, and is not a result of ulceration of the small intestine.

Hemorrhage is seen in 4 to 6 per cent. of the cases. In point of time it is usually late, though some assert that in 30 per cent. it occurs in the first two weeks. It is less frequent in relapses and in children (1 per cent.). Ulceration into the muscularis is its chief cause. Oozing, from simple hyperæmia, bleeding in the hemorrhagic forms (blood dissolution), and in patients inheriting the bleeding tendency, are exceptional. Bleeding is more common in ambulatory, delirious, and carelessly fed patients. There may be no symptoms if the hemorrhage is small (occult hemorrhage); but if it be large, acute anæmia suddenly develops, with collapse, which is evidenced by a fast pulse, lowered temperature, and pallid skin. The intellect often clears, the spleen becomes smaller, and fresh blood, or, later, black, tar-like movements, are voided (manifest hemorrhage). The abdomen is often distended. There may be dulness on percussion from blood retained in the bowel, if the peristalsis is weak and the hemorrhage is very profuse. In two rapidly fatal cases the entire abdomen became flat and one and two pints of blood were vomited. Confusion with bleeding piles and bismuth stools is possible. The average number of hemorrhages is four.

Fig. 4.



Typhoid-fever curve, with fall of temperature (solid line) after hemorrhage and rise of pulse (broken line). Note crossing of curves at A.

Some patients improve at once after hemorrhage, as was remarked at an early date by Graves and Trousseau; yet 20 to 50 per cent. die. Large blood-clots are ominous, indicating erosion of a large artery Hemorrhage may cause death within an hour (Trousseau, Leymarie)

Perforation occurs mostly at the time of eschar-formation, at the end of the second, or in the third week; but it may also occur later,— in the third to fourth week (50 per cent., Fitz); or from the third to the fifth week (Leube). Scott found that 92 per cent. occurred between the second and fifth weeks. It develops in 9 to 12 per cent. of fatal cases and in 3 per cent. of all cases, but it very rarely occurs in children. Scott's figures, including 9,713 English and American cases show perforation in one-third of all fatal cases. Perforation is usually low in the ileum (81 per cent.); or in the colon or sigmoid (12 per cent.) in the appendix (3 per cent.); in Meckel's diverticulum (2.4 per cent.) or in the jejunum (1.2 per cent.). The percentages are from Fitz' figures. In 83 per cent. of the cases but one perforation is found though several may occur, twenty-five being once observed in one patient. It is promoted primarily by deep ulceration, and secondarily by severe infections, movement, strains, coughing, constipation, gas or solid diet, which increases peristalsis. It is claimed that 75 per cent. of the cases of perforation occur in severe infections. Thre types are encountered: (1) Least frequently, undiscovered latent perforation; the typhoid state masks the perforation, which affords an unpleasant postmortem surprise. (2) More frequently, cases with gradual onset, and therefore ambiguous symptoms. (3) Most frequently, cases with sudden symptoms and more or less clear diagnosis. The *symptoms of perforation* are: (*a*) Pain (in 75 per cent of the cases) due to the perforation itself, usually localized in th hypogastrium or right lower quadrant of the abdomen and followed later by the pains of peritonitis. In 70 per cent., the pain was sudden and severe; in 20 per cent., gradual in onset.; and in 10 per cent. it was absent. (*b*) Tenderness on palpation (75 per cent.), evidenced by the board-like rigidity of the abdominal muscles. (*c*) Limitation o the respiratory movements of the abdomen, due to pain and tenderness. (*d*) Vomiting of gastric contents or fæcal matter. (*e*) Intestina obstruction. (*f*) Abdominal distention, appearing possibly for th first time—in which event its significance is enhanced—or increasing if already present. There are cases in which the abdomen is flat o even retracted; in one typhoid patient, seen first in a dying condition the scaphoid abdomen contained three pints of pus. Abdominal distention crowds upward the diaphragm, heart, and lungs, and causes hurried breathing. (*g*) Disappearance of the flatness of the liver b reason of free gas in the peritoneum. This is most significant if th abdomen was not previously distended. Perihepatic adhesions naturally prevent obliteration of the liver dulness, and it must always b remembered that a gas-distended colon may overlie the liver; an that in some cases the colon normally overlaps the liver. (*h*) Othe occasional abdominal findings are flatness in the flanks, caused b fluid; the Beatty-Bright friction-rub; absence of paristalsis; rect or vesical pain or tenderness. (*i*) Marked leukocytosis is not always found, and special emphasis on its absence is most misleading, as the leukocytes are occasionally decreased. (*j*) Sudden fall of temperature, rapid rise in pulse-rate, and other signs of shock, may occur at the time of perforation, though less frequently than the usual descriptions indicate. The writer has seen cases in which the pulse-rate did not increase. Dieulafoy mentions a case, successfully operated for perforation, in which two later perforations resulted fatally. The sudden clearing of the patient's intellect may deceive the inexperienced physician. In the advancing generalized peritonitis, the fever, if it has fallen, rises; the pulse becomes rapid, small, and thready; the skin clammy and dusky, and the features pinched (facies Hippocratica). Hiccough is common. The peritonitis rarely remains local. In six of the author's cases, the local abscess was ruptured later. Ninety-eight per cent. of the unoperated cases die.

Peritonitis occurs frequently in ambulatory cases, from rupture of the bowel, gall-bladder, spleen, lymph-glands; occasionally from post-typhoid appendicitis or without rupture of any organ or tissue. It is found in 2 per cent. of fatal cases.

9. The Respiratory Tract.—This is the seat of frequent and manifold symptoms and complications.

(*a*) THE NOSE.—*The nose* is dry. Epistaxis occurs in 7 to 20 per cent. of the cases, usually early, and in young subjects; it is often profuse, sometimes dangerous, but rarely lethal. Croup and diphtheria are rare; coryza is very rare. The author has seen two cases begin with profuse coryza.

(*b*) LARYNX.—*Catarrh* is common. *Decubitus*, with erosions of the swollen hyperplastic lymph-follicles (like the intestinal changes), occur in the posterior wall, followed often by œdema glottitis. These bedsores are seen at the height of the fever; occur rarely in children; and are found in 5 to 20 per cent. of the lethal cases. *Perichondritis* and *necrosis* are attended by pain, aphonia, stenosis, mediastinitis, cervical cellulitis, and diffuse emphysema. Tracheotomy may be indicated. The author has seen but three cases of perichondritis. Dupuy (1903) collected 255 cases of perichondritis and submucous laryngitis, and believes they explain 3 to 10 per cent. of typhoid fatalities. Without operation, the mortality is 98 per cent. Laryngeal neuritis, with cord paralysis, follows typhoid in 25 per cent. of the cases.

(*c*) AIR TUBES.—A *dry catarrh* of the trachea and bronchi is almost invariable, while *ulceration* (in one-third of 1 per cent.) or *fibrinous inflammation* is rare. Rales, retrosternal dryness, rawness, and pain, are present in the bronchitis of typhoid. At autopsy the bronchial glands are often swollen, and Eberth's bacillus is sometimes found. ***Bronchiolitis*** is regular and specific. It is found everywhere in the lungs, but especially in the lower lobes, and (rarely) wholly above and in front. Coughing is not invariable but its presence in early typhoid is of value in differentiation from simple intestinal catarrh. The outlook is less favorable when severe and early coughing occurs.

(*d*) LUNGS.—*Hypostasis*, from a dorsal decubitus and weak heart, is often relieved by change of posture. It is very common, and occurs in the first half of the disease, or at its height, mostly in weak adults and in cases of severe infection. Lesser degrees of hypostasis may be overlooked. The percussion-note is tympanitic or dull. The fremitus is increased, if the patient can talk, or decreased from stagnation of secretions. The breathing, and especially the expiration, is distant (or sometimes bronchial). Rales, moist, crepitant, and subcrepitant, are heard.

Lobar pneumonia may, in rare instances, dominate the early clinical picture (pneumo-typhoid) and may be of two types. The first type, (*a*), is due to Eberth's bacillus. The course is that of an initial pneumonia, which does not end typically, but shows later a typhoid temperature, rose spots, Widal reaction, etc. These cases are naturally confusing, especially in their incipiency, and the typhoid is more apt to be overlooked than in the next type, (*b*) in which the pneumonia, usually seen at the height (third week) of the disease, is a true complication, due to the pneumococcus, though sometimes due to the colon bacillus and streptococcus, especially in sepsis. The pneumonic symptoms are variable, atypical (as in most secondary pneumonias), often without chill, with a slower course, and often with lysis, or, less often, with such complications as abscess or gangrene. More than one-half of these cases die. *Lobular pneumonia*, from inhalation, puscocci, and Eberth's bacillus, is a true complication. *Abscess* follows pneumonia or sepsis; it occurs in 0.7 per cent. of the fatal cases. *Infarct* occurs in 6 per cent. of typhoid fatalities. Marantic thrombi from the leg may reach the lungs by way of the right heart. This is indicated by hæmoptysis, chill, temperature, pain, etc. *Gangrene* (1 to 2 per cent. of typhoid autopsies) occurs more often than abscess, is seen late in the disease, and is usually metapneumonic, but may be caused by perichondritis or aspiration of food.

(*e*) PLEURA.—*Serofibrinous pleurisy* is usually rare unless the lungs are diseased. Sears observed 18 cases in 1,065 typhoid patients, and Leube has seen several with pleural friction. Postmortem statistics show it in 6 to 8 per cent. It is most common as empyema in convalescence, and is due oftenest to Eberth's bacillus. In 1903, fifty-five cases of pleurisy were collected in which the typhoid bacillus was found. *Pneumothorax* and *hæmoptysis* are very uncommon.

(*f*) TUBERCULOSIS.—Old latent tuberculosis may be aroused, usually late in the disease. It is associated sometimes with pneumococcus infection. Other forms, such as *acute caseous pneumonia*, *subacute tuberculous peribronchitis* with a few bacilli, fever, emphysema and aggravation of the bronchial symptoms, and occasionally miliary tuberculosis, may occur.

10. Genito-Urinary Tract.—The following conditions may be found: (*a*) The *urine is febrile;* as water is retained in fever, the urine becomes decreased, acid, concentrated, with a specific gravity of 1,030 or more. Uric acid, urea, and toxins, are increased; the chlorides, decreased. Typhoid bacilli are present in 25 to 50 per cent. of the cases, or even 80 per cent., according to other statistics. The bacilli may give the urine a glistening appearance. Bacilli may persist from four months to several years after recovery. In convalescence, the urine is increased, is neutral in reaction, and has a lower specific gravity. Sometimes polyuria may occur during the fever. (*b*) Serum albumin, globulin, and rarely peptones, are found in *febrile albuminuria.* Serum albumin is found, in quantities varying from traces to considerable amounts, in 15 to 20 per cent. of the cases; cylindruria is approximately half as frequent. The prognosis is three times as unfavorable when albumin is found. It appears from the seventh to the tenth day, is frequent in the third week, and its average duration is twelve days. It corresponds anatomically to parenchymatous and fatty degeneration of the kidneys. (*c*) *Nephritis* in typhoid is of the acute parenchymatous type, with the interstitium of the organ but little involved. It is often hemorrhagic, and occurs usually in the first three weeks but occasionally in convalescence. It is found in 1 per cent. of the cases, mostly in men, rarely in children. Nephritis makes the prognosis less favorable, as it indicates a more severe infection, from which 50 per cent. of the cases die. Uræmia and suppression of urine are rare. If not fatal, nephritis usually clears up entirely and rarely remains in chronic form. Early and marked renal symptoms constitute the nephrotyphoid of French writers. Sepsis may produce septic nephritis and abscesses. Miliary typhoid abscesses in the kidney may keep up the bacilluria. Lymph-nodes are found in some cases, as in the liver (Wagner). (*d*) The urine of typhoid exhibits the *diazo reaction* of Ehrlich (1882). Two standard solutions are kept in separate bottles: No. 1—a 5-per-cent. solution of hydrochloric acid saturated with sulphanilic acid, which should be fresh; No. 2—one-half-of-1-per-cent. solution of sodium nitrite. To make the test, forty parts of No. 1 and one part of No. 2 are mixed, producing diazo-benzine-sulphonic acid. Equal parts of this mixed solution and urine are thoroughly shaken; one part of ammonium hydrate is then allowed to flow carefully down the side of the tube to stand above the urine mixture. At the junction of the two fluids, a dark garnet or cherry-red ring will form in typhoid urine, and in normal urine only a brown ring. The pink color of positive tests is also imparted to the foam on shaking, or in marked dilutions gives a rose-red hue. Osler found it in 69 per cent. The author has found it in 98 per cent. of his cases. It occurs in the early stages of the disease, also at the acme and during relapses. It is a valuable sign, though of secondary rank. Since it is found in numerous other acute and chronic conditions, it has no differential or pathognomonic value. Its absence, together with the absence of other findings, contraindicates a florid typhoid. (*e*) *Cystitis* and *pyelitis* occur during the disease or in convalescence, and are due chiefly to pus organisms, or typhoid or colon bacilli. Simple retention of urine is not uncommon. Urobilinuria (Tissier) and indicanuria occur, but neither is proportionate to the severity of the disease. *Hæmoglobinuria* is quite rare. (*f*) The *genitalia* are more often implicated in women than in men. Menstruation is disturbed. It often appears early in the onset of typhoid, and is suppressed in 60 per cent. of the cases. In some instances hæmatocele or hæmatometra may develop. Endometritis, either croupous or diphtheritic, vaginal erosions, ulcers, inflammation, or gangrene and mastitis, are rare complications. *Pregnancy* is of grave import the outlook being worse in its later stages than in the earlier (*e. g.* eight to ten weeks). It is most dangerous at the acme of the fever abortion and premature delivery being very frequent (65 per cent of the cases). The maternal mortality is increased at least twofold Blumer and Dobbin have reported puerperal infection from the typhoi bacillus. *Orchitis* usually develops late, or in convalescence. It occur with chills and fever. The patient almost always recovers in ten t fourteen days, although suppuration and atrophy have occurred—fifty-three cases were collated by Kinnicutt. It is frequently associate with urethritis. Emissions are common in convalescence.

Anomalous Courses.—According to Curschmann, the followin anomalous courses are distinguished:

1. **Malignant.**—The *malignant*, hyperpyretic or foudroyant form i rare. It is severe even in the earliest stages. The temperature rise rapidly and there may be hyperpyrexia. The pulse is soft, weak, an rapid. The status typhosus is profound. Meteorism, diarrhœa, and albuminuria, are present, and death from deep intoxication usually occurs

2. **The Slow Severe Form.**—The *slow severe* form lasts from fou weeks to four months. The fever is often remittent with frequen exacerbations. Defervescence may occur or death intervene, marasmus alone being found at autopsy. The severe types of typhoi have grown rarer, even during the last decade.

3. **Typhus Abortivus.**—*Typhus levissimus, typhulus, typhoidette* c Brouardel, exhibits a short and mild course, temperature with short initial stage, or often a crisis, lasting six to twenty days. Bronchitis is frequent, the spleen enlargement is constant, and meteoris diarrhœa, roseolæ, hemorrhage, or perforation, are rare. Relapses ar however, more frequent.

4. **Typhus ambulatorius** (of the German, or latent typhoid of Frenc writers—walking typhoid), occurs in adults, especially in men, wh often endure it by the help of alcohol. Some malaise may exist. Ther are two types: (*a*) the lighter and atypical, and (*b*) the usual cours very often ending in hemorrhage, perforation, etc. The temperature often not marked, but is usually less at night than during the diurn activity. The pulse is faster because of exertion. The spleen is ofte enlarged, and the roseolæ are frequent.

5. **Typhus afebrilis** (Liebermeister and Gerhardt) is most often m with in family practice. It may assume the ambulatory form; an is diagnosticated by exclusion and by the pulse, spleen, roseolæ, dia reaction, Widal test, etc. Atypical cases have apparently increase in recent years.

6. **The hemorrhagic form** is characterized by nasal, subcutaneous, and particularly intestinal hemorrhage, constituting the blood dissolution of the older writers. It occurs especially in children and alcoholics, and constitutes less than one-tenth of 1 per cent. of typhoid cases. The prognosis is usually but not invariably bad. The four cases which the author has seen occurred in 1900, in St. Luke's Hospital, where a severe house-epidemic prevailed.

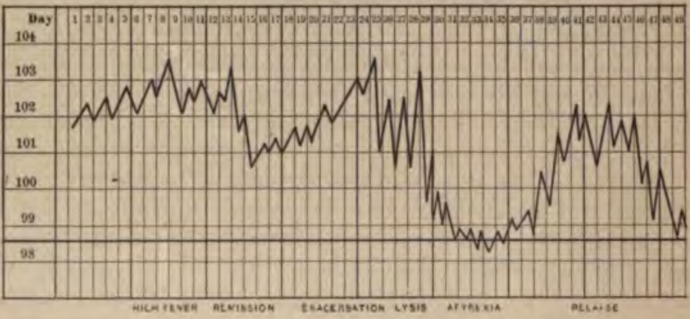
7. **Visceral Forms.**—The so-called *visceral forms* should seldom cause confusion, if watched with care. They include the gastric or bilious types, nervous fever, meningo-typhoid, cerebro-typhoid (mania, psychoses), laryngo- or tonsillo-typhoid, nephro-typhoid, pneumo-typhoid with pneumococcus mixed infection or Eberth's bacillus, (Lépine, Chantemesse, Widal,) which is pneumo-typhoid in the narrowest sense.

8. **In children** but few cases occur in hospitals. The temperature course is shorter and milder than in adults. The younger the patient, the less is the degree of fever. It may begin with a chill. It is often remittent, the "infantile remittent fever" of Abercrombie being really typhoid. Epistaxis is less common. The pulse in children is faster, and more often irregular, than in adults; though after the twelfth (fifth, Filatow) year, it is equally slow. A weak and irregular pulse is very rare, and a fast, irregular pulse generally indicates meningitis. Opinions differ as to the frequency of roseolæ; they vary with the epidemic, but it is probable that they occur as often in children as in adults. They may be fewer and may appear later, but in some cases are abundant and confluent (in children between the fifth and tenth year). Bedsores are very rare, but noma is more frequent than in adults. The nervous system is generally less frequently involved in children, though if the infection is severe, convulsions, irregular pupils, rigidity of the neck, headache, etc., may occur. (See TYPHOID STATE.) Neuralgias and psychoses are rare. The frequency of aphasia has not yet been explained. Bronchitis occurs as often as in adults, but atelectasis, hypostasis, and lobular pneumonia are more frequent. Children exhibit less meteorism and hemorrhage (1 per cent.), and much less frequent perforation. There are fewer anatomical changes. Splenic enlargement is less frequently palpable (in 50 per cent.), and occurs somewhat later. Initial vomiting, pain, and perhaps diarrhœa, are more frequent. Albuminuria and nephritis are relatively rare. Griffith and Ostheimer have assembled 394 cases in children, of which 325 are beyond doubt. Of these, 139 occurred under one year, 187 in the second, and 68 between two and two and one-half years. The mortality under one year (*v. i.*) was 73 per cent., and of the entire group, 50 per cent. Typhoid is rare in the first two years of life. It is sometimes overlooked, being very atypical. The course is short. Bronchitis, splenic tumor, and roseolæ, are infrequent. If the disease is severe, a very high temperature, meningeal symptoms, and initial vomiting occur, and also 50 per cent. mortality caused by toxæmia, pneumonia, perforation, or ulceration of the larynx.

9. In the aged typhoid is infrequent, especially after the fortieth year. In such cases the fever is lower, atypical, and often absent. The pulse is faster, irregular, and without dicrotism. There is greater lung congestion owing to weakness of the right heart, and lung complications are more frequent. The nervous symptoms are dominant, often occurring with great depression. Roseolæ are fewer and bedsores are more frequent. The spleen is much less often enlarged because of old infarcts, induration, and a thicker capsule. Tympany and hemorrhage occur more often. Convalescence is naturally slower. In 1903, the author had under his care, in Wesley Hospital, a woman, aged fifty-seven, and her mother, aged seventy-two, both with typhoid, ending in recovery.

Relapses and Exacerbations.—Relapses and exacerbations differ in degree only. Their explanation is still obscure, reinfection from the gall-bladder, spleen, glands, and the bones, having been advanced as causes. In the complete relapse the temperature falls to normal, or nearly to normal, for five days on the average, and then fever and other signs of typhoid reappear. It occurs in 6 to 12 per cent. of the cases, and vary with the epidemic, sometimes occurring in as many as 35 per cent. Relapses are more common in young (in one-sixth of Henoch's cases) than in old subjects. They are more likely to occur (*a*) in light or moderately severe cases (75 per cent.) than in severe ones (25 per cent.); (*b*) when the splenic tumor does not disappear (Gerhardt; (here the question may, of course, arise whether or not the spleen was enlarged from some previous disease, such as malaria;) (*c*) when the temperature in convalescence does not become subnormal (Curschmann); or (*d*) when the pulse is variable and rapid while the patient is quiet (Curschmann).

FIG. 5.

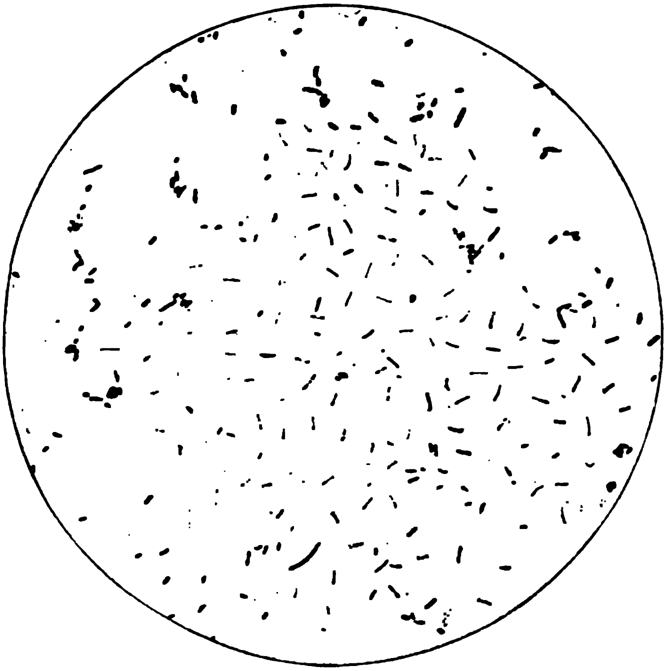


Showing remission, exacerbation, lysis, and relapse.

After an average defervescence of five days, the fever runs the sam course as in the first attack, though it is shorter by ten days (Murchi son). The pulse is faster (110 to 130 in women, children, and wea men), labile, and less dicrotic. There are less roseolæ (absent in 2 per cent.), nervous symptoms, respiratory symptoms, hemorrhag

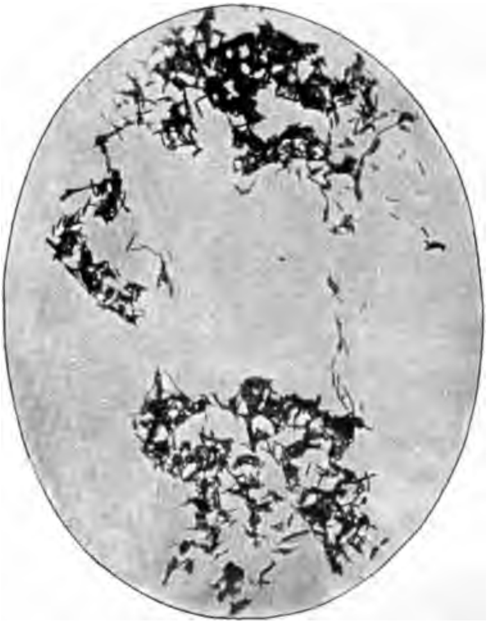
PLATE I.

FIG. 1.



Bouillon Culture of Typhoid Bacilli before the Addition of Diluted Typhoid Serum. × 800. (After Cabot.) Serum diagnosis.

FIG. 2.



he Same, Five Minutes after the Addition of Typhoid Serum (dilution 1 to 10), showing Typical Clump Reaction. × 400. (Cabot.)

(4 per cent.), and perforation (0.7 per cent.), whence, as a rule, the prognosis is relatively good. The duration of the relapse is six to fifteen (even twenty-one days), and there are seldom more than two,—three or four being very rare. The author has seen five relapses, covering, with the original attack, as many months. It is said that they may be afebrile, with a return of the roseolæ, spleen, etc. Relapses should not be confused with Biermer's "after fever," due to inanition or possible sepsis. *Recrudescences* are renewals of the fever after it has lessened, but not entirely subsided. In 50 per cent. of the cases recrudescence is followed by more severe symptoms.

Convalescence.—Convalescence requires fourteen to twenty-one days, but its duration is variable, and it is not certainly established till subnormal temperature, at least in moderate or severe infections, is observed. The pulse tends to run high on exertion or excitement. Anæmia, emaciation, œdema, phlebitis, gall-stones (formed by Eberth's bacillus), tuberculosis, neurasthenia, psychoses, bone, muscle, and other complications, may occur. If the appetite is poor in convalescence, complications are to be expected.

Diagnosis.—**General Rules.**—1. The bacteriological diagnosis is not usually practicable, for the colon bacillus confuses the inexperienced physician in the examination of the urine and fæces. Splenic puncture is never indicated, though positive in 95 per cent. of cases tested. The bacilli may be found in the blood in nearly all cases, and practically always in the roseolæ.

2. Observe long and carefully. Say that "a diagnosis is impossible in the first few days."

3. No single sign is absolutely diagnostic, except bacilli in the blood, etc., and the characteristic Widal reaction.

4. Diagnosticate (*a*) from positive findings, and (*b*) by careful exclusion.

5. The "typhoid state" only indicates some toxæmia, not necessarily typhoid.

6. In doubtful cases remember the great statistical frequency of typhoid.

7. Remember its manifold forms; *e. g.*, pneumo-typhoid, etc.

8. Be on the safe side in light, ambulatory, or abortive cases as to the diagnosis and the still more important matter of treatment.

The Cardinal Signs.—These are: (*a*) The Widal test; (*b*) bacilli in the blood, in the urine, in the roseolæ, etc.; (*c*) the roseolæ; (*d*) the slow pulse; (*e*) the fever-curve; and (*f*) the splenic tumor.

(*a*) The sero-test, successful in 95 to 97 per cent. of cases, is important. As stated above, its absence does not exclude typhoid.

(*b*) The bacilli in the blood are found in practically all cases.

(*c*) The eruption has a diagnostic value in its location, occurrence in crops, time of appearance, and short duration.

(*d*) The slow pulse, and to a much less extent its dicrotism, are suggestive. It is rarely to be confused with the slow pulse of tuberculous meningitis.

(*e*) The fever-curve shows a gradual, step-like onset, continuou fastigium, steep curves in the breaking stage, and subnormal registe in established convalescence.

(*f*) While the splenic tumor occurs in many other infections, it i *per se* suggestive after the first week.

Secondary Signs.—These are, in order of importance, the typhoi tongue, the stools, bronchitis (especially after the first week), th typhoid state, leukopenia, intestinal symptoms and complications, an the diazo reaction.

Symptoms contraindicating typhoid are, according to Leube, coryz early vomiting and sweating, herpes, endocarditis, retraction the abdomen, persistent constipation, and constant failure of tl diazo test. To these may be added leukocytosis (unless some complic tion has occurred), icterus, and rigors. All of the above-mention symptoms may, exceptionally, complicate typhoid.

Differential Diagnosis.—Differentiation from acute miliary tuberc losis may be most difficult. In both diseases protracted fever, splen tumor, brain symptoms, bronchitis and bronchopneumonia, rap pulse, etc., may be observed, and miliary tuberculosis may develop fro typhoid (Birsch-Hirschfeld, and in two personal observations). T phoid may be complicated with septicæmia, very rarely with malar in this climate, or with meningitis. The table on pages 44 and illustrates the differential diagnosis of the diseases most likely to confused with typhoid.

Exanthematous typhus (*q. v.*) and recurrent fever (*q. v.*) are easi distinguished. The *exanthematous diseases*, such as scarlatina a measles, are rarely confused, except, perhaps, in the initial stage. most extensive eruption of roseolæ resembling measles is easily d tinguished by the course, temperature curve, and catarrhal sympton

Fibrinous pneumonia rarely resembles typhoid, or still more rar occurs with typhoid in its earliest stages. Though typhoid may beg with severe bronchitis or pneumonic symptoms, doubt usually exists or during the first few days. The pneumonic type of typhoid, caused the localization of the typhoid bacilli in the lungs, is most uncomm The intestinal symptoms and cardinal findings of typhoid usua appear after a few days or one week, and all doubt is then dispell Pneumonia may undoubtedly occur without local findings, and yet t diagnosis of central pneumonia should rarely be made, as it is usua incorrect. Sudden onset with chill, pain in the side, sudden rise pulse and temperature, rapid respiration, bloody, viscid sputu herpes, icterus, marked leukocytosis, and usually signs of lo infiltration or a tympanitic note over one lobe and a few cre tant rales, followed later by some typical pneumonic infiltration the disease advances from within toward the periphery of the lu establish the diagnosis of pneumonia, usually on the second or th day.

Intestinal anthrax is also accompanied by fever, splenic tumor, a nervous symptoms. Hemorrhagic vesicles in the mouth, vomiti colic, hemorrhage from the intestines and kidney, cyanosis and dyspnœa, are suggestive, and the detection of the anthrax bacillus in the blood, urine, stools, or sputum, and the results of inoculation, establish the diagnosis.

Febrile gastritis may simulate typhoid, but the fever is usually low or irregular, and is more often absent than present. Marked early gastric symptoms are not common in typhoid. Febrile *enteritis* is usually characterized by much mucus in the stools. Though typhoid may occur with an old enterocolitis, the layers of the typhoid stool result from the lack of mucus. Splenic tumor has been observed occasionally in enteritis, but it is most uncommon. The fever in enteritis is lower or more irregular, than in typhoid; and the bronchitis, the slow pulse, and the other cardinal signs of typhoid, are absent. Cases of *typhoid developing suddenly* may be, at first, confused with grippe, diphtheria, or other diseases. Febrile *syphilis* should seldom cause diagnostic trouble.

Paratyphoid (paracolon infection) is due to a bacillus differing from the bacillus of typhoid and of dysentery, and from the Bacillus coli, and resembling the Bacillus enteritis, Bacillus psittacosis, etc., yet different from all of these. Two types of organisms are recognized—one producing a permanent and the other a transient reaction in milk. Infection may occur from milk, water, and tainted meat. The bacilli are found in the stools, blood, roseolæ, and urine, and agglutinate in higher dilutions (1 to 1,000 or 2,000) than the typhoid bacilli. The first cases were reported by Schottmueller, in 1900. In paratyphoid the temperature is more irregular than in typhoid, shows greater remissions, and may end with a crisis. The pulse is slow and dicrotic. Roseolæ are often present (80 per cent. of the cases), the spleen is usually enlarged, and no leukocytosis is observed. Prodromal catarrhs, diarrhœa, cervical adenitis, œdema of the lids, vomiting, epigastric pain, herpes, myositis, arthritis, pleurisy, endocarditis, and meningitis, are more common than in typhoid. The diazo test gives the characteristic reaction in 30 to 60 per cent. of the cases. The clinical course covers two weeks (twelve to eighty-four days). Relapses are common. The infection is usually mild, sometimes severe, and rarely fatal (3.6 per cent.). Ulceration of the intestinal lymph-structures is absent in the fatal cases, though other necroses occur, thus explaining the occasional hemorrhage (5 per cent.). Pneumonia and perforation are unknown. The disease is comparatively rare. The blood does not respond to the Widal test nor are typhoid-bacilli found, but otherwise it is indistinguishable from typhoid. Grunberg and Rolly hold that the Widal reaction is less a specific than a group reaction, as they found agglutination in the paratyphoid group by typhoid serum. This does not imply, however, that failure to respond to the Widal test excludes typhoid (Johnstone). A mixed infection with typhoid may occur.

The paracolon organism (recognized by Widal, in 1897) presents a serum reaction peculiar to itself when brought in contact with the blood of a patient suffering from the disease.

PATHOGNOMONIC SIGNS		PULSE AND CIRCULATION	FEVER	SPLENIC ENLARGEMENT	BLO
TYPHOID.	Typhoid bacilli in the blood, spleen (by puncture), urine, roseolæ, or stools. Widal test.	Relatively slow dicrotic pulse early in the disease, especially in men, and in not too severe infections. Endocarditis and pericarditis very rare.	Typical rise, acme, defervescence, and subnormal in convalecence. Remissions and intermissions following continuous fever speak for typhoid. Responds to hydrotherapy as a rule. Sometimes chills at onset or in course.	Very frequent; larger than in miliary tuberculosis; appears in first week.	Bacilli in early; Wi later. N cytosis unl plications, kopenia, morphonu decreased, anæmia.
MILIARY TUBERCULOSIS.	Tubercle bacilli in the blood or splenic pulp; tubercles in the choroid.	Rapid, 120 or more high even during remissions; slowed in tuberculous meningitis and in the aged. Dicrotism uncommon. Sometimes pericardial rub.	No cycle; irregular or remittent. Seldom long continued, and if so, longer than in typhoid. Exacerbations with renewed dissemination of tubercle bacilli. Brain involvement may lower fever or make it variable, as inversed type.	Less frequent, smaller and usually appears later.	Sometimes bacilli in bl Widal. Le sis not of more than phoid.
MALARIA.	Plasmodium in the blood (or splenic pulp).	Pulse moderate in rate; sometimes dicrotic; very rarely endocarditis.	Intermittent, beginning very often with chills, type varies (quotidian, tertian, etc.); often rises high at once; if continuous (subcontinuous) at least made irregular by quinine or usually wholly abolished (for a time at least).	Much enlarged; more than in typhoid; and persists frequently as ague-cake.	Plasmodiu anæmia, n cytosis sav nicious fo Great and anæmia. N
SEPSIS (including ENDOCARDITIS).	Cultivation of pyogenic organisms, pneumococcus, from the blood or various foci.	Usually fast and irregular; cardiac localizations very frequent in sepsis, puerperal especially, and in endocarditis (a cardiac form of sepsis). Cardiac enlargement, thrill, bruit, accentuated pulmonic tone, etc.	Chills, sweats, steep elevations, and sudden depressions almost always at some time, and usually repeated with each discharge into the blood of infective material.	Especially enlarged from sepsis and infarcts. Also often the lymph-nodes.	Various or Marked p phonuclear cyotsis ve quent; so absent in ditis. No
MENINGITIS.	Lumbar puncture (*a*) meningococci, tubercle bacilli, pyogenic organisms, etc.; (*b*) increased, turbid fluid, etc.	Rate differs with type; variable.	Differs with variety, but seldom as high as typhoid; no cycle.	Rare.	Leukocyto idemic an lent types; tuberculou Widal.

SKIN	LUNGS	NERVOUS SYSTEM	SPUTUM, URINE	ALIMENTARY TRACT
Roseolæ, typical in location, crops, appearance, containing typhoid bacilli. Sweating and miliaria less often and later. Icterus rare Herpes rare.	Breathing somewhat increased from toxæmia (cerebral breathing) hypostasis, or other complications. Rales below and behind. Tympanitic note never heard in miliary tuberculosis.	Early headache, replaced later by delirium, typhoid state, actual meningitis very rare; intoxication may closely simulate meningitis. Very rarely disk changes.	Typhoid bacilli in urine. Diazo test in nearly all cases.	Mouse-like breath, semi-characteristic tongue, ochre-yellow stools, tympany, diarrhœa, bowel hemorrhage, perforation, bacilli in stools. Retracted abdomen or early vomiting rare.
Roseolæ absent; extremely rare, and not then in crops. Drenching sweats common. Herpes more than in typhoid. Skin anæmic and very often cyanotic.	Breathing increased even 40 to 60; cyanosis and dyspnœa without adequate physical findings. Fine and localized rales heard above and in front. Tympanitic percussion note from lung relaxation. Evidences of pleural adhesions, apical changes, fresh pleural rub.	Meningitis tuberculosa very frequent; optic neuritis; choroidal tubercles.	Tubercle bacilli sometimes found in sputum, urine and fæces, but are indicative of old tuberculous lesions, not of miliary tuberculosis. Diazo test frequent.	Diarrhœa not common, but may rarely occur, as indeed may hemorrhages; both may come from tuberculous ulceration of bowels.
In severe, eyes subicteric or markedly icteric. Herpes very frequent and urticaria the next most frequent exanthem.	If lung congestion (severe forms), it is variable, recurrent, shifts from lobe to lobe.	Headache at onset is rare; later is often pulsating or neuralgic. Delirium from beginning in aestivo-autumnal forms is frequent. Restlessness, anxiousness. In pernicious forms may have convulsions, coma, etc.	Hæmoglobinuria in pernicious malaria. Diazo test fairly frequent, though much less than in typhoid.	Nauseating breath. Sometimes abdominal pain even in mild types; may rarely have diarrhœa, even hemorrhage in alimentary pernicious types. Liver congestion frequent, also icterus.
Frequent sweating, miliaria. Septic (polymorphous) rashes Subicterus, frequently purpuric skin eruptions, herpes not uncommon.	Bronchitis frequent. Pulmonary infarcts and pleurisy.	Dependent on cause. Septic meningitis (with arthritis, pleurisy, etc.) cerebral embolism; retinal hemorrhages or embolism.	Acute embolic nephritis. Peptonuria.	Septic diarrhœa.
Herpes in epidemic cerebrospinal form. Tache cérébrale.	Variable breathing; Biot's breathing.	Early triad, headache retracted neck and hyperæsthesia. Headache violent throughout the disease, convulsions, paralysis, Kernig's sign, etc. Optic neuritis retinal hemorrhage, tubercles in choroid.		Early cerebral vomiting. Retracted scaphoid abdomen.

Trichinosis is differentiated from typhoid by the history, by muscu lar pain and œdema in the striped muscles, by trichinæ in the stoo and muscles, and by eosinophilia. In typhoid these symptoms a absent, while the cardinal findings, such as the Widal reaction, ro spots, and enlargement of spleen, are present. Two or three cases double infections have been reported (McCrae).

Prognosis of Typhoid Fever.—The death-rate was formerly per cent., based on 50,000 cases collected by Jaccoud. At present it lower (5 to 12 per cent.) because of: (*a*) more accurate diagnosi thus including lighter cases in our figures; (*b*) the virulence of typho becoming less; (*c*) better hygiene; and (*d*) better treatment, t Brand method, etc.

The *prognostic factors* are:

1. Age: The least mortality is between the second and the ten years; the highest after the twenty-fifth.
2. Constitution: Meagre and muscular patients are more likely recover than the plethoric, adipose, or robust.
3. Sex is immaterial, aside from pregnancy.
4. Acute diseases modify the prognosis. These are scarlatin measles, variola, diphtheria (rare), malaria, erysipelas (from 0.7 to per cent.), varicella, etc.
5. Chronic diseases, such as gout, diabetes (low temperature, t sugar often disappearing), cardiac, pulmonary, and renal disea alcoholism (34 per cent. mortality from weak heart or hemorrha diathesis); convulsive affections, such as epilepsy or chorea, may cea

The prognosis is better in relapses than in the original ca Recrudescences are dangerous. In war times, the prognosis is usua less favorable; the Spanish-American war was an exception, t death-rate being but 7 per cent., while in the Boer war the mortal was 21 per cent.

Death usually occurs between the eighteenth and twenty-eigh days, and is caused by—

1. *Toxæmia*, the most frequent cause of death (30 to 50 per cen including: (*a*) hyperpyrexia; (*b*) hemorrhagic types; (*c*) heart wea ness, vasomotor paralysis; and (*d*) brain paralysis.
2. *Local typhoid lesions*, as meteorism, diarrhœa, peritonitis (in per cent. of fatal cases), or hemorrhage (in 20 per cent.).
3. *Complications*, as lung inflammations (in 10 per cent. to 15 cent.), nephritis (4.5 per cent.). Septicæmia (secondary streptococ invasion) has been little studied except by French writers ("*for septicémique généralisée*"), the symptoms of which are high fever, w chills and sweats, abscesses, enlarged glands and spleen. Pyæmia m result from acute osteomyelitis, suppurative parotitis, adenitis, ph bitis, or splenic infarction.
4. *Collapse*. *Sudden death* has been discussed under changes the heart muscle. Dieulafoy estimates that sudden death occurs i per cent. of typhoid cases. The pulse is always more important th the fever, and early rapidity is unfavorable.

5. We have already considered the ominous significance of pseudo-collapse, the typhoid state, early delirium, diarrhœa, and meteorism; of the various severe complications, such as noma, gangrene, laryngeal perichondritis, and pneumonia; and of the more severe types, such as the hyperpyretic and hemorrhagic; and of the ambulatory varieties. Typhoid beginning suddenly entails a high mortality.

Treatment.—Dujardin-Beaumetz said "The best treatment for typhoid is a good physician." The treatment concerns (*A*) prophylaxis; (*B*) specific treatment; (*C*) hygiene and rest; (*D*) the question of intestinal antisepsis; (*E*) antipyretic measures (hydrotherapy); (*F*) diet; (*G*) symptomatic or expectant treatment; (*H*) treatment during convalescence.

(*A*). **Prophylaxis.**—Prophylaxis has as wide a scope in medicine as asepsis and antisepsis in surgery. In Chicago, during the years 1889 to 1892, inclusive, there were 4,747 deaths from typhoid. In the four years after the drainage-canal was opened there were 2,235 cases; that is, per population, the mortality was reduced 75 per cent. In 1905, typhoid cases were rare. General prevention is difficult, because of the ubiquity of the germ and the carelessness of individuals. Systematic coöperation regarding sewage regulation and water supply could efface typhoid. Koch has particularly emphasized this point lately and succeeded in wiping out certain typhoid foci. One difficulty lies in the infrequent recognition of light cases, and another in the selfish carelessness of many persons, and a third in the fact that some individuals "carry bacilli" for years. It is easy to boil water for home use, and yet it is done in but few families, even in those of physicians. The traveler should avoid unboiled water and milk. In the treatment of a case, everything with which the germ may come in contact must be sterilized. Next to the fæces, the urine is the most important conveyer of the bacillus. They are the usual means of dissemination, and their proper disinfection in every case would surely obliterate typhoid. The bacilli may persist for years in the urine. Chloride of lime should be placed in the bottom of the urinal, or bed-pan, before and after use. Acidulated sublimate (1 to 500) or 10-per-cent. crude carbolic solution may also be used; but neither at once disinfects, for which reason each one of the dejections should be disposed of only after an hour or two. Both the pans and urinals must then be sterilized. The same urgent disinfection is indicated in the case of thermometers used in the rectum, and rectal syringes or tubes. The perineum, after each movement, should be sponged with a 1-to-2,000 bichloride solution, and also the back and thighs, when there are involuntary evacuations. The rubber draw-sheets and linen should be sterilized regularly with carbolic acid or bichloride of mercury, and the sheets, blankets, pillows, and mattresses, steamed when taken off the bed. The bath-tub should be carefully cleaned with carbolic solution, as the patient's soiled skin and the urine often voided during the bath are obvious sources of danger. It seems superfluous to urge cleaning the hands of the nurse or physician after examination or tubbing, but the not infrequent direct transmission of the disease to nurses, orderlies, and doctors, is often forgotte Direct infection is growing in clinical importance. In the Spanis American war 20 per cent. of our soldiers were directly infect by flies, and in the Boer war more died of typhoid than were killed bullets, infection being carried by flies and sand. The few cas among the Japanese is one of the many lessons they taught us.

In convalescence the room should be fumigated with formaldehyd not with the useless sulphur method (see SMALLPOX).

(*B*). **Specific Treatment.**—Specific treatment awaits the further dev opment of serotherapy. Chantemesse, Widal, Brieger, Wasserman Wright, and others, have obtained some results. The injecti of a sterilized culture sufficiently strong to kill a guinea-pig pr duces local reaction, sometimes nausea, restlessness, fever, increas bactericidal power of the blood, and an increase in its agglutinati properties. Preventive inoculation in India and South Africa reduc the percentages of typhoid morbidity from 14 to 2 per cent., and t mortality from 3 to ½ per cent. The injections used by Chantemes gave him a mortality of but 4 per cent., while the death-rate at th time was 12 to 18 per cent. in the other Paris hospitals.

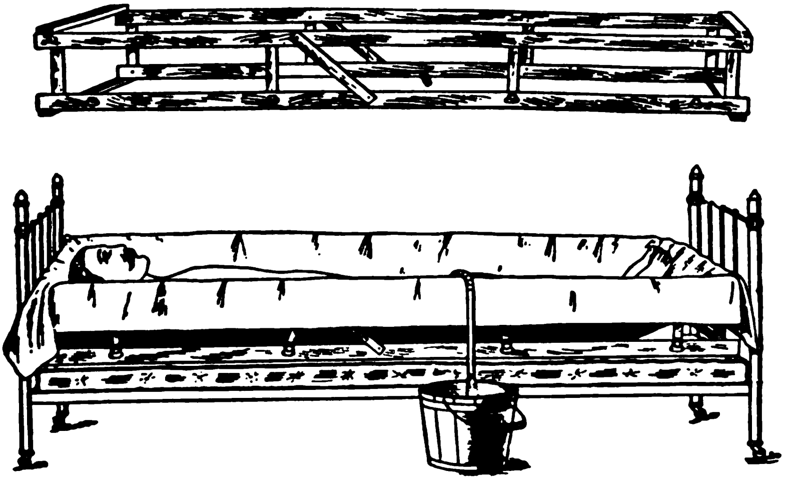
(*C*). **Hygienic Treatment.**—Absolute physical and psychical r is imperative, and the best results occur in cases which have been p to bed early. Cases of the ambulatory type, and the ones which f any reason are not put to bed at the onset of the typhoid, never fa as well. Typhoid patients should not be entertained. The invaria use of the bed-pan and urinal should be insisted upon, as every p tient can learn to use them. In all infections, the furniture, carpe and hangings, should be removed from the room, and it should ha free ventilation,—fever patients do not catch cold. The sunlight sho fall across the bed in order to avoid confusing shadows. The nu should have written instructions. The stools, baths, etc., should watched or directed by the physician, especially at the onset. T single bed should be low, should have a firm mattress, rubber dra sheet, and perfectly smooth cotton or linen sheets. A second b is desirable for change and convenience.

(*D*). **Antiseptic Therapy.**—Antiseptic therapy is now largely of h torical interest, as typhoid is not a local enteric disease but a sep cæmia or bacillæmia. Calomel was given early in typhoid by v Ziemssen and Liebermeister, who thought that it reduced the vi lence of the attack and lessened bowel symptoms and complicatio it is unnecessary, and at the acme is actually harmful. Beta-naphth grs. v. to x. every four hours, is given by Bouchard and Rossba Salol (phenylis salicylas) is the least objectionable, since it is mi it is given in doses of grs. x., t. i. d., and has some value as an int tinal antiseptic. The urine should be closely watched for signs renal irritation. A few drops of hydrochloric acid (the Swedish fev cure), bichloride of mercury, one-thirtieth grain every eight ho (Broadbent), or thymol, two grains every three hours in a soap p are also advised. No one of this group of agents essentially fluences the clinical course. They are used not against the Bacillus typhosus but solely against the common organisms of fermentation.

(*E*). **Antipyresis.**—(1). DRUG ANTIPYRETICS.—Drug antipyretics in typhoid are not often indicated, for they merely reduce fever and do not eliminate its cause, the toxins. Quinine is recommended by Liebermeister and Curschmann, in one or two doses of fifteen to twenty grains, given before the fever reaches its highest point. No cardiac depression results, and its effects last from eight to twelve hours; the fever, however, is but moderately influenced by quinine. Antipyrin and allied products, in full repeated doses, depress the brain, circulation, respiration, and metabolism; cause vomiting and eruptions; and increase the danger of collapse. They are not indicated in typhoid as they are in the fugitive fevers. Kryofine, seven to fifteen grains, is a good febrifuge, and in the author's experience has never caused depression. In practically all cases, drug antipyresis is far inferior to—

(2). HYDROTHERAPY, which is the only logical method of reducy ing temperature, since it alone eliminates the fever-exciting toxins. Nature's method of reducing temperature by sweating should not be ignored. Evaporation from the moist skin should be helped by removal of the bed-clothing. (*a*) The *full cold bath* of Currie (1787) and Brand is given with water at 64° to 68°; it lasts fifteen to twenty minutes and is repeated every three hours for temperature of 102° or over. Chilling is averted by vigorous friction, which must not abrade the skin; by cold affusions to the head; or by administration of alcoholic stimulants. The apparent brutality of the Brand method has excited considerable popular and professional prejudice, but it is more valuable than any other measure in typhoid. As Jür-

FIG. 6.



Burr's bed.

gensen remarks, every deviation from the simple principles above outlined gives less satisfactory results. Early resort to hydrotherapy gives the best results. When the portable bath-tub is not available, the method of A. H. Burr is equally good. He uses a wooden frame, shaped like a large box; the base fits over any bed; its upper part sustains a heavy rubber sheet, which passes under the patient and constitutes the sides and bottom of the tub (Fig. 6). Its use obviates the necessity of lifting the patient, and saves excitement. Water is poured over the patient from pails and is removed with a siphon. The *advantages* of Brand's method are: (i) The mortality is lowered 6 to 8 per cent.; (ii) toxæmia is lessened, and therefore nervous and digestive symptoms, heart weakness, and pulmonary complications, are relieved; fever is successfully reduced, and there is less visceral degeneration; (iii) the renal secretion is stimulated, thereby washing the toxins out of the blood, as is shown by a five- or sixfold increase in the urotoxic coefficient; (iv) the skin becomes more active and bed-sores less frequent, although furunculosis is seemingly increased; (v) the clinical course is shorter, and, on the whole, fewer complications occur, although the percentage of hemorrhage and perforation is not lowered, and relapses seem slightly more frequent. *Contra-indications* to the use of Brand's method are hemorrhage, perforation, great prostration, alcoholism, advanced arteriosclerosis or cardiac disease, and marked phlebitis. Pregnancy, nephritis, and pneumonia, are not contra-indications. Various *substitutes*, for Brand's method, though less effectual, may be indicated for special reasons. (*b*) The *gradually cooled bath* (Ziemssen and Immermann) is identical with the Brand method, except that the temperature of the water is gradually reduced from 90° to 75° or 65°. It is adapted to cases coming under treatment late and to those with great dyspnœa, or to those tending to collapse (*c*) The *protracted tepid bath* (Riess, Eichhorst, Unverricht). (*d*) *Cold sponging*, first with warm water to dilate the skin-vessels and then with cold water to abstract heat. Equal parts of alcohol and water should be mixed, to aid evaporation. One part after another o the body is exposed and sponged. It is a refreshing and quieting placebo, but enormously inferior as an antipyretic. (*e*) The *cold pack* is also much less successful. In sthenic cases, ice applications to the head, axilla, spine, etc., may be used, or the body may even be ironed with ice. Ice-water enemas are never used in typhoid, though sometimes employed in other fevers.

(*F*). **Diet.**—Fifty ounces of water should be given daily at definit intervals, without waiting for the stuporous patient to complain o thirst. This flushes the kidneys, lowers temperature by increasing th sweat production, and lessens parenchymatous degeneration of th viscera. Pure water is superior to barley- or albumin-water, lemonade etc., which dull the appetite and often derange digestion. Care o the mouth may be considered here. The mouth and pharynx shoul be carefully cleansed of sordes and food particles, lest infection trave to the ears, salivary and lymphatic glands, or aspiration pneumoni result. A boric-acid solution, with tincture of myrrh or tincture of hydrastis, meets every requirement. The cleansing must be gentle, in order that no fresh abrasions may be inflicted. The diet must be fluid, since there are intestinal lesions. It must also be easily digestible, since fever lessens all the digestive juices. In the diet no reference is usually made to individual symptoms, as there is no correspondence between the clinical symptoms and the actual anatomical ulceration of the intestines. Graves was the first to feed fever patients, who up to that time had been starved, but there is, at the present time, almost equal danger of overfeeding. Milk is the standard diet. Six to eight ounces should be sipped at intervals of three hours, day and night, as the patient, after being wakened, readily falls to sleep again. It is best given diluted with half an ounce of lime-water or with a little carbonated water, which should not be cold, since the gas expands at body heat. Though milk alone is said to contain insufficient albumin to meet the tissue-waste, there is risk of overfeeding; and the physician himself should inspect the stools for curds, for milk, curdling in the stomach, practically becomes solid food. One pint of milk is equivalent to one pint of oysters or a good chop. Very few patients cannot take milk. In other words, it can be forced upon unwilling patients. Recalcitrant patients may be given some variation, such as koumyss, matzoon, buttermilk, whey, or certain baby-foods. If the digestion is peculiar or weak, peptonized milk, given with very thin, strained gruels, or egg, may be tolerated. Carbohydrates are theoretically indicated to lessen nitrogenous waste, and well-boiled cornstarch and tapioca are allowed by some clinicians. They are permissible if no tympany results, and the author agrees with Hare that taka-diastase aids their assimilation. Meat-juice is obtained from freshly slaughtered beef, by a meat-press. If its taste is disagreeable to the patient, a few drops of peppermint oil may be given first. It may also be given frozen (meat-juice, ʒ iij; sugar, ʒ ij.; lemon-juice, ʒ j.; yolk of one egg; cognac, ʒ j.). The following articles afford some variety: Nutrose, somatose, eukasin, Valentine's meat-juice, Brand's essence of beef, Mosquera's predigested beef-jelly (containing pineapple juice and over 50 per cent. soluble albumin); yolk of egg in soup or wine, and white of egg with twice its weight of water. Foods containing gelatin conserve metabolism (Senator) but may irritate the bowels. Beef-tea should be avoided, as it contains only stimulating substances, less than 1 per cent. albumin, and no fat or carbohydrates. Furthermore, it often irritates the kidneys and intestines. In soporose patients with anæsthetic throats, food may enter the larynx and cause aspiration pneumonia. The nasal catheter is then indicated. To its outer end a funnel is attached, through which food, drink, and medicines, are given. A safety-pin across its outer end prevents its escape downward. The catheter should be long enough to pass into the œsophagus beyond the larynx, and care should be exercised that it does not enter the latter. Rectal feeding is sometimes indicated.

(*G*). **Symptomatic or Expectant Treatment.**—1. DIGESTIVE SYSTEM. (*a*) *Vomiting.*—It must be ascertained that the patient has been given no solid food by solicitous friends and that perforation has not taken place. Rectal feeding, the use of warm carbonated water, cocaine gr. ¼, or tincture iodine ♏ ½, for several doses, sinapisms or an ice-bag to be placed on the epigastrium, are indicated. Bartholow's formula is very serviceable:

℞

Acidi carbolici	gr. iv.
Bismuthi subnitratis	ʒiss.
Cerii oxalatis	ʒj.
Sodii bromidi	ʒss.
Sacchari albi Acaciæ	āā q.s.
Aquæ menthæ piperitæ	q.s. ad ℥ij.

M. et ft. emulsio.
S.—One teaspoonful, undiluted, every 15 to 30 minutes.

(*b*) *Constipation* should be treated with enemas of water or sweet oil every other day. Typhoid cases with constipation do better than those with diarrhœa. Cathartics are never used, as they cause meteorism. (*c*) *Diarrhœa* is most frequently due to toxæmia and is, therefore, most efficaciously treated with the cold bath; curds (prevented by diluting the milk), beef-tea, or solid food, may be its cause. Treatment is indicated when there are more than four movements daily, and when there is colic or tenesmus, unrest or weakness. Cold tea with whiskey and pulv. aromaticus gr. v., or paregoric and bismuth each ʒj., after each movement, extr. opii. gr. i., or the starch-and-laudanum enema usually give relief at once. Pepper considered silver nitrate almost specific (gr. ¼ to ½).

℞

Bismuthi subnitratis	ʒj.
Morphinæ sulphatis	gr. j.

M. et div. in pulv. xij.
S.—One to four a day. (Alonzo Clark).

℞

Acidi tannici	gr. x.
Tincturæ opii	♏vj.
Spiritus terebinthinæ	♏xv.
Mucilaginis	ʒij.
Tincturæ chloroformi compositæ	♏xx.
Aquæ menthæ piperitæ	ad ℥j.

M. f. haust.
S.—To be taken every two hours. (Murchison).

℞

Plumbi acetatis	gr. iss ad iij.
Opii pulveris	gr. i ad ijss.
Sacchari albi	gr. lx.

M. et divide in pulv. vj.
S.—A powder every three hours. (Yeo).

(*d*) *Tympany.*—This is sometimes an indication that the patient has been given solid food. In severe cases albumin-water must be substituted for milk. The cold bath often gives relief, since most cases of meteorism are due to toxæmia. The turpentine stupe of Jenner should be applied to the abdomen, flannel cloths being dipped in a mixture of warm water and ʒss ol. terebinthinæ and pressed hard together to spread the oil evenly and thus avoid blistering. The ice-coil is also beneficial. The old turpentine emulsion of Wood (ol. terebinthinæ, ♏ iij. to v.) is excellent, though the urine must be watched carefully for signs of renal irritation:

℞

Ol. terebinthinæ	ʒiss.
Ol. caryophylli	gtt. vj.
Glycerini Mucilaginis acaciæ	āā ℥ss.
Syrupi et aquæ	āā ℥iss.

M. et ft. emulsio.
S.—A dessert spoonful every 3 or 4 hours.

Whiskey and spices are effective. Emulsio asafœtidæ, Oj; Spr. chloroformi, ʒj.; and ol. terebinthinæ, ʒss; as a warm enema, often give relief. (*e*) *Hemorrhage* necessitates absolute quiet, mental, physical, and alimentary. The patient should not be moved for any reason whatever. In severe cases he should lie in the blood which has been passed, with cotton tucked around the hips and between the legs to catch the urine and fæces, which should pass into it without his being lifted on the bed-pan. No food, no water, no enema, no bath, should be given. Morphine is primarily indicated. It quiets the excitement attending hemorrhage, lessens peristalsis and enforces bodily quiet. It should be given in sufficiently large amounts; gr. ¼ being injected every half-hour for three doses, and then a quantity sufficiently large to produce its full physiological effects, for the dose of a drug is not measured in grains but in its effect; it should be repeated every two, four, or six hours in order to sustain the primary indications above named. The author has given even four to six grains in the first day, when the greatest danger occurs, but in such cases the patient must be very closely watched. The author considers that the sole valid objection to morphine is that it may mask coincident perforation, which occurs once in every five cases of hemorrhage; he believes, however, that close circumspection invalidates this objection. Opium or styptics by the mouth are dangerous and uncertain. The lead- or iron-salts probably never reach the bowel. The ice-coil placed on the abdomen may be beneficial. Gelatin may be injected as in aneurysm (*q. v.*). The question of stimulation is a difficult one. On the one hand, there is the risk of forcing blood through the leak by stimulation; on the other hand, the volume of blood must not sink too low; but first the heart must be allowed to run down,—nature's own and most certain method of hemostasis. At a later time only, should strychnine or digitalis be administered hypodermically, or saline transfusions be given; and delay in their use is better than haste. (*f*) *Perforation* necessitates an early diagnosis, which is often difficult, and immediate operation, often without general anæsthesia. Most reported recoveries were operated on in the first twenty-four hours.

Haggard, Cazin, and Zezas, collated the operations up to 1904–5. Recovery occurred in 30 per cent. of 358 operations (Cazin). In twenty cases operated on at Johns Hopkins Hospital, 35 per cent. recovered.

2. Circulatory System.—Heart weakness is best prevented by absolute rest, and hydrotherapy. An ice-bag placed over the heart tends to regulate and calm its action. Strychnine, gr. $\frac{1}{30}$, is the best heart tonic, and may be given two or three times a day as a vasomotor tonic in cases which do not show signs of heart-failure. It is best, however, to await definite indications for stimulation. Digitalis is rarely beneficial in fever cases, and may produce urgent signs of cumulative action if the fever should fall. The author has seen one case in which, with rather sudden defervescence, the pulse dropped from 140 to 40. Camphor, gr. ij., disagrees when given by the mouth and should be given under the skin in 1 to 10 parts of sterilized olive oil. Other cardiants are considered under valvular disease and pneumonia. Sudden dilatation should be treated by venesection and stimulation. Alcohol may be considered under this head. In typhoid i may be indicated (*a*) as a food, with milk and eggs, thus aiding digestion: ʒj. to iv. of whiskey may be given every two to four hours i hot milk. Stokes's mixture contained cognac, ℥ij.; Vitelli ovi., one Syr. aurantii, ʒv.; water q. s. ad ℥v. Eggnog is valuable as a foo which may be readily absorbed. (*b*) As a cardiant (though pharmacologists hold that pure alcohol is not a cardiant), Meltzer state that it equalizes the circulation. It is indicated especially in youn alcoholics, and in most cases over forty years of age. Cognac may b given by the mouth, in coffee; or by rectum, in peptonized mil with two drachms of tincture of valerian. (*c*) As a nervine in debilit or old age, as whiskey, champagne (especially for vomiting), sherry or port. (*d*) To a less degree as an antipyretic, since it increases th sweat production. Its use must be suspended when the pulse become bounding, the respiration hurried, the skin or tongue dry, and whe there is restlessness, or the odor of alcohol appears on the breath Like other good things, alcohol has been abused, and of late the re action against its use is in part sound and in part excessive. Wit care and discretion alcohol is invaluable in severe cases.

Thrombophlebitis is treated by quiet, elevation, warmth, and ban daging from the toes upward.

3. Respiratory System.—Epistaxis usually ceases spontaneousl If the blood runs into the throat a tampon should be introduce Laryngeal complications may necessitate early tracheotomy. Onl 2 per cent. of severe laryngeal complications recover without opera tion. Bronchitis is treated most efficaciously by hydrotherapy, c sometimes, though rarely, by codeine. Expectorants should alwa be avoided since they are likely to produce gastric disorder. Hyp stasis is best treated by hydrotherapy, cardiants, and change of po ture every two hours, for which written orders should be given.

4. Nervous System.—The initial headache may be relieved by h drotherapy, the ice-cap, ergotin and antipyrin, each gr. v., for sever

doses. Depression is relieved by valerian and whiskey. For delirium, and insomnia, hydrotherapy is the most useful measure; veronal, gr. v.–vij. in warm water, produces a very natural sleep; alcohol; morphia, gr. $\frac{1}{4}$; codeine, gr. $\frac{1}{2}$; sodium bromide, ʒss to j.; and hyoscine hydrobromate, gr. $\frac{1}{150}$ to $\frac{1}{50}$; may be indicated.

℞

Morphinæ hydrochloridi	gr. $\frac{1}{4}$.
Sodii bromidi	gr. xxxxv.
Syrupi aurantii	ʒiij.
Aquæ chloroformi	ad ℥ij.

M. f. mist.
S.—Half to be taken at bed-time, and the remainder in three hours, if necessary. (Whitla).

Typhoid patients should never be left alone, lest they leave the bed or leap from the window. Restlessness may suggest a full bladder.

5. The Skin.—Massage with alcohol or olive oil is useful. The skin functions are helped by the full bath. In the formation of bed-sores the following are factors: Local heat, pressure, moisture, maceration, or filth. All of these can generally be avoided by frequent changes of posture, thick pads or rubber rings, and cleanliness. In some cases great debility may frustrate prophylaxis. The least erythema, papule, abrasion, or pustule, on dependent parts is a matter of solicitude. If bed-sores have already developed, the simplest treatment is the best, since antiseptics and powders sometimes only aggravate. The balsam of Peru is excellent. Collodion and adhesive straps are always to be avoided as far as possible.

6. Bacilluria.—Bacilluria is met by salol and urotropin, each gr. x., t. i. d. Special care is necessary in the use of catheters.

(*H*). **Treatment in Convalescence.**—The convalescence must be actual, and the possibility of relapses must be excluded before the patient can have substantial diet or be allowed to sit up. It is best to err on the safe side, as embolism and collapse are not unknown in convalescence. Eggs, toast, gruels, rice, etc., may be given after one week of normal temperature. The patient should not leave his bed for two weeks. Irregular fever lingers in some cases until solid food is given. In other cases, nitrogenous food will cause a higher temperature (*febris carnis*). Vegetables and fruits should be postponed for a few weeks. An excellent tonic in convalescence is the following:

℞

Strychninæ sulphatis	gr. j.
Acidi nitrohydrochlorici	ʒj.
Tr. gentianæ co	℥v.

M. et S.—One teaspoonful after meals in half a glass of water.

TYPHUS FEVER

Synonyms.—Typhus fever; exanthematous typhus; war, prison, ship, or famine fever.

Definition.—An acute, highly contagious, specific infection of unknown causation; mostly epidemic, with a sudden onset and a high temperature, falling by crisis, and with a cyclic curve, a characteristic eruption, and severe nervous symptoms. Typhus, first described by Corradi (1083), was first clearly distinguished by Fracostorius, in Verona (1505).

Etiology.—All stages of the disease are highly contagious. The virus, which is as yet unknown, exists in the exhalations from the skin and lungs (Murchison), and survives for a long time in clothing and furniture. Infection is usually direct from person to person. The danger to attending nurses and physicians is therefore great, especially i severe epidemics. The more intimate the contact the greater is th danger of direct infection. It is also communicable indirectly by third person, by means of fomites, etc. The point of entrance to th system is probably the lungs. Cases appear from time to time, th history of which cannot be traced. Immunity is usually conferred b one attack, but may be congenital. Susceptibility to the disease i general.

Geography.—Ireland, Russia, Galatia, Hungary, Italy, and Mexico are its most frequent homes, and there are always cases in China Asia Minor, and Hungary. It is endemic in Berlin, occurring amon the lower classes, among vagabonds, and in the unclean, poorly-fed closely-packed and unhygienic population. Typhus was one of th greatest scourges of the middle ages, with its wars and famines, bu it has decreased in the past century, the epidemics in Ireland (181 and 1846) and in the Turko-Russian war (1877–78) being the las general outbreaks. Its most recent occurrence in this country wa in New York (1881–82 and 1893), in Philadelphia (1883), and i Baltimore in 1901 (Osler).

Age.—Most cases occur between the fifteenth and twenty-fifth year It is rare in the young or aged, though it has been observed before th first year and after the eightieth. It may occur with other epidemic such as typhoid, recurrent fever, malaria, and smallpox. The ba teriology of typhus is undetermined. A streptococcus was found b Hlava (1891) and a diplococcus by Dubieff and Bruhl (1893).

General Clinical Picture.—1. **Incubation.**—The incubation las twelve days (seven to fourteen), or less in children, and is usual unaccompanied by symptoms.

2. **The Stage of Invasion.**—The stage of invasion is *sudden*, e hibiting one or more chills and a high temperature on the first da The patient therefore comes early under treatment. Thirst follow with anorexia, coated tongue, nausea, vomiting, epigastric oppressio suffusion of the face, conjunctivæ, nose, and pharynx; dysphagi apathy, delirium, epistaxis, backache, severe pains in the extremitie rapid pulse, and early splenic intumescence.

3. **The Exanthematous Stage.**—The exanthematous stage is in cated on the third or fifth day by a roseolous eruption, at first pal red and purely hyperæmic. It is seen first on the abdomen, a

chest, extending rapidly to the back and extremities. The eruption then develops a petechial character, with some serous infiltration in the centre or edge of the flecks, which become dirty and copper-colored. It lasts seven to ten days. In this stage there is no pain, but often apathy, prostration, delirium or mania. The tongue and mouth are dry and dark. Catarrhal inflammations of the pharynx, larynx and bronchi develop, with hoarseness and cough. The fever is continuous. Coma-vigil, rapid respiration and pulse, and hypostatic pneumonia may end the case.

4. **Defervescence.**—The crisis usually occurs on the tenth to the fourteenth day. The defervescence may occupy two or three days (lysis). The pulse becomes slower; the skin often desquamates in dusky scales; profuse sweats are common; the tongue clears, and convalescence is established in from twelve to seventeen days. Long defervescence is suggestive of complications.

Special Symptomatology.—1. **Fever.**—After a rigor, the temperature rapidly rises to 103°, 105°, or even 107°, on the first or second day (Wunderlich). No other acute exanthematous disease produces an equally high early temperature. Remissions are few and slight in the first week, and the maximum temperature is observed on the fifth day. On the tenth day the temperature often remits, except in the most severe cases. The crisis occurs from the tenth to the fourteenth day, with sweating, exhaustion, relief of symptoms, and often with subnormal temperature. Curschmann considers that lysis is as frequent as crisis. Remissions rarely occur in this disease. Afebrile typhus is rare. So also are precritical elevations and pseudo-crises. Death occurs either with a preagonal rise or with collapse of temperature.

2. **The Skin.**—The eruption consists of two elements: Roseolæ changing to petechiæ, and a fine, dusky-red mottling under the skin. The *roseolæ* are seen on the third or the fifth day; they increase rapidly and abundantly, though never in recurrent crops, and are at their height within two days. They are located chiefly on the lower abdomen, chest, back or shoulders, are least abundant on the thighs, and do not appear on the face except in women and children whose skin is delicate. The palms of the hands and the soles of the feet are generally exempt.

The roseolæ are pale-red, washed in outline, round or oval, the size of a pin-head or a lentil. They are hyperæmic, and therefore disappear on pressure and after death. They are easily overlooked at night. They last one, two, or at the most three days, later becoming dark, livid, or copper-colored *petechiæ* persisting on pressure and after death, because hemorrhagic. The petechiæ occur mostly on the back, or in the folds of the groin, and may lie between the roseolæ. The hemorrhage may extend more widely either into the skin or in the subcutaneous tissue. Typhus cases presenting no rash are rare. A great multiplicity of petechiæ is an ominous feature, especially in cachectic and alcoholic subjects. A morbilliform eruption on the arms and legs is occasionally observed before the usual typhus rash, especially in thin-skinned individuals. The skin is usually dry, but miliaria may appear (in 6 to 10 per cent. of the cases), notably in the young. Icterus without decolorization of the stools is frequent and is an unfavorable sign. Desquamation occurs, and is sometimes as profuse as in scarlatina; herpes occurs in 5 to 20 per cent., and bedsores in 3 per cent. of the cases. The skin has a peculiarly pungent, offensive, or ammoniacal odor.

3. The Nervous System.—This is markedly involved, toxæmia being profound. Headache is constant until the exanthem appears; it is frontal and temporal, infrequently occipital, and radiates into the neck or shoulders. It is associated with vertigo, backache, and hyperæsthesia of the fingers and toes. The sensorium first shows depression, characterized by apathy, insomnia, incoherent speech, or disturbing dreams, which increase as the pains disappear. In the second week coma-vigil, low delirium, mania and hallucinations often develop. Suicidal tendencies may be noted, especially in the young and robust. Involuntary evacuations and noisy cerebral respiration also occur. The earlier the nervous symptoms, the more ominous is their significance. Neuralgia is occasional in defervescence. It is transient, and exists mostly in the lower extremities. Tremor of the forearms and hands is almost constant. Convulsions are rare, but ominous. They are observed chiefly at the end of the second week in alcoholic or very irritable subjects and in children. They are more often epileptiform than partial, tetanic, or trismic. Paralysis occurs from brai hemorrhage, embolism, etc. Cases of paraplegia and aphasia ar recorded. Meningitis is most rare.

Special Senses.—There is conjunctival catarrh and the "pin-hea pupil" of Graves. The ears are involved in 32 per cent. of th cases.

4. Circulation.—The heart and vasomotor system suffer greatl (Stokes). The pulse, which is of greatest prognostic value, is smal and greatly increased, reaching 100 to 120, and even higher in wome and children. It is slow only in light cases. Dicrotism is rare. It i labile and often slow in convalescence. Acute myocarditis may occu with acute dilatation, which is highly dangerous, especially in alco holics. Its symptoms are those described under Typhoid. In fat cases the heart muscle is granular and deep-red. The blood is dar and fluid; the red cells and hæmoglobin are decreased, and the whit cells are increased.

5. Respiration.—Epistaxis is occasional. Laryngeal involvemen varies with the epidemic. Hoarseness, erosions, fissures, ulceration œdema, and perichondritis, may develop and may possibly lead t secondary abscess of the lung or empyema. Catarrh of the upp air-passages, bronchitis, and tracheitis, are part of the disease. The are present at the onset, increase with the fever, and are evidenced b a cough, with a slight expectoration, which is sometimes streake with blood. Catarrh with weak heart results in atelectasis, hypostasi and lobular pneumonia.

Pneumonia occurs in 15 per cent. of typhus cases, usually in the second week, and is frequently atypical.

6. **Gastro-Intestinal Tract.**—The tongue, at first moist and white, becomes dry and dark—the "parrot tongue." Thirst is common. Nausea and vomiting are fairly common, and meteorism is infrequent. The epigastrium only is tender. The liver is swollen, soft, and grayish. There is usually constipation (90 per cent. of the cases), although diarrhœa may appear at the crisis. Gastro-intestinal hemorrhage, a hemorrhagic diathesis, diphtheritic ulcerations, parotitis, ulceration of the œsophagus and pharynx, phlegmons, and peritonitis, are infrequent.

7. **Genito-Urinary Tract.**—The urine is febrile. The urea is often decreased but may be increased after the crisis. The chlorides are decreased or may be absent entirely. Albumin frequently appears in the second week. Nephritis (in 6 per cent. of the cases) is dangerous. Uræmia and hæmaturia are rare. The urine will frequently show the diazo reaction. Diacetone, leucin, and tyrosin, may be found. Dysuria, orchitis, and menstrual disturbances, occasionally occur.

8. **Spleen and Glands.**—An early splenic tumor is palpable in 33 to 66 per cent. of the cases. It disappears late in the second week. The splenic pulp is fluid and degenerated. Rupture has been described. The lymphatic glands are not usually enlarged, though adenitis was noted during the Crimean and Turko-Russian epidemics.

Recurrence.—This is very rare in typhus. It happened only once in 5,000 cases (Buchanan).

Anomalous Courses.—Light forms run a course of six to ten days, with less fever, fewer nervous symptoms, and less rapid pulse than in the more severe types. They include the abortive type without eruption, and the ambulatory type. Very severe forms are the hyperpyretic (fatal in two to three days), or the adynamic (in which the patient dies with low temperature, hemorrhages, etc.). Complication with other diseases, or the patient's advanced age, may intensify the symptoms.

Differentiation.—This is not difficult if the case is seen from beginning to end, or during an epidemic. There are no absolute pathological or bacteriological criteria. Mistakes in the diagnosis of sporadic cases .are often inevitable. In the initial stage only a probable diagnosis can be made.

In *smallpox* the chills, fever, prostration, severe aches, splenic tumor, and backache, are not final, since they are less marked in mild forms of the disease. The initial scarlatiniform eruption in the angles of the arms and groins is characteristic of smallpox. The morbilliform eruptions occur also in typhus. Smallpox invades the face more than does typhus, and the relation of the eruption to the temperature remission is not observed in typhus. Even at postmortem examinations, differentiation between the hemorrhagic form of typhus and the *purpura variolosa* is frequently impossible.

In *recurrent fever*, the fever-curve is important, but the diagnosis may be difficult during the first attack. The history, the lack of involvement of the sensorium, the generally favorable condition of the patient, the absence of pain, the characteristic anæmic yellow-gray skin, the spirilla and successive attacks, indicate recurrent fever.

In irregular epidemics, *meningitis*, with chills, fever, nervous symptoms, coma, and headache (without rigid neck and with a roseolous eruption), at first presents difficulties, though in the later course, it may be distinguished after lumbar puncture and examination of the retinæ. The eruption is at first roseolous but later is petechial. If the petechiæ occur first, the disease is not typhus.

TYPHUS.	TYPHOID.
Invasion: sudden; chill, coryza.	Slower and more gradual; no coryza.
Roseola: More numerous and earlier, second day to fifth day.	Usually less points of eruption (are exceptions,—*e. g.* measles-like); in extremities, as in Franco-German war
Never in crops.	In successive crops.
Distribution on body and extremities.	On body; rarely on extremities.
Sparingly on face.	Almost never on face.
Flat, pale, washed, hyperæmic, not disappearing upon pressure when they become petechial.	Papular, pink, sharply outlined, alway hyperæmic, disappearing upon pressure.
Course: more rapid.	Slower.
Face and eyes injected.	Paler; conjunctivitis rare.
Fever: abrupt rise, higher, less remission, if any; shorter; crisis or, short lysis, on fourteenth day.	Slow rise and regular stages.
Soon bedridden.	Oftener ambulatory.
Earlier and severer nervous symptoms.	Second week or later, and less severe.
Pulse: rapid and not dicrotic.	Dicrotism of less importance than slowness.
Spleen: early swelling, but less frequent.	Later, but lasts longer; two or thr times more frequent.
Less meteorism or none.	Meteorism.
Stools: not characteristic.	Ochre-stools.
Blood: leukocytosis.	Leukopenia, Widal reaction; bacter in blood, urine, roseolæ.

DIAZO REACTION IN BOTH.

In *Measles* the face is the first seat of eruption; the rash is rarel hemorrhagic; catarrhal symptoms precede the eruption, when th fever again rises. Typhus is rare under five years of age.

Prognosis.—The prognosis depends upon the intensity of the toxæmia, the fever, the nervous symptoms, and on the complications—especially pneumonia. Death from toxæmia usually occurs in th second week (twelfth day); from pneumonia in the third week. The mortality varies between 6 and 20 per cent., but reaches 50 per cent. in virulent epidemics and in patients over fifty years of age. Children rarely die of typhus.

Treatment.—The treatment is that of typhoid, with the added indication of strict isolation. Open-air treatment in tents is indicated, with thorough disinfection of everything coming in contact with the patient. Cardiac stimulants are more often required than in typhoid.

RECURRENT FEVER.

Synonyms.—Relapsing fever; febris recurrens; typhus recurrens; seven-day fever; famine or prison fever; bilious typhoid.

Definition.—A specific infectious disease caused by Obermeier's spirillum; acquired more often by direct personal contact than by indirect infection; endemic in Ireland, Russian Poland, and India, though sometimes epidemic; clinically characterized by a cyclic course of febrile attacks lasting six or seven days, followed by an equally long apyretic interval, which cycle may be repeated several times.

Etiology.—Recurrent fever has been known for nearly two centuries. It was first described by Rutty, of Dublin, in 1739. It appeared in America in 1844, and was last seen in 1869. Obermeier in 1873 described a specific spirillum or spirochæta (*Spirillum Obermeieri*). Recurrent fever is transmitted not by the spirillum itself, but by its spores, since the disease may recur after the spirillum has been killed by quinine. The spores are possibly the glistening bodies sometimes seen in the blood (*q. v.*). The breath and exhalations are infectious, though less so than in typhus. The secretions are apparently not infectious. The disease has been inoculated in monkeys and in man, from postmortem cuts. Bed-bugs may also convey it. Infection is carried chiefly by vagabonds and emigrants. Males are especially affected (80 to 98 per cent.). Most cases occur between the twentieth and the fortieth years. It is rare after forty-five, and has been seen in the fœtus. Epidemics may occur with typhus, and more rarely with typhoid, malaria, or smallpox.

Symptoms.—**Incubation.**—After an incubation of from five to seven days, the invasion occurs, usually, without prodromes.

The Invasion.—The invasion begins with a chill, a high temperature (reaching possibly 104° on the first day), a throbbing headache, and, in the young, convulsions and vomiting. The vertigo and cerebral confusion may resemble acute alcoholism. Because of the severe neuralgic pain in the back, legs, and calves, the patient lies motionless. There is great depression, conjunctival suffusion, slightly icteric scleræ, a pale (even cachectic) facial color, epigastric oppression with or without vomiting, and tenderness over the liver and spleen.

The Specific Findings are in (1) the blood; (2) the spleen; (3) the bone-marrow.

1. *The blood* is dark and reveals the pathognomonic, motile, cork-screw-shaped spirillum, which is found usually with the appearance o the fever, though occasionally a few hours before.

It often disappears and becomes less motile before the fever falls and is, on rare occasions, found from one to three, or even fourteen day after the fever. It appears again with the next attack. The spirillu is found in the blood only. The number is variable and not proportionate to the severity of the disease; as, for instance one spirillum i ten to seventy fields at first, and twelve to thirty spirilla in a field at th acme. Without the immersion-lens, they may be located by the lashin about of the red cells. They always lie between and never in them and appear as fine threads, sixteen to forty millimeters in length with five to ten waves in each, and move by undulation along their lon axes. They stain best with fuchsin, after the extraction of the hæmoglobin by acetic acid. Their life in the blood is usually thirty-seve days.

The white blood-cells are increased. Large, fatty, granular protoplasma-cells, probably from the spleen, may sometimes be seen.

2. *The spleen* is nearly always enlarged, painful, and palpable, an varies in size directly with the temperature. According to Leube, in n other infectious disease is the spleen so large, being increased five- t sixfold and weighing even twenty pounds. The capsule is tense sometimes with perisplenic adhesions, and the pulp is a fluid-red. Th Malpighian bodies are increased, and necrotic areas and infarcts ar common. Suppuration and rupture (6 per cent. of the cases) may occu Microscopically there are hyperæmia, hyperplasia, spirilla, large fatt cells (in the portal and other vessels), and fatty desquamate endothelium from the veins, thus explaining the spindle-cells found i the blood-stream.

3. The *bone-marrow* resembles the splenic follicles and contain many granular cells, and, occasionally, aggregations of white blood-cell

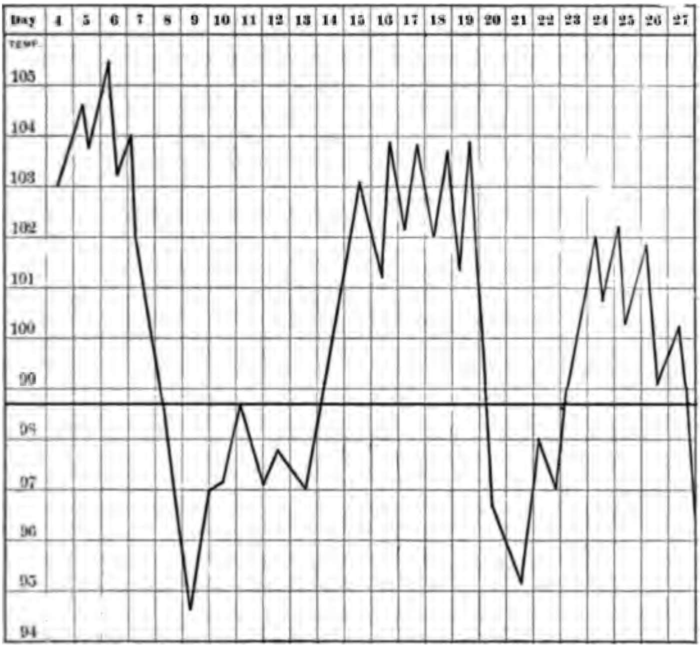
Other Symptoms.—1. Nervous System.—Headache is frequen The sensorium is usually free, delirium and insomnia being rare.

2. Temperature.—The temperature rises abruptly to 103° or 104,° an remains continuously high (105.8° to 107.6°), from five to seven day when the crisis occurs. In no other disease is the defervescence s precipitate (Leube). The fever falls 5° to 13° (even 16°). In from fi to seven days the patient is well, when in 99 per cent of cases the cycl is repeated. In the second or following attack there may be no chil There are usually but two or three relapses, though as many as seve have been recorded, each successive one being shorter by one to o and one-half days. The temperature-curve is diagnostic. Lysis is ra (1 to 2 per cent. of the cases). The crisis is followed by rapid convale cence, sweats, shrinkage of the spleen, and disappearance of the spiri lum; sometimes post-critical diarrhœa and collapse develop.

3. Circulatory System.—The pulse is rapid (120 to 140). Compar with the temperature, it is full and hard, seldom dicrotic, sometim irregular, and often slow after the crisis.

4. Respiratory Tract.—Bronchitis (20 to 47 per cent. of the cases) and increased respirations (thirty or more) have been observed. In fatal cases atelectasis, hypostasis, pneumonia (40 per cent.), glottis œdema (31 per cent.), and pleurisy (2 to 3 per cent.), have been found.

Fig. 7.



Fever chart in recurrent fever.

5. Gastro-Intestinal Tract.—The tongue is, at first, white and later becomes yellow-brown. The appetite may be unimpaired. Hæmatemesis, a tender and swollen liver, icterus (8 to 25 per cent. of the cases), gall-bladder distention, bloody effusions in the alimentary mucosa, and hyperplasia of the lymphatics, may be observed.

6. Genito-Urinary Tract.—The urinary findings resemble those of typhoid. Nephritis is infrequent (2 to 4 per cent. of the cases), and abortion occurs in 66 to 100 per cent.

7. Skin.—The skin is usually subicteric, yet the stools remain colored. Herpes (2 to 32 per cent. of the cases), œdema, erythema, petechiæ, urticaria, desquamation, and even roseolæ, have been noted. The hot and dry skin becomes drenched at the crisis.

Diagnosis.—The specific points are: A prevailing epidemic; onset with chill and fever; swelling and tenderness of the liver and spleen; muscular pains; an abrupt crisis; an apyretic interval with recurrence of the cycle, and the spirilla found by blood examination.

Loeventhal has found that the serum acts as a bactericide on the spirochætæ. In the bilious type (bilious typhoid or septic recurrent fever), there is severe icterus, destruction of the red corpuscles, and pathological liver-findings which are suggestive of acute yellow atrophy Hemorrhages, involvement of the sensorium, and death from collaps in the first attack (60 per cent. of the cases), are common. The colore stools, rapid pulse, severe bronchitis, and enlarged spleen, are significant Malaria, typhoid, typhus, yellow fever, acute yellow atrophy of th liver, and Weil's disease, are excluded by one examination of the blood

Complications of recurrent fever include pneumonia and othe lesions already mentioned. Meningeal hemorrhages, psychopathies paralysis, neuritis, and eye complications, are very infrequent.

Convalescence.—Convalescence is rapid, after four or five weeks Death may occur from toxæmia, heart collapse, or from complications The prognosis is good, the mortality averaging 2 to 10 per cent., althoug reaching 60 per cent. in the bilious type. One attack is not certain t confer immunity.

Treatment.—The treatment is that of typhoid, as no drug has an effect on the parasites. Pain often necessitates the use of opiates. Th heart weakness is treated as it is in typhoid.

SMALLPOX AND VARIOLA. VACCINATION.

Definition.—Smallpox is an acute, highly infectious, specific diseas It begins suddenly with a chill, headache, vomiting, and inten epigastric and lumbar pain, and is characterized by a typical feve curve and by an eruption on the skin and contiguous mucous su faces consisting of papules, vesicles, pustules, and crusts in successi stages.

History.—Smallpox is said to have prevailed in China many ce turies before the Christian era. Galen's *pesta magna* of the seco century is thought to have been variola. Epidemics occurred in t sixth century, and during the crusades. Rhazes (Arabia, ninth ce tury) gave us the first accurate clinical description of the disea Smallpox was introduced into Mexico in 1520, by the Spaniards, a three and a half million persons contracted the disease. Sydenha classic of the seventeenth century is most accurate and reliable. In 17 Lady Montague introduced into England preventive inoculati which had been practiced for centuries in Asia, and, though she redu the mortality by this means to 1 per cent. or less, she only dissemina the disease in a mitigated form. Jenner's discovery of vaccination 1796, has greatly lessened the terrors of variola, which in Eur alone carried off nearly half a million persons yearly, and in Icel cost the lives of 36 per cent. of the population.

Etiology.—The virus is still imperfectly known. Weigert and oth have found pyogenic organisms, although suppuration is due to a l mixed infection. Protozoa were first mentioned by Van der Loeff 1886. The *cytorytes variolæ* of Guarnieri (1892) is an amœba foun the blood in the incipient stage, in that of vaccinated children an calves, and in the exanthem. Pfeiffer, Roger, Weil, Funck, Copeman, Councilman, Mallory, Calkins, Howard, and Perkins, have found similar structures. Small bodies are found in the lower epithelial layers. These bodies are structureless, measure one to four microns, and lie in vacuoles in the cells. They enlarge and become granular and segmented.

The virus causes constitutional infection before the eruption appears. The greatest infectiousness is noted during the eruption. Its chief sources are the dried pustules in which the virus localizes. That the blood is infectious at an early stage was demonstrated by the development of the disease in an individual on whom skin had been transplanted from a person in the early stages of smallpox. Variola without eruption is contagious, and it therefore seems that the secretions, excretions, and exhalations, convey the virus. The virus adheres to objects having rough surfaces, such as bedding, clothing, public conveyances, and money. The disease persists in infected communities for years, becoming quiescent, and afterwards breaking out anew. Infection may be direct from person to person, or indirect by a third party or by fomites. Cadavers of variola victims are dangerous. In short, every new case comes directly or indirectly from a patient with variola. The virus is very penetrative, and infection may exist beyond the room, the building, or the area of the isolated case.

The chief factor in dissemination is human intercourse, and not telluric, atmospheric, or clinical conditions. Susceptibility to smallpox is almost universal, although individual disposition plays a great role. Only 1 per cent. of people are immune. Some can never be successfully vaccinated. Individual immunity was noted by Morgagni, Boerhave, and Diemerbroeck. The author knows two persons in whom vaccination has never been successful. Temporary immunity is more frequent than congenital, which, however, has been observed. Ordinarily and practically, immunity is only acquired by vaccination or a previous attack. In prehistoric times, inoculation was practiced to afford immunity. Second attacks are infrequent. Either sex and all ages may be affected. Mothers with the disease may deliver babes with the florid eruption or with scars. Some children born in pest-houses escape if vaccinated, but most of them die. The virulence of the virus is great in aboriginal and colored races (Indians and Mexicans), and in pregnant or menstruating women. Though it varies in different epidemics, vaccination is more important than virulence *per se*. Severe cases may follow mild infection, as shown in Osler's description of the Montreal epidemic, in which 3,164 persons, infected by a mild case in a Pullman-car conductor, died among the French Canadians, who oppose vaccination. It is frequently conveyed by vagrants.

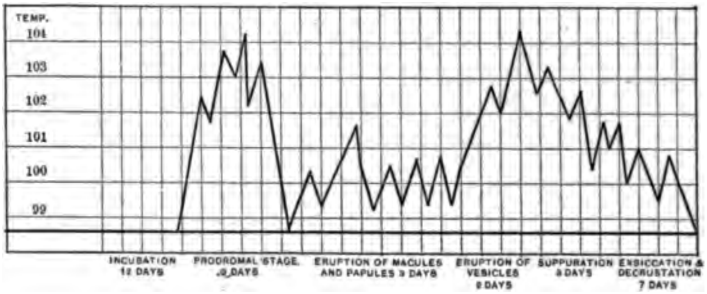
Point of Entrance.—Smallpox infection unquestionably occurs through the respiratory tract, and perhaps also through the digestive tract. Variola may coexist with or closely follow measles, scarlatina, syphilis, erysipelas, pemphigus, typhoid, malaria, and tuberculosis. Variola occurs sporadically, epidemically, or panepidemically.

Symptoms.—Forms:

I. Variola Vera —1. Discreta, discrete.
—2. Confluens, confluent.
II. Variola Hemorrhagica —3. Purpura variolosa (black smallpox).
—4. Variola hemorrhagica pustulosa.
III. Varioloid —5. Smallpox modified by vaccination.

1. Variola Vera Discreta.—Pfeiffer's Table, with fever-curve.

FIG. 8.



Temperature chart and stages of smallpox (Pfeiffer.)

(*A*). THE INCUBATION.—The incubation, or period of laten ranges from five to twenty days, and averages twelve days. It las eight days after inoculation, and usually presents no symptoms, thoug sometimes there may be headache, anorexia, and pharyngitis.

(*B*). THE PRODROMAL STAGE.—The prodromal stage, (initial stag or stage of invasion,) dating from the first symptom to the cutaneo eruption, is the same in all types, and its duration averages thr days (rarely six to fourteen). It is shorter in children (Rilliez a Barthet) and in the confluent type of the disease; the longer it is, t more severe is the infection (Trousseau). Its intensity bears no i variable relation to prognosis, for varioloid (mild smallpox), m present a severe onset; if the onset, however, is mild, the disease w not be confluent or hemorrhagic in form. Prodromal symptoms a absent in rare instances.

The invasion begins acutely with chill, fever, rapid pulse and respir tion, nervous toxæmic symptoms, intense headache, and backach vomiting, and other symptoms, all of which will be considered detail: (*a*) The *chill* is usually severe, and is frequently repeate It may follow one or two days of fever (Jaccoud). In children it often absent. (*b*) The *initial fever* is almost invariably high, risi suddenly to 103° or 104°, and reaching its maximum on the second third day. In rare instances there is no fever. (*c*) The *pulse* is rap (120), full, and not often dicrotic except in severe cases. The mo frequent respiration (30 to 36) is somewhat characteristic, constituti the so-called toxæmic or "cerebral respiration." (*d*) The *skin* is r excessively hot (*calor mordax*), and usually dry, but there may sweating in the discrete variety (Sydenham) and in favorable ca (Trousseau). The face is flushed but the eyes are clear. (*e*) *Nervo* *toxæmia* is evidenced by the benumbed sensorium, depression, restlessness, insomnia, delirium (especially in alcoholics), or convulsions and meningeal symptoms in children. Coma is rare. Headache is almost constant, usually appearing with or just before the chill. It is frequently frontal or temporal, and may be generalized, occipital, or neuralgic. When severe or agonizing in character, it may be suggestive of meningitis, especially when accompanied by delirium, hard vomiting, and neckache. (*f*) *Backache* appears with the chill, and lasts one or two days. It occurs in nearly all cases (certainly in over 50 per cent.), though it is rather less frequent than headache and vomiting. It is severe like lumbago, or actually agonizing. The "veritable paraplegia" described by Trousseau is usually only immobility due to pain caused by toxins or by intense congestion (Jaccoud) of the kidneys, spinal muscles, or meninges. It is rare in other fevers likely to be confused with variola. Pain is sometimes observed in the pharynx, larynx, or heart, and produces a sensation of constriction; in the chest it results in oppression or actual dyspnœa; it may occur in the intercostal nerves, sciatics, colon, bladder, muscles, and joints. (*g*) *Vomiting* and early epigastric pain (especially constant in children) occur with other febrile digestive symptoms. The spleen is not often enlarged in the initial period. It is often tender, as are the liver and epigastrium. (*h*) The *initial* or *prodromal eruptions*, studied especially by Sydenham, Simon, Osler, Knecht, and Scheby-Busch, are present in 10 to 16 per cent. of the cases. They vary with the epidemic and have considerable diagnostic value. They may be general but are more commonly limited to the lower abdomen, the inside of the thighs, the sides of the thorax, the axillæ, and sometimes to the extensor surfaces of the knees and the elbows (Simon). There are two varieties:

1. The *morbilliform variety* (measles-like, roseola variolosa), which appears during the second day on the face and body as small maculæ. These are sometimes crescentic, disappear upon pressure, and usually have a life of one day with complete involution, although in exceptional cases they remain until the smallpox papules appear. They are due to hyperæmia but are often purpuric, occurring without the measles catarrh. They are seen most often in varioloid, and therefore have a favorable prognostic import.

2. The *scarlatinal form* (erythema variolosum) is more rare than the morbilliform variety and occurs earlier, either on the first day, or before any other symptoms. It is less punctate, extensive, and bright, than it is in scarlet fever. If it is purple in color and purpuric, it is almost always variolous. It occurs on the lower abdomen, and on the thighs (forming a triangle with base upwards when the legs are together), but may even reach the knees. Sometimes it is found on the axilla or over the shoulders, and is seen in women more frequently than in men. These areas are rarely invaded by the pustular eruption (Trousseau and Hebra; though denied by Eichhorst). Some writers (Gigon, Barthelemy) speak of an erysipelas-like prodromal rash, and Osler and others have observed prodromal urticaria.

(*C*). THE ERUPTIVE STAGE.—The eruptive stage consists of several serial sub-stages:

1. The stage of *macules and papules* (*stadium maculosum et papulosum*) occurs on the fourth day, and advances for three days. The eruption begins on the forehead near the hair and on the cheek, with itching and burning macules, resembling flea-bites, which soon become papules (like those of prurigo or lichen, Trousseau). The papules are reddish, discrete, circular, elevated, and hard or "shotty." They may be observed in photographs taken a number of hours before they can be seen by the naked eye. A day after their eruption on the face and head, they appear on the body, and on the next succeeding day they appear on the extensor aspects of the extremities; *i. e.*, the papular eruption advances for three days, and is descending. In children the papules may develop first near the groin. The papules are thickest on the face and trunk. Sometimes they are very thick on the neck. The eye-lids are œdematous. According to Stokes, the amount of the eruption depends on the vascularity of the part. Trousseau and Sydenham maintained that if the eruption appeared on the second day of the disease, the confluent type might be expected; if on the third or fourth (or even the fifth) day, the discrete type might be anticipated. If the eruption ceases at this point, the disease is called variola cornea, or verrucosa.

Weigert holds that the first skin changes are not inflammatory but necrobiotic or diphtheroid, and that inflammation begins later with exudation into and about the necrotic areas in the rete Malphigii. Other authorities consider inflammation the primary, and necrosis the secondary process.

Unlike measles and scarlatina, the fever remits when the eruption appears, and the backache, and headache improve. In two or three days the fever reaches normal by irregular steps. The lower the fever falls in this remission, the lighter is the type of smallpox.

2. In the stage of *vesiculation* (*stadium vesiculosum*), lymph gathers in many but not in all of the papules. This occurs on the seventh day of the disease and lasts about two days. The vesicles are multilocular, since they occur in the cells of the rete Malpighii (Weigert). Umbilication occurs in the centres of many vesicles, caused by elevation of their edges by infiltration and by the follicles in their centers. It corresponds to the area of primary necrosis and is suggestive but not pathognomonic of smallpox (Jaccoud, Eichhorst). On the soles of the feet and the palms of the hands the fluid is situated deep in the resistant tissues. (If the changes stop short of pustulation, the disease is called variola crystallina.) The mucous membranes may be the seat of simple inflammation, specific eruption, or pseudomembrane.

3. The stage of *suppuration* (*stadium pustulosum*) or maturation begins on the ninth day with clouding of the vesicles and with inflammatory congestion (areola) about them. It advances for usually three and rarely more than five days. Increased mucous membrane catarrh develops simultaneously with the cutaneous eruption. The pustules

become opaque, then yellow, and the thick pus obliterates the umbilication and renders the eruption globular. The areola, or halo, becomes more vivid and inflammatory œdema may result from fusion of the areolæ. This causes increased tension, deformity (especially in the face and parts where the skin is most loose), closure of the eyes, occlusion of the nose (of particular moment in children), and great tenderness and pain. Pustulation follows the descending order of the initial eruption and the pustules are less thick on the trunk than on the extremities and head. On the volar and plantar surfaces they are less prominent and there is less œdema. The pustules evacuate spontaneously, especially on the face, but may dry without rupture. Various stages of the eruption may be observed in different parts of the body at the same time; not all vesicles become pustules. The skin exhales a peculiar, penetrating, offensive odor, and bed-sores may develop.

(Anomalies are variola *abortiva*, in which the pustular stage is not reached; variola *siliquosa*,—air in the vesicles or pustules; and very large eruptions three quarters of an inch in diameter.)

Pathologically, the pus originates from the vessels of the papillæ, which may undergo much pressure, atrophy, or even necrosis.

The mucous membranes are involved in various ways, but the pock eruption occurs mostly in the upper mucosæ (mouth and nasopharynx), and therein resembles its cutaneous distribution; the successive stages of papulation, vesiculation, pustulation, and crustation, may develop, but they are less typical than in the cutaneous eruption; erosions often result with diffuse catarrhal stomatitis, and sometimes with glossitis, phlegmonous angina, etc. The eruption has been found in very rare instances in the external ear, Eustachian tube, eye, œsophagus, larnyx (with œdema or perichondritis), trachea and bronchi (mostly erosions), stomach and intestines with swelling of Peyer's patches, rectum, bladder, cervix, prepuce, distal end of the urethra, vulva, and vagina, with appropriate symptoms of local irritation. Pathologically, the changes resemble those in the skin, but they have been less thoroughly studied.

With pustulation there is in most cases a gradual rise in temperature, *secondary fever*, the fever of suppuration. It is never sudden, is sometimes associated with chills, especially in women and children and in nervous patients, and is due to the smallpox virus and mixed pyogenic infection. The duration of the rise is proportionate to the degree of pustulation and of dermatitis. In variola discreta the temperature does not remain high more than twenty-four to thirty-six hours, with morning remissions. A marked *leukocytosis* attends the fever, or it may even precede suppuration by one, two, or three days. It parallels the degree of infection and may even attain 30,000 to 34,000 cells per cubic millimeter. It ceases with involution unless complications intervene. The lymphocytes are increased (to 50 or 66 per cent.), the eosinphiles are decreased (to 50 or 33 per cent.), and the myelocytes may reach 10 to 12 per cent.

The red cells occur in clumps rather than in rouleaux, and some deglobulization, even as low as 2,000,000, is observed. The fever may show a preagonal increase or collapse. Hyperpyrexia may occur. Repeated rises in temperature are very dangerous. Cerebral symptoms occur, such as great excitement, or delirium with suicidal tendencies, as happened in the case of one of the author's classmates. Albuminuria (33 per cent. of the cases), acute exhaustion, and heart paralysis, especially in the aged, may develop.

(*D*). THE STAGE OF INVOLUTION.—The stage of involution (*exsiccation, decrustation*), begins on the twelfth day. It occurs in the same descending order as the florition and suppuration, first on the head, then on the body, etc., with decrease in the œdema, in the redness, and in the pain. As the pain decreases, there is an intolerable itching and some pigmentation. Crusts form, and remain longest on the soles of the feet and palms of the hands where they may form complete casts of the part. Mucous erosions heal more readily than crusts. The hair falls out, and by the end of the second week the fever reaches normal. There is no desiccation fever, as claimed by some writers, and fever at this stage indicates some complication. Scars result from the granulations, and the higher the granulating surface, the deeper is the cicatrix. Scars occur when the true skin has been involved, and are seen chiefly on the face, scalp, palms of the hands, and soles of the feet. Sydenham's statement, that discrete smallpox rarely leaves its mark, holds good in most cases, according to Osler. Others hold that variola vera implies suppuration with fever and cicatrices as necessary stigmata. Complete convalescence, according to Jaccoud, follows the disappearance of the last crust. Scar formation lasts three or four weeks.

2. Variola Vera Confluens.—This is a malignant type, which was more frequently observed in prevaccination days, and is now seen largely in unvaccinated persons or in those vaccinated but once. Personal tendency, the *genius epidemicus*, and propagation *per continuum*, further the development of this type. The initial stage is violent. The headache and backache are agonizing. The status typhosus and gastric symptoms are marked, the fever remission is slight or absent, and there is little or no improvement in the symptoms when the fall in temperature occurs. The exanthem is precipitate both in its development (on the second or third day), and in its extension. The earlier the exanthem in variola (*e. g.*, on third day), the greater is the probability of confluent smallpox. The patient usually dies if the eruption does not appear. The eruption becomes confluent especially on the face and head, and sometimes also upon the hands and feet whereas on the body and limbs it is largely discrete. With fusion of the eruption, great inflammatory œdema appears, the symptoms increase swelling and erosion of the mucous membranes, closure of the eyes keratitis, and obstruction of the nose, develop.

The general aspect is desperate. High fever, high pulse, rapid noisy, even irregular or stertorous breathing, delirium, albuminuria persistent nausea and vomiting, great thirst, salivation in adults and diarrhœa in children (Sydenham), husky voice, enlarged cervical glands and often parotitis, are present. This type is severe from the onset and the prognosis depends upon the number of pustules upon the face. Sydenham says that "if upon the face they are as thick as sand, it is no advantage to have them few and far between upon the body." Death occurs from acute toxæmia (*variola typhosa seu adynamica*), with hyperpyrexia and cardiac asthenia, or, if the patient lives longer, from septicopyæmia, phlegmon, erysipelas, gangrene, pneumonia, pleurisy, and nephritis. Recovery from variola confluens is infrequent; in convalescent cases permanent deformity, such as ectropion, is probable. These cases run a course of three or more weeks.

3. Purpura Variolosa.—This is the black smallpox—smallpox with primary hemorrhage in the initial stages. It is the worst type, and results almost uniformly in early death. It was frequent in the great pandemic of 1870-72. It is also important because of the difficulty in its diagnosis. It is seen most frequently in the unvaccinated, but may occur in the vaccinated, in women during the puerperium, in the young, and in robust males. The incubation is short (six to eight days), the invasion is stormy, with agonizing lumbar pain, great prostration, a rapid, small, soft, and sometimes dicrotic or irregular pulse, and disproportionately increased respiration. The initial epigastric pain and vomiting may last until death.

On the second day (or even on the first), there appears a dark scarlet or plum-colored eruption, with brick-red, purple, or inky ecchymoses, which appear especially about the eyes. The aspect of the discolored and swollen face, and ecchymotic, sunken and intelligent eyes, is desperate. Hæmaturia is the most common of internal or visceral hemorrhages. Other forms are: Hæmatemesis, enterorrhagia, epistaxis, metrorrhagia, and hemorrhage from the gums, ears, and bronchi, sometimes accompanied by gangrene of the pharynx. Tympanites and genuine albuminuria may be noted. Miscarriage is frequent. The spleen is not enlarged. The disease does not usually reach the real eruption of variola, or, at the most, only the papules are observed, because, as Rhazes observed, the course is almost invariably fatal in four or five days. The mind often remains lucid to the end. In the majority of cases the fever is not high, but in the most fulminant type it reaches 105° or 106°, with delirium, collapse, and coma. Death results in a few hours, even before cutaneous hemorrhage is seen, although internal hemorrhages are found at autopsy. According to Unna, the "blood dissolution" of the older writers is a mycotic coagulation thrombosis.

4. Variola Pustulosa Hemorrhagica.—This is more common than Purpura Variolosa (primary hemorrhage). In this type of smallpox hemorrhage occurs after the eruption appears (secondary hemorrhage). This form occurs in weakly or alcoholic subjects. The initial stage is severe, and secondary hemorrhages occur into the vesicles or pustules, or into the lower parts of the body if the delirious patient has left his bed, or there may be "blood dissolution," epistaxis, hæmaturia, and metrorrhagia. Adynamic manifestations are usual, and the outcome is almost always fatal, though in one type, hemorrhage at the vesicular stage may be followed by rapid abortion of the rash and speedy recovery (Osler).

5. **Varioloid.**—Varioloid (*variola modificata s. mitigata, variolois*) is variola mitigated by vaccination. The more marked the initial eruption, which is often morbilliform, the less the number of pustules. Varioloid begins suddenly. The fever is atypical. It may reach 103° and usually falls with the specific eruption, reaching normal on the fourth day. Secondary suppuration fever is rare and euphoria is common. The eruption is superficial, and, according to French clinicians, there is no suppuration. The eruption varies in amount, being present on the trunk only, or being typical chiefly on the hands and feet. It may be absent entirely. It may not be umbilicated and may desiccate rapidly without rupture. The exanthem is usually small and scars are rare. The mucous surfaces are little involved, and are entirely unaffected. The entire course is shorter, more irregular, and rudimentary, and more benign than in variola vera.

Complications and Sequelæ.—The complications and sequelæ of variola are many, occurring chiefly in the more severe forms and during the stage of suppuration.

1. **The Nervous System.**—Nervous complications are very frequent. Delirium is caused by brain hyperæmia, which results from swelling of the face and scalp (Curschmann); Jaccoud's types of nervous complications are (*a*) simple toxic, in nervous individuals, (*b*) alcoholic, (*c*) hyperpyretic. Convulsions are frequent in children. In adults they render the prognosis uncertain. Psychoses are usually asthenic (Kraepelin) and less frequent than they are after typhoid. Meningitis suppurativa is unusual and is seen most often in children (Gregory); encephalitis, aphasia in children, hemiplegia, embolism, cerebral softening, phlebitis, and abscess, are occasional complications. Old writers, such as Trousseau, spoke of paraplegia, to which reference has already been made under backache. True motor paraplegia undoubtedly may occur, and also monoplegia, both caused by myelitis. Landry's paralysis has been recorded, as well as neuritis in the trunk and extremities, such as acute ataxia, or paralysis in the brachial, pharyngeal, œsophageal, bulbar, facial, and optic nerves.

2. **The Vascular System.**—Vascular changes are more rare. The heart muscle may degenerate, becoming soft and relaxed, and microscopically revealing segmentation, and cloudy and fatty conditions. Dilatation may occur. French writers speak of a coronary endarteritis and myocarditis (Desnos, Huchard). A systolic bruit may be heard at the apex, with weak first tone, and tachycardia, as is the case in typhoid. Aortitis with brain embolism (Potain) has been noted. Pericarditis and ulcerative endocarditis are rare. Marantic thrombosis occurs occasionally.

3. **The Respiratory Tract.**—Œdema of the larynx and perichondritis are usually fatal, for laryngeal anæsthesia promotes inhalation

pneumonia. Obstruction of the larynx by submucous hemorrhage is an uncommon complication. Bronchitis is invariable in severe cases. Pneumonia, usually lobular, occurs in 40 to 100 per cent. of fatal cases (confluent and hemorrhagic variola); lobar pneumonia is rare. Lung abscess, embolism, and gangrene, are uncommon. Pleurisy is present in some epidemics, is quite common according to Quinquad, occurs chiefly in the stage of suppuration, and is especially and rapidly fatal in children (E. Smith).

4. **The Digestive System.**—Glossitis is rare and is observed chiefly in smallpox of the confluent type. Retropharyngeal abscess, gangrene, parotitis, noma, phlegmonous œsophagitis, and hemorrhage, or membrane formation in the throat, are ominous though uncommon complications. The early vomiting rarely persists. Diarrhœa is not common, but a simple form is seen in children (Trousseau, Sydenham), and a dysenteric form is occasionally seen in adults. Peritonitis and perineal abscess are infrequent. The alimentary tract, with the exception of the rectum, is usually considered exempt from the eruption. Isolated cases of retroperitoneal cellulitis and mediastinitis occur. The liver may suffer cloudy or fatty change, focal necrosis, or diffuse hepatitis. The spleen is usually swollen and soft; but is sometimes firm and hard.

5. **The Genito-urinary Tract.**—This is not often involved. Albuminuria may occur early (Trousseau) in 25 to 33 per cent. of the cases. It may also occur during the eruption (Jaccoud), during suppuration, or in convalescence (Cartaz, Bourgin, 2 to 10 per cent.). Nephritis is not frequent, but may develop during the fever or in convalescence; it is often glomerular. Focal necrosis may be noted. Pyelitis from cystitis or myelitis, diabetes mellitus, ovaritis, and vulvar gangrene, have been observed. Menorrhagia is common in the early stages of onset or in hemorrhagic forms, and abortion from hemorrhagic endometritis is the rule in pregnant women. Orchitis variolosa (Trousseau, Chiari, Velpaux), is very frequent, the inflammatory foci being multiple. They lie mostly in the connective tissue, but are rarely suppurative, or associated with focal necrosis.

6. **The Special Senses.**—The eye is involved in 1 per cent. (Hebra). Pox in the conjunctiva are rare, as are conjunctivitis, keratitis, diphtheria of the conjunctiva, iridochoroiditis, neuroretinitis, metastatic panophthalmitis, and retinal hemorrhage. Otitis, mastoid caries, and suppurative thrombophlebitis, may occur.

7. **The Locomotor System.**—Muscular abscess or hemorrhage and arthritis, especially in the knee and shoulder, may occur during desiccation. Arthritis is more often suppurative than serous, and is a feature of the secondary sepsis. In the bones there are almost invariably hemorrhage into the marrow, hyperplasia of the marrow cells, and multiple foci of necrosis or nonsuppurative osteomyelitis (in 72 per cent. of the cases [Chiari]).

8. **The Skin.**—Decubitus, erysipelas, phlegmon, furuncles, abscesses (local or pyæmic), post-variolous eruptions, erythema, pus-

tules, impetigo contagiosa, bullæ, great desquamation, acute gangrene (especially of the scrotum, and usually fatal), may develop in severe cases. In purpura variola, superficial hemorrhages occur in the skin in addition to subcutaneous hemorrhagic infiltration. Hemorrhage into existing exudates, and less often hemorrhage in the heart, spleen, kidney, liver, stomach, intestines, cellular tissue of the thoracic and abdominal cavities, brain, and spine, also occur.

Diagnosis.—The diagnosis of smallpox is usually easy when chill, fever, headache, backache, and vomiting, occur in the presence of an epidemic or after exposure to infection. Vaccination and revaccination are factors and their scars must be searched for. It is often impossible to avoid mistakes in the diagnosis of the first cases of an epidemic. In abortive or purpuric cases a diagnosis is also difficult or impossible. Hemorrhagic scarlatina or measles sometimes cause difficulty. In the former the mucous hemorrhages are less frequent than in smallpox. Differentiation between smallpox and virulent hemorrhagic or fulminating purpura may be difficult. The initial suggestive scarlatiniform eruption is important but it has been unduly emphasized; the prodromal eruptions *plus* purpura are highly suggestive. Exposure to infection followed by an acute onset and high temperature is significant, although acute onset and high fever may occur in typhus (chill, macular and petechial rash, especially on the body, and persistently high fever), in pneumonia (chill, vomiting, herpes, pain in the chest, and rusty sputum), in scarlatina and influenza, with an initial rash, and in meningitis (joint involvement, headache, or vomiting). Lumbar and sacral pain is more common in smallpox than in typhus fever, pneumonia, or scarlatina. Intense frontal headache, severe backache, vomiting, chill, and fever, are sufficient grounds for a tentative diagnosis, and are important enough to necessitate isolation. Nevertheless, the diagnosis is uncertain until the eruption is seen. In the *variola sine exanthemate* (the *febris variolosa* of Rhazes and Sydenham, the *variola sine variolois* of de Haen, the *varioles frustes* of Costes) the diagnosis must be made from the history of exposure, the presence of an epidemic, fever, pain, delirium, possibly the initialr ash, (Sydenham, Frank, de Haen,) and the absence of secondary fever because there is no suppuration. An eruption which appears on the third day of the affection, accompanied by a fall of the fever and euphoria, is most important.

VARICELLA——*vs.*——VARIOLA.

Etiology different. Vaccination and smallpox do not protect.	Smallpox may closely resemble chickenpox (epidemic of Lee); very mild cases are especially misleading.
Age: is not distinctively differential; varicella usually occurs before puberty; it may occur in adults.	Usually after puberty, with numerous exceptions.
An initial stage is practically absent.	It is severe, even in mild cases.

(Concluded on next page.)

VARIÇELLA——*vs.*——VARIOLA.

Temperature: no remission with outbreak of rash.	Typical remission with secondary fever (except in varioloid).
Prodromal rash: very exceptional.	Frequent (10 to 16 per cent.).
Vesicles and roseolæ (maculæ) occur in crops and are simultaneous; very rarely shotty.	Never in crops; first macules, then hard, shotty papules, followed by vesicles, etc.
Rash evolution: is much more rapid; vesicles in the first or second day.	Much slower; vesiculation on the seventh day (see Pfeiffer's table).
Eruption is universal, although successive crops may occur; most abundant and characteristic (denied by Thomas) on back; begins on body; less on face, hairy scalp, mucosæ, hands, and feet.	Note place of first development; development progresses downward—face first, then hands and feet; less on trunk.
The vesicle is superficial, and the transparent fluid shines through. (Wyss has observed confluent eruption and umbilication).	The fluid is pearl-colored, not transparent, and has a thicker covering.
Areola (halo) is usually absent.	Marked.
Involution is rapid.	Tardy.

Mistakes may occur among smallpox experts, but attention to the history, somatic findings, and course, rather than to the eruption, prevents many disastrous results.

MEASLES——*vs.*——VARIOLA.

Both present the same prodromal duration; in each the eruption begins on the face; according to some observers, simultaneously on face and trunk.

Age: eminently a childhood affection.	Chiefly in adults.
Catarrhal stage characteristic.	Catarrh present, if at all, in rudimentary form.
Fever: present in catarrhal stage, but low before exanthem, and rises with its appearance	High before eruption, and falls with florition of the exanthem even in severe cases.
Exanthem: Koplick's spots on buccal and labial mucosa; bluish-white surrounded by red.	Absent. Early papular eruption on palate, etc.
Eruption later with rise of fever.	Initial morbilliform eruption with the primary fever.
Papules "grouped," crescentic.	Never so.
Papules remain relatively flat, smooth, velvety.	Change into vesicles, pustules, crusts, scars; hard and shotty.
Grisolle's sign absent (the eruption disappearing under pressure).	Grisolle's sign present; *i. e.*, persistence in smallpox of the papule on stretching the skin, which distinguishes its eruption from scarlatina, varicella, etc.
Areola (halo) absent.	"Halo" present.

SCARLATINA——— *vs.*	———VARIOLA.
Rash on the second day, angina, strawberry tongue, desquamation, glands, nephritis, etc. (See cardinal symptoms of scarlet fever).	Rash later; slow evolution of different phases. No angina (or late, at least), no glands, no strawberry tongue; the initial scarlatinal eruption is not punctate, less extensive and bright, and does not desquamate.
No double elevation of fever-curve.	Double summit to curve.

In *drug rashes* (copaiba, etc.), the pustules occur largely on the face, head, arms, and forearms, and much less frequently upon the trunk or legs. In *syphilis* the initial lesion, the glands, the polymorphism of the rash, and the absence of fever, or its presence in moderate form, are easily distinctive.

The lumbago form of *acute nephritis* (Robin), acute myelitis or rheumatism, erythema, menstrual rashes, septicæmic rashes, acne (cachectic and vulgar), and lichen, only superficially resemble variola.

Glanders may cause more difficulty in making a diagnosis. Its features are malaise, pyrexia, pains, papules, pustules, nasal discharge, considerable exudation, and the proper bacteriological findings. Its general symptoms are more severe than the degree of skin eruption would indicate.

Prognosis.—The prognosis depends on (*a*) previous vaccination, the disease being very fatal (40 to 50 per cent. mortality) in those unprotected by vaccination and revaccination. Smallpox is rarely fatal in persons who have been vaccinated within five or six years; only 8 per cent. of those vaccinated once, die and only 4 per cent. of those vaccinated twice. (*b*) On the *genius epidemicus:* In our last epidemic the death rate was but 3 per cent. (*c*) On the type, being (i) practically 100 per cent. in purpura variolosa; (ii) nearly 100 per cent. in variola pustulosa hemorrhagica; (iii) 60 per cent. in variola confluens—especially fatal in the first decade of life; (iv) 25 per cent. (15 to 35 per cent.) in variola discreta,—particularly dangerous in those over fifty or under ten years, in pregnancy, in the puerperium, in weakly and in alcoholic subjects; (v) 1 per cent. in varioloid; (vi) recovery usual in *variola sine exanthemate.*

The prognosis also depends upon *individual symptoms:* 1. Incubation is short in unfavorable forms, especially in purpura variolosa.

2. Initial symptoms are mild, chiefly in varioloid, but may be severe in both the benignant and malignant forms. Very severe headache indicates the hemorrhagic form.

3. The initial rash is usually a partial erythema in mild forms of smallpox; and, in general, morbilliform eruptions occur in varioloid. Scarlatiniform eruptions occur in rather severe forms, and a universal scarlatinal hemorrhagic eruption, especially in the groins, occurs particularly in purpuric types.

4. The danger is proportionate to the amount of the eruption on the face and hands (Sydenham). A slow typical exanthem indicates variola discreta, and a precipitate atypical eruption is a symptom of variola confluens.

5. A prompt fall in temperature after the eruption is favorable. A slow, complete fall occurs chiefly in the severe forms. Persistent apyrexia after florition and early desiccation indicate a mild type. Accession of fever after pustulation is ominous (Rhazes). There are fewer complications with an early fall of temperature. Hyperpyrexia or temperature collapse during suppuration is a sign of imminent death.

6. In the stage of suppuration the danger is parallel to the height of the fever and the amount of cutaneous and mucous efflorescence. Enlargement of the spleen is ominous.

Treatment.—I. **Prophylactic Vaccination.**—Protection from smallpox by cowpox vaccination was discovered by Jenner, who published his results in 1798, although he was practically certain of his evidence twenty years earlier. The idea had been long prevalent among farmers that cowpox afforded immunity to smallpox. Professor Waterhouse, of Harvard, in 1800, introduced vaccination into this country, and, among others, President Jefferson was instrumental in disseminating its practice.

The nature of cowpox, or vaccinia, is still bitterly disputed. The most probable view is that cowpox and horsepox are human smallpox modified by transmission through animals. Copeman suggests that vaccinia and variola have descended from a common stock. Variola inoculated in the cow produces typical vaccinia. The other view, held largely by the French, is based upon Chauveau's experiments, which conclude that the two affections are distinct. The bacteriology of vaccinia is also uncertain. Micrococci (Quist, Martin, and Ernst), bacilli (Klein and Copeman), and amœbæ (Guarnieri's *cytorytes vacciniæ*, Pfeiffer, Ruffer, and Reed), have been found.

USUAL SYMPTOMS.—The incubation period is three days, during which there may be slight traumatic local reaction. The eruption occurs on the third day as a hard papule surrounded by a delicate halo. The papule enlarges, and on the fifth (or sixth) day shows Jenner's vesicle filled with clear serum, and umbilicated in the centre. It merges peripherally into the already indurated and painful skin, in which, by the eighth day, the magnifying lens shows many minute vesicles. By the tenth day, the serum of the vesicle has become purulent and the surrounding tissues are very hard and painful. By the eleventh day, a small central crust appears, which, by the end of the second week, covers the entire vesicle and is accompanied by a recession of the adjacent cellulitis. In one or two weeks, the ulceration, decrustation, and granulation, are usually complete, and a scar or pit remains, which pales with time. Constitutional reaction is greatest in children. The fever in most cases appears on the fifth day, and sometimes lasts four or five days, with leukocytosis, restlessness, headache, or backache. A swelling, depending on the site of inoculation, occurs in the axillary or inguinal glands.

UNUSUAL SYMPTOMS.—The vesicles may develop early and run a course which may be shorter by half than the usual one. Crusts may form in a week. In other cases the eruption may be tardy and atypical. In both instances revaccination is indicated. The eruption may recur as many as four times (Sutton), and in rare cases may be hemorrhagic, or even fatal.

COMPLICATIONS.—These are rarely fatal. In Germany, in 1895, two deaths (from erysipelas) occurred in two and a half million vaccinations. To offset this theoretical risk there is the almost universal susceptibility to smallpox in all unvaccinated individuals. (1) Erythema, urticaria, roseolæ, or erysipelas (third to eighth day). (2) Accessory vaccinia near the original inoculation, or generalized vaccinia, which develops about the eighth day on the trunk or extremities, and often on the wrists; bullous or miliary vesicles sometimes continue to develop for over a month, and in weakly subjects are very exceptionally fatal. (3) Secondary infection in the first week or two, either local (deep ulceration, gangrene, lymphangitis, or lymphadenitis), or systemic (sepsis, pericarditis, meningitis, and parotitis). Very few cases of tetanus have occurred; McFarland (1903) collected 68 certain cases, the majority of which occurred in Philadelphia. Albuminuria is infrequent. (4) Later infections, such as tuberculosis or syphilis. Tuberculosis is but seldom inoculated, because it is rare in calves. Its inoculation can easily be prevented by the Belgian method of not using the lymph until the animal has been killed and found healthy. In some cases a latent tuberculosis in the patient may be awakened by vaccination, which seems to act like tuberculin. The danger of inoculating syphilis is far less now than when human virus was used. Fifteen days are necessary for the development of a chancre, and after about eight weeks more the secondaries appear. These are the chief diagnostic criteria. Syphilis may be inoculated accidentally and also intentionally, as in the oft-cited case of Dr Cory. In exceptional cases syphilis already in the system may be aroused by vaccination. There are no clear cases of inoculation of leprosy.

AGE.—Children should be vaccinated in the third or fourth month and it should *never* be delayed beyond the first year.

TECHNIQUE.—The arm (or leg, in the case of girls), should be washed but no antiseptics should be employed, lest they neutralize the virus A fold of skin should be raised and either rubbed with a piece o sterile gauze or scraped with a knife held almost parallel to the skin until the epidermis is removed and slight oozing of lymph appears The virus must reach the lymph stream, and therefore blood should not be drawn, as coagulation somewhat inhibits "taking." The vac cine should be slowly rubbed over and into the abraded skin. The spot should be covered with a sterile dressing or cloth held in place by adhesive plaster, only after the virus has thoroughly dried. Boric carbolic, or bichloride gauze must not be used.

LYMPH.—Vaccinia virus is harmless if it is pure and has been col lected in the vesicular and not in the pustular stage. It can best be preserved in the fluid form, in sealed sterile tubes with three or fou parts of glycerin. This obviates pyogenic infection, preserves the virus longer (four months), and is more economical than any other method; as twenty to twenty-five times as many vaccinations are possible with the same amount of virus when preserved by this method. When human virus must be used it should be taken from a perfectly sound child which has been carefully examined for tuberculosis and syphilis. It should be taken from the clear and unbroken vaccinia vesicle on the seventh or eighth day. No blood should be withdrawn, and the vesicle must be free from pus.'

Results.—The successful vaccination, accompanied by typical papulation, vesiculation, pustulation, crustation, cicatrization, and by local and often general reaction, confers immunity in almost all cases, though there are isolated instances when variola has closely followed successful vaccination. Although immunity is not absolutely infallible, vaccination is the greatest triumph of prophylaxis yet achieved. Immunity is not as a rule life-long, as Jenner thought. In some few cases, the individual is permanently protected by one vaccination, and revaccination is unsuccessful. Protection lasts on an average ten years, after which 60 (or 90) per cent. can be once or twice revaccinated. Conversely, the child should be revaccinated in his tenth to twelfth year, and if the vaccination does not "take," it should be repeated. The reaction, fever, induration, and size of the vesicle and scar, are less in revaccination. It is also held that immunity is more certain and enduring when more than one inoculation has been made; the Germans, especially, vaccinate two areas, and, sometimes, even four or six. The nature of the immunity conferred is still disputed, but, unlike the immunity of diphtheria, it is probably due to actual tissue change. Pfeiffer advances the idea that in smallpox the germ endures in the tissues, and in this way maintains the usually permanent immunity. Vaccination immediately after exposure is also thought to prevent or modify an impending attack. Partial immunity is evidenced by mild varioloid (smallpox mitigated by vaccination). The general beneficence of vaccination needs no multiplied statistics. In general, epidemics prevail in communities which are least protected, and conversely, they disappear with systematic compulsory vaccination, as was the case in the German army.

II. **Other Methods of Prophylaxis.**—Isolation hospitals and stations are indispensable, because smallpox patients are infectious in all stages of the disease. Isolation is necessary from the incipiency until the disappearance of the last crust. The dead body is dangerous and the funeral must not be public. The clothes used by the patient must be steamed, and other articles must be washed with bichloride of mercury and fumigated with formaldehyde vapor; Schoch's method has the advantages of not requiring any special form of apparatus, of cheapness, and freedom from danger of fire. The following are required: good quicklime in lumps, commercial sulphuric acid, and ordinary 40-per-cent. formaldehyde solution. For every 1,000 cubic feet of space one pound of formaldehyde solution, one-half pound of sulphuric acid, and three pounds of quicklime are required. The acid and the formaldehyde are first mixed in an earthenware vessel by pouring the acid into the formaldehyde. Then the lime is placed in a shallow vessel in the centre of the room. All openings to the room are carefully closed, the mixture is poured on the quicklime, and the operator leaves the room. The rooms should remain closed for from five to eight hours. Disinfection of the face, hands, beard, and hair of attendants by bichloride solution is imperative. The family should be isolated for sixteen to twenty days.

III. **Actual Treatment.**—As Jaccoud early announced, there is no specific treatment for smallpox. Its *hygiene* and *dietary* are the same as in typhoid. For *headache*, the ice-bag, opium, and hydrotherapy should be resorted to. *Pain* is relieved by opiates.

The initial *vomiting* is difficult to overcome, but its duration is fortunately short Ice-pills, lime-water, iodine, chloroform, morphia, carbolic acid, and bismuth, may be used as they are in typhoid, but mustard plasters should never be applied lest they initiate necrosis of the skin.

Early hydrotherapy is important in giving relief from *fever*. It lessens the dermatitis, though later, when the rash has developed, baths are contraindicated.

Delirium may be relieved by hydrotherapy, by chloral, bromides, hyoscine, and morphia. The latter, in half-grain doses, is invaluable as a routine treatment, since it lessens *itching* and pain and conserves metabolism. There is great danger that the patient, if delirious, may escape, injure himself, or commit suicide. Dr. NcNamara, a classmate of the author, killed himself by jumping through a window which had been carelessly left open near him. Patients should never be confined by straps, for this may cause serious skin trouble (necrosis). It is better to dress them, or even walk them up and down the room.

The *eruption* may be pricked and cauterized with silver nitrate (Arabian method), treated with bichloride or boric compresses (Hebra), covered with mercurial plaster, or with 1-per-cent. carbolic acid and vaseline salve. The latter is the best, because it counteracts the particularly offensive odor. Carbolic salve should be used on the face only, since general use over the body may cause carbolic-acid poisoning. These measures are not specific nor do they prevent cicatrix formation, but they relieve dermatitis and itching. Covering the face from the light with wet compresses seems to lessen the pitting. The crusts should be kept soft with olive oil. The hair should be cut short, and the eyes should be closely watched, and douched frequently with boric-acid solution.

The mouth, the larynx, the circulation, and diarrhœa, should be treated as they are in typhoid fever. For bed-sores, a water-bag or -ring should be used. Sydenham allowed his patients to leave bed as soon as the fever declined.

For sepsis, quinine (*v.* sepsis) and alcohol (*v.* typhoid) are given by the rectum, since dysphagia is always present. Eggnog properly prepared will not injure the throat. During convalescence the crusts should be carefully washed, and the patient may be considered safe only when the skin is clear of the last scab.

CHICKENPOX (VARICELLA).

Definition.—An acute specific febrile contagious disease, chiefly one of infancy and childhood, characterized by an exanthem of vesicles which usually appear in crops.

History.—Varicella was recognized by Heberden in 1767, and by Trousseau, as a disease distinct from smallpox. It is still classified by the Hebra school as smallpox.

Etiology.—It is transferable by direct inoculation, by personal contact, by the air, or by a third person. It is infective while crusts remain. Epidemics of varicella may coincide with, precede, or follow, smallpox. Smallpox does not prevent an attack of varicella nor is the converse true. The disease occurs chiefly in epidemics, although sporadic cases are frequently seen. A second attack is rare. Season has no influence upon the appearance of the disease. It occurs chiefly in the first year of life, or, according to others, 66 per cent. of the cases occur between the years of two and six. It has never been observed congenitally, and some deny its existence in the adult, although such cases do occur.

The actual cause is not determined. Bareggi described a peculiar coccus; Guttmann, the yellow and white staphylococcus; and Pfeiffer and Guinon, a protozoön. It may occur with pertussis, morbilli, scarlatina, diphtheria, pneumonia, or smallpox.

Symptomatology.—I. **Incubation.**—The stage of incubation, like that of measles, lasts thirteen or fourteen days (eight to nineteen days), or three to six days after inoculation.

II. **Stage of Prodromes.**—Prodromal symptoms are generally rare. They are frequent in adults, according to Tordeus, and may be severe. If there are any prodromal symptoms, they last but twenty-four hours (Trousseau). Severe intoxication is rare, but the following have been observed: delirium, convulsions, violent vomiting, hyperthermia, dyspnœa, angina, conjunctivitis, pale urine, tenesmus, dysphagia, muscle pains, lassitude, intestinal irregularity, bloody vomiting and stools, and initial erythema (usually scarlatiniform, rarely morbilliform, in the fifteen cases recorded).

III. **Stage of Eruption.**—The exanthem sometimes appears suddenly after a slight chill, usually in the first twenty-four hours, as red *macules* or *papules*. They are comparable to the typhoid roseolæ, are slightly elevated, disappear on pressure, and are lenticular. Some regress, but most become *vesicular* in from six to twelve hours. The vesicles mature within a day. They are very superficial, and do not occupy the whole roseola, but leave a slight areola about them, which is hyperæmic and not inflammatory as it is in smallpox. In some cases the areola may be absent, the eruption appearing like drops of water sprinkled on the skin. Though usually discrete, Wyss and Immermann describe confluent eruptions. The vesicles may be few or quite numerous; Thomas counted 800 in one of his cases. In shape, the eruption is mostly elliptical or circular. Rare forms are reported with jagged, sinuous edges, when the vesicle overlaps the skin. In size, the vesicles may be very large, and, in exceptional cases, resemble pemphigus; they are alleged to have attained a size of four centimeters.

The eruption begins on the trunk, back, and chest (Osler, Guersaut, Blache); on the face, temples, forehead (Eichhorst, Immermann), and, according to Thomas, rarely fails to appear on the scalp. Trousseau held that there is no point of predilection for the eruption. Henoch finds it often on the part on which the patient lies or where irritation exists. No part can be considered exempt, though most vesicles appear on the trunk, fewer on the face, arms and legs, and the least number upon the hands and feet. The eruption is sometimes grouped, as it is in herpes zoster.

The contents of the vesicle are usually clear at first, and become turbid in thirty-six to forty-eight hours; neutral or alkaline, and never acid. Pustulation may occur (Leube), though this is denied by some. Hemorrhage rarely occurs into the vesicle. The vesicle may contain air after rupture. Umbilication, though rare, may occur (Wyss). Eichhorst denies the statement made by Goruall that the umiblication of variola exists from the beginning of the eruption, while that of varicella occurs late. Crusts may simulate umbilication. That the vesicles of varicella are always unilocular is also denied.

Occurrence in crops is frequently observed; the skin then shows simultaneously macules, vesicles, and crusts. Yellowish-brown crusts form on the third and fourth days and gradually disappear, leaving a somewhat flattened epidermis, which may desquamate slightly. Distinct scars are rare, but may result from scratching or infection, especially on the body or on the face. A few cicatrices are usual in almost every case of varicella (Guinon), but they are smooth, white, and some of them disappear with time.

Enanthems are not frequent. Vesicles are sometimes seen on the palate but may easily be overlooked. Breaking down, they resemble aphthæ. They may precede the exanthem. On rare occasions they are seen on the tongue, cheek, nose, larynx, external ear, prepuce or labia.

Fever.—The fever is slight, if indeed there be any. It is atypica and is not parallel to the eruption. Fever as high as 106.8° is recorded by Thomas. It is very rarely present in the prodromal stage, but may occur with profuse eruptions, or with successive crops; its persistence suggests complications.

General Symptoms.—These are few, but sometimes include dysphagia, cervical and submaxillary swellings, tracheitis, bronchitis albuminuria, or acetonuria.

The entire course of an uncomplicated case lasts from one to two weeks.

Complications and Sequels.—These are usually dismissed with a few words. *Nephritis*, first described by Henoch and since by Vichmann, Unger, Janssen, and others, is generally mild, tubular, and occurs from three to fourteen days after the rash declines. Though it is usually mild, numbers of fatal cases have been lately reported as well as some which have resulted in chronic nephritis, cardiac hypertrophy, and uræmia. *Gangrene* of the skin, seen especially in delicate or tuberculous children, and first described by J. Hutchinson, may occur in the extremities, scrotum, or eye-lids. *Pneumonia* is rare. Other complications which are infrequent, are abscess of the lung, pleurisy, acute miliary tuberculosis, erysipelas, sores in the skin which may lead to tuberculosis, glandular swelling, pemphigus, infantile hemiplegia, osteitis, measles, scarlatina, synovitis, simple or suppurative polyarthritis, peritonitis, otitis media, neuritis, spasm of the larynx, suffocative laryngitis, and the persistence of an unmanagable mixed papular and vesicular eruption on the palms of the hands and soles of the feet.

Diagnosis.—The diagnosis is very easy if the case is seen early. Differentiation from smallpox has been considered. There is usually no fever, the eruption is more vesicular, without halo, larger, but less in number than in smallpox. In *pemphigus* the vesicles are larger and run a slower course of weeks or months. *Miliaria rubra* are preceded by sweats, occur chiefly on the covered parts, have acid contents, are smaller than the varicellous eruption and disappear more rapidly. Sometimes consideration must be given to molluscum contagiosum (soft, umbilicated tumors with white granular contents); prurigo varicelliformis (with crop-like eruptions at longer intervals); eczema vesiculosum, which always occurs with great itching; herpes, always appearing in groups and often along nerve-trunks; varicellæ syphiliticæ, and medicinal rashes from cantharides, bromide, arsenic, and iodide.

Treatment.—Active treatment is rarely indicated.

SCARLET FEVER (SCARLATINA).

Definition.—A specific infective disease of unknown bacteriology, characterized by sudden onset, fever, diffuse exanthem, and angina.

History.—It was recognized by Ingrasseas and Coyttar in the sixteenth century, but was first fully described by Sydenham (1660); before this, it was known as measles. Scarlatina was introduced into America in 1735.

Etiology.—The etiology is unknown. Class described a diplococcus, but this has not been confirmed. Babes, Babinsky, Pearce, Wright, and others, have found a streptococcus in 58 per cent. of fatal cases, and, while their results in no way prove that it is the cause of scarlatina, it is probable that tthe streptococcus is the most important factor in the production of the complications and in their mortality. Hektoen, Weaver, and Ruediger, in 100 cases, found it in the blood in 12 per cent. Combinings everal statistics, the streptococcus as found in about 70 per cent. of the fatalities; *i.e.*, it is the cause of the malignancy of the disease, but not of the disease itself. The organism is also found in diphtheria, measles, and variola. The virus of scarlet fever produces severe necrosis, but no suppuration. Mallory describes a protozoön, the *cyclaster scarlatinalis.*

Susceptibility is not universal; only 38 per cent. of children and 5 per cent. of adults exposed to infection acquire the disease. Susceptibility varies, also, from time to time in the same family.

So-called surgical and puerperal scarlatina, noted in 1864, by Paget, are, in the vast majority of cases, sepsis, erythema congestivum, erythema toxicum, or drug rashes. Alice Hamilton (1905) collected 174 cases from the literature, and concludes that these forms are mostly septic, and occasionally mere coincidents.

The virus circulates in the blood, whence the possibility of fœtal infection even though the mother may escape. The mother may contract the disease and the child may escape. It is usually held that the disease is probably not infectious before the eruption, and that the virus is especially active during desquamation. Aaser holds, and justly, that the throat secretions are most dangerous. The streptococci may be spread by coughing (Hamilton). Infection is often spread even two months or more after desquamation is complete In no other disease is the virus so tenacious; it may persist as long as ten years in clothes, furniture, and on the walls. Light forms are as contagious as severe ones. Inoculations, from the living subject and from the cadaver, have occurred. Leube acquired the disease by a cut during an autopsy upon a scarlatina case. The entrance is not known; but it is probably through the throat.

Infection may be spread by servants, by physicians, by any third person, and by means of toys, books, clothes, and milk, and in schools but the mode of infection is often unknown. One attack usually confers immunity, but second, and even third, attacks are known. In some individuals there appears to be a temporary or permanent immunity. Scarlet fever occurs at all seasons, but probably more often in the autumn and winter. Scarlatina is observed more in the cities and measles prevails more diffusely through the country. Scarlatina is more permanent than measles. When it becomes epidemic it i likely to spread widely, and usually has a mortality increased three or even thirtyfold.

Ninety per cent. of cases occur under ten years of age (Murchison) although adults are not exempt. It is rare in the first year of life The sexes are equally involved.

Scarlatina sometimes occurs with other infections, such as diphtheria or measles, and less often with variola, varicella, herpes, mumps pertussis, erysipelas, or typhoid.

General Symptomatology.—1. **The Stadium Incubationis.**—The incubation, with no noticeable symptoms except perhaps anorexia or slight depression, lasts from two to four days, though sometimes one to seven days.

2. **The Stadium Invasionis.**—The invasion lasts one day. Symptoms begin suddenly, with perhaps a chill. This is followed by (*a*) headache, dulness, convulsions, and delirium; (*b*) vomiting, early and characteristic, which occurs in scarlatina more often than in other diseases of childhood except pneumonia; (*c*) sudden elevation of temperature, even to 105°, with disproportionate pulse-rate, 120 to 150, with dry, burning skin, and febrile urine; (*d*) dryness, redness, burning, pain in the throat, dysphagia, and intumescence of the cervical glands.

3. **The Stadium Exanthematicum.**—The exanthem lasts four to six days. It appears on the second day, first below the clavicles, on the upper trunk, and on the neck, then on the extremities, and least on the face and scalp. The skin about the mouth is pale. The exanthem pales on pressure. It consists first of small red spots (swollen hair follicles), the size of a pin-head, which fuse as the skin swells, and result in an intense diffuse lobster-colored erythema. The tongue swells, becomes covered with a grayish-yellow coating, is red on the tip and edges, and shows swollen red papillæ—"cat's tongue," or "strawberry tongue." Later the whole tongue becomes red and dry. On the tonsils and palate a grayish-yellow coating and confluent spots appear. The nostrils become slightly swollen, and redness of the conjunctivæ develops with secretion. Some rales over the larger bronchi, a slight cough, swelling of the cervical and other glands, and some albuminuria, may be noted.

4. **The Stadium Desquamationis.**—The desquamation lasts several weeks, though the rash itself lasts but three to six days. Desquamation begins first on the face, from the sixth to ninth day. The glandular swelling disappears, the fever falls by lysis, and convalescence is established unless complications intervene.

Special Symptoms, Complications and Sequels.—These are given in detail because of the extreme variability in the clinical symptoms:

1. **The Fever.**—The fever is less typical (Wunderlich) than in most exanthemata; *e. g.*, smallpox and measles. In the invasion there is a rapid rise to 104° or 105°. The fever is usually somewhat higher on the second day, with slight morning remissions and gradual subsidence when the rash is mature.

In the most severe types there may be low fever or no fever at all, or it may rise to 107° or 109°. We usually note a close parallelism between the abundance of the exanthem and the height of the fever. The temperature should be taken during one month after the rash, lest such complications as sloughing of the throat, pleuritis, endocarditis, pericarditis, synovitis, or adenitis be overlooked. A long course of fever may exceptionally occur without complications, but it is rather ominous.

2. The Eruption.—The eruption is often atypical. Typically, it begins in the form of light-red, pin-head-like points which become darker as the skin swells and the eruption diffuses. It then appears as an even red, and on close inspection is found beset with small points. These are the first part of the exanthem to develop, and the last to go. The eruption, then, first consists of red spots, especially in the axilla and groin; second, of red spots in a diffuse erythema; and lastly, of red spots again. This minute papulation is a very important aid to diagnosis in the second week. There may be minimal white lines in the eruption, but rarely ever spaces as there are in measles. Fürbinger compares the skin in measles to splashes of red paint, and in scarlatina to an even red coating. The skin is sometimes vesiculated. Though it spreads less regularly than in measles, the eruption appears first on the neck, below the mastoid process, and below the clavicles, then upon the head, trunk, arms, and hands, and lastly upon the legs and feet. It is most intense over the extensor surfaces of the joints and on the trunk, and is least developed on the face and scalp. The palms of the hands and soles of the feet are usually free. The pallor around the mouth contrasts vividly with the scarlet cheeks and minutely papulated forehead. This pale area is circular or triangular, bounded by the nasolabial folds above and the chin below. Though not pathognomonic, it is always valuable in differentiation, and is due possibly to vascular spasm. The eruption is occasionally roseolous about the ankles and wrists. The skin is always swollen, usually pales on pressure, and sometimes itches, or is wet with sweat and is vesiculated. The eruption is hyperæmic, and it therefore pales on pressure and disappears after death, unless there are punctate hemorrhages, which are not uncommon in severe types of the disease. After twenty-four hours of the eruption, the tension disappears, and the bright-red color becomes a yellowish-red. Pathological changes in the skin, according to Unna, consist of hyperæmia, especially in the papillæ, but rarely œdema, inflammation, or hemorrhage. After complete florition the skin becomes dry, and desquamation begins. This is usually bran-like as it is in measles, on the head, face, neck, and trunk, and it is more scale-like and lamellar on the hands and feet, complete casts of which are sometimes shed. The scales are often large in proportion to the intensity of the eruption. In severe cases even the hair and nails are shed, and the nails become furrowed. Desquamation results from a slight exudation of serum between the layers of the skin. It begins on the cheek with the "rouge-and-powder" appearance, and is usually first observed when the fever and angina decline, but it may be delayed from two to seven days later. The skin may present a "pin-hole" or "worm-eaten" aspect before desquamation begins. Its duration is most variable, usually lasting from one-half to three weeks, or even seven or eight weeks (Trousseau). Other occasional skin complications are: Bed-sores, gangrene, œdema (either with or without nephritis), and eczema on the face, scalp, o ears. This is usually impetiginous. *Anomalies of eruption: Scar* *latina lævigata* is the typical rash which has been described above. Other forms are *scarlatina lævis* (rudimentary or localized eruption); *scarlatina sine exanthemate* (the rash being wholly absent); *scarlatina papulosa* (marked papulation); *scarlatina miliaris* (promoted by sweating, but also the result of exudation between the rete Malpighii and the epidermis); *scarlatina variegata* (like measles, though not equally elevated); *scarlatina hemorrhagica* (usually ominous, and at times part of a general hemorrhagic diathesis). In general, irregular forms are dangerous.

3. **The Throat.**—The throat changes are the most constant lesions in scarlet fever, and constitute the internal eruption (enanthem). The disease begins in the throat with dryness, uniform redness, and invariably with early swelling. The throat very soon becomes speckled with fine red points like the skin papulation. Though always present, it varies in intensity. All forms, from simple angina to diphtheritic or gangrenous inflammation, may be seen. (*a*) In mild cases, a simple catarrh with little swelling, a thick mucous coating, and more or less reddening of the palate and tonsils, develop. (*b*) In moderate cases, there is, in addition to the above, more swelling of the palate, and tonsillitis. (*c*) In severe cases, membrane, small abscesses, gangrene, small hemorrhages, sloughing, and, perhaps, sudden hemorrhage, œdema, adenitis, periadenitis, or cellulitis, develop. Severe inflammation in the throat occurs in 65 per cent. of the cases (Von Ranke).

Clinical and anatomical diphtheria are often confused. "Necrotic inflammation" in scarlatina is that caused by the virus of scarlatina alone. Genuine diphtheria may accompany or follow scarlatina; of these, post-scarlatinal diphtheria is the more dangerous (Trousseau).

DIPHTHERITIC MEMBRANE.——*vs.*	——SCARLATINAL MEMBRANE.
Firm, adherent.	Softer, loosened in fragments.
Yellow-gray.	Brownish tint.
Deep ulceration rarer, in more severe cases only, and later.	More frequent and earlier (even in twenty-four hours).
Phlegmon rare.	Not infrequent.
Laryngeal membrane (croup) frequent and deeper.	Rare, membrane thinner; lung complications rare.
Bacteriologically, the Klebs-Loeffler bacillus.	Streptococcus (Klebs-Loeffler bacillus found only as a complication).
Paralysis frequent.	Rare, except local paralysis due to local swelling of pharynx.
Recurrence frequent.	Infrequent.

Since the bacteriological cause of scarlatina is undetermined, it is doubtful whether the pyogenic cocci found in the throat are integral or complicating.

4. The Respiratory Tract.—Though often unaffected, the respiratory tract may become secondarily involved by a descending process. The nose is involved in direct ratio to the severity of the throat developments, such as necrosis, diphtheria, and gangrene. Acute œdema of the glottis is infrequent. The bronchial mucosa is more often involved than is usually recognized. Bronchopneumonia, acute lobar pneumonia (usually in the upper lobes and coëxisting with nephritis), gangrene, infarcts, inflammatory œdema and hypostasis, are not common. Pleurisy, described especially by R. P. Howard, occurs in 5 per cent. of the cases. It is often overlooked, is likely to be severe, is very frequently purulent, and often occurs during convalescence.

5. The Heart.—The heart may be greatly damaged by the scarlatinal virus, and is involved in 35 per cent. of the cases, not including the cases of sepsis and nephritis (Schmaltz). Endocarditis is frequently mural, whence its frequent latency, but it may result in chronic valvular disease, especially of the mitral valves. "After rheumatism, scarlatina is the most frequent cause of endocarditis" (Leube). Tachycardia may often be observed from toxæmia or acute infective myocarditis. With increased arterial tension due to kidney disease, the heart is slowed, and hypertrophy may result, unless pancarditis. (endo-, myo-, and pericarditis), exist. Pericarditis is usually septic, Disquieting cardiac dilatation may be seen in severe cases, accompanied by the usual evidences of acute cardiac insufficiency; it may occur during the fever (Rhomberg), or in convalescence (Schmaltz). Dilatation and hypertrophy are much more frequent in children than in adults, and often complicate renal lesions. Leukocytosis develops early and may become extreme in fatal cases. It has been recently stated that the leukocytes are normal in number, but the polymorphonuclears are absolutely and relatively increased two or three days after the eruption. The eosinophiles are also increased. The hæmoglobin and red cells suffer an early and increasing reduction. The red cells average 4,000,000. Hemorrhagic diathesis seldom occurs except as a result of sepsis.

6. The Digestive System.—The tongue is swollen, indented, broad, and thick. Its papillæ become larger and separated from each other. Its coating is first grayish, and only the edges and tip of the tongue are reddish, but later the coating disappears, leaving the tongue very red, dry, and glistening, on which the papillæ appear granular and warty—the "strawberry," "raspberry," or "cat's tongue." This appears from the third to the fifth day and lasts a week. It is quite pathognomonic, according to Leube, and is only second in importance to the angina. McCollom, of Boston, found it the only constant sign in 1,000 cases of scarlatina. The breath may often be sweet and of a very heavy odor. Diphtheritic ulcers and secondary necrosis in the stomach, œsophagus, and intestines, have been found. Vomiting may result from nephritis, brain toxæmia, or coughing. The liver and spleen may be slightly swollen. There may be abdominal pain. Constipation is usual. Diarrhœa, however, may result from (*a*) simple catarrhal enteritis, which is transitory and not unfavorable (Litten); (*b*) dysentery, with tenesmus and blood; (*c*) the so-called "scarlet-typhoid" of Thomas, which is accompanied by tympany, hemorrhage, fluid fæces, very rapid pulse (herein differing from typhoid), and lethal outcome. Its resemblance to typhoid is also heightened by the autopsy findings of splenic intumescence, and hyperplasia with ulceration in Peyer's patches.

7. **The Kidneys.**—The urine is febrile. It will respond to Gerhardt's reaction and to the diazo test, though to a less degree than in measles. Increased phenol and beta-oxybutyric acid may be found. Albuminuria occurs in 77 to 92 per cent. of all cases. Early renal changes may occasionally be observed. The "initial catarrhal nephritis," which runs a short course, is accompanied anatomically by hyperæmia, parenchymatous changes, some fluid in the glomeruli, a few cylinders in the tubules, and little interstitial involvement.

Nephritis usually develops in the third week (ninth to fortieth day). Its frequency varies with the epidemic, between 5 and 70, or even 90 per cent. No apparent relation exists between its frequency and the severity of the infection. No single anatomical form is invariable, but the glomerulonephritis of Klebs is most frequent. It is characterized anatomically by hyperæmia, increased consistence, and such glomerular changes as gray color, diffuse fatty granular degeneration, increase of nuclei, and increase by 50 per cent. in the size of the tufts, which become sausage-like solid masses. We also note exudation of leukocytes about the afferent vessels, multiplication of capsular epithelium with occlusion of the lumen, hyaline alteration of the vessels, and small and early interstitial changes, and later connective-tissue growth. Friedländer observed septic interstitial nephritis and the large variegated kidney, in cases accompanied by diphtheria, or phlegmonous complications.

Hydrops in the vast majority of cases is renal and shows the characters of renal dropsy. It appears first under the skin, and the cutaneous blood- and lymph-vessels are altered by the virus. It shifts its location, varies without cause, and may produce hydrothorax, ascites, œdema of the larynx, of the lungs, or of the genitalia. The older writers claimed that hydrops occurred without nephritis, and that kidney inflammation might exist without appropriate urinary alterations. Such a view can be proved by autopsy only. The *temperature* is inconstant. There may be lumbar pain, vesical tenesmus, and uræmic symptoms, such as headache, twitchings, convulsions, wide pupils, amaurosis, disturbance in motility, in sensation, and in cerebration, coma, involuntary evacuations, vomiting, diarrhœa, cardialgia rapid pulse and breathing, and pale skin tinged with cyanosis. The retinæ are usually normal. Many convulsions usually imply a lethal termination in coma. The prognosis is more favorable if the pupils are not immobile. The urine is decreased or suppressed, is heavy with sediment and albumin (averaging 0.5 per cent.), is poor in urea, turbid (specific gravity 1030), and is full of casts of all types—especially, long, flat, band-like, twisted, and frayed casts. Much oxalic and uric acid is found and, in many cases, blood. Uræmia, hydrops, and intercurrent inflammation, are always dangerous. Convalescence is the rule after four to six weeks (eight to eighteen months), and the nephritis rarely becomes chronic.

8. The Special Senses.—Decreased secretion in the eye is common. Cerebral complications promote inflammation in that the eyes close less frequently. Catarrhal conjunctivitis, diphtheritic inflammation, involvement of the lachrymal sacs, herpes or necrosis of the cornea, panophthalmitis, muscular paralysis, and disturbance in accommodation, are possible complications. Uræmic amaurosis, neuroretinitis septica seu nephritica, and retinal hemorrhage, are rare. Middle-ear inflammation (11 to 33 per cent. of the cases) usually begins before desquamation, but may be observed later. It may easily be overlooked, and is accompanied by fever, pain, glandular swelling, difficult hearing, tinnitus, a reddened, glistening, sunken membrane, and, in children, by some brain manifestations. It is quite frequent in early life and rare after the fifteenth year; 10 per cent. (even 42.6 per cent.!) of cases of acquired deafness are said to result from scarlatina.

9. The Lymphatic System.—The lymphatic system is generally involved; *e. g.*, glands, spleen, follicles, and in severe cases, all the lymphatic structures, are affected by the sepsis. The "collar of brawn" results from cervical adenitis and periadenitis. Gangrene, retrosternal abscess, and jugular phlebitis with sepsis, occur in desperate cases After recovery the neck may remain swollen for a long time.

10. The Bones and Joints.—Scarlatinal "rheumatism" is found in (2 to 12) per cent. of the cases, usually in the second or third wee (with desquamation and subsidence of the fever), and usually last three or four days. It attacks the smaller rather than the larger joints the wrists rather than the elbows or shoulders, and the ankles rathe than the knees. It develops with pain, and is not so migratory a ordinary rheumatism. It may leave no autopsy findings, or it ma become purulent. Swelling of the joints does not always occur. I is claimed that swelling occurs in those who have had rheumatism and that it may respond to salicylates. It occurs in old rather than i young subjects. Endocarditis follows in less than 3 per cent. of scar latinal rheumatism. The prognosis is usually good. Bone involve ment is rare.

Scarlatinal sequels are anæmia, hemorrhagic diathesis, retarde development, and decreased physiological resistance. Tuberculosi rarely follows scarlatina, because the two diseases are rather ar tagonistic. Latent sepsis with brain foci, valvular disease, chroni nephritis, chronic adenitis, otitis, mastoiditis, deafness, meningitis, hem plegia, cerebral venous thrombosis, facial paralysis, ascending spin paralysis, chorea, and exhaustion psychoses, are fortunately the mor rare complications and sequels.

Diagnosis.—Typical cases are easily determined when an epidemi prevails, and when the eruption is not considered of chief diagnosti reliance. The criteria are as follows:

1. Sudden onset and rapid development, with fever and vomiting.

2. Angina, pain, redness, punctate spots in vault of the mouth, swelling and dysphagia, which are practically always present. Severe symptoms in the throat, especially during an epidemic, are suspicious.

3. Strawberry tongue, which is practically constant.

4. Exanthem. This appears about the clavicles, and shows a characteristic progression. The face is less involved, and the mouth is free; it is intense on body; papules then erythema develop; there are no free spaces; it results in desquamation.

5. Lymphadenitis. In this, the cervical are less important than the inguinal glands.

6. Fever, hydrops, albuminuria, and nephritis, in atypical cases.

7. Scaling, tender joints, and albuminuria, are strong indications of scarlatina if the initial symptoms were not observed or were not clear; they help in making a retrospective diagnosis.

Diagnosis of Atypical Scarlatina.—1. Severe scarlatina. (*a*) Anginose form. This resembles septic diphtheria, with marked toxæmia, necrosis, and adenitis. (*b*) Atactic form. Violent early intoxication; high fever and death even on the eleventh hour of the disease (Trousseau), before the eruption appears; blood dissolution, vomiting, diarrhœa, convulsions, coma, and an excessively rapid, weak heart, are the chief symptoms. (*c*) Hemorrhagic scarlatina. This is characterized by severe fever and brain symptoms at the onset; incomplete exanthem, necrosing angina, marked glandular and splenic swelling; subcutaneous, serous, and mucous hemorrhages with exulceration. It is usually lethal after ten to twenty hours from hyperpyrexia, exhausting vomiting, diarrhœa, sopor, delirium, convulsions, and dyspnœa. It is encountered especially in reduced subjects under two years of age.

2. Very light cases may present (*a*) all the usual signs, though but mildly developed; or (*b*) some symptoms may be absent (*scarlatina fragmentaria*), as in cases with no temperature (*scarlatina sine febre*), no rash (*scarlatina sine exanthemate*), or no angina (*scarlatina sine enanthemate*). In some atypical cases a definite diagnosis is impossible, as in acute nephritis with uræmia, fever, sore throat, and gastro-enteritis, without the characteristic rash. Recurrent forms are not frequent. They occur in the middle of the third week, and result from incomplete elimination of the virus during the first attack. Many of these cases are septic complications rather than actual recurrences. According to Koerner, only forty relapses are on record.

The differentiation is as follows:

SCARLET FEVER.——*vs.*	——SEPSIS.
Bright-red erythema, with small red papules.	A very deep purple-red rash, sometimes spreading over entire body.
The eruption is much the same in both diseases, the same places being exempt.	
Miliaria are rare.	Frequent.
Rather typical desquamation.	Desquamation also observed, but less frequently.
Criteria (*q. v.*): angina, tongue, abrupt onset, glands, etc.	Etiology (lacking in cryptogenetic type), chills, sweats, fever irregularity, clinical course, polymorphous exanthems, etc.

Diphtheria is often differentiated with difficulty. A simple erythema is sometimes, though not very often, observed in diphtheria. When present it is darker, is located more on the trunk, and is more transitory than in scarlatina (see THROAT COMPLICATIONS).

Drug rashes, from belladonna, quinine, iodide, chloral, or copaiba, may be easily differentiated, if the cardinal signs rather than the rash alone are considered.

Measles and *German measles* (*q. v.*). *Acute exfoliating dermatitis* also begins with fever, with a spreading erythema, and very early desquamation, but the other cardinal criteria of scarlatina are lacking. *Erythema* is more transitory; the points are absent; it occurs with less fever and without angina or adenitis.

Erysipelas presents a sharp border, œdematous swelling, marked pain, and a slow advance.

Prognosis.—This should always be guarded for at least one month. The outcome often depends on the *genius epidemicus*, since the mortality ranges between 2 and 40 per cent. The death-rate averages about 10 per cent. The prognosis is least favorable in hospital cases and in children. Most deaths occur under six years; in children under five years, the mortality is 20 to 30 per cent. (Holt). In the first year of life the disease is very fatal.

The social status, severity of the infection (hemorrhagic forms, severe nervous toxæmia), complicating sepsis, necrosis in the throat, otitis, nephritis (uræmia, hydrops), heart complications (high pulse-rate being always unfavorable), respiratory complications, and laryngeal obstruction, are factors in the prognosis.

Treatment.—There is no specific therapy and the disease runs its self-limited course.

I. **Prophylaxis.**—Prophylaxis is equally imperative in both severe and light cases. Indeed, the light or ambulatory forms are often the more dangerous. Isolation is important, because susceptibility, especially in the young, is not universal and the virus is less penetrative than in measles, and it is therefore possible to circumscribe its diffusion. Care should be taken to prevent transmission through schools and by means of fomites and by third persons. The infected room should be kept empty for one month, and should be exposed to the wind and sun for several weeks. No susceptible person should use the room for several months. The physician should wear an operating gown and carefully wash his face, beard, and hands, after leaving the sick-room. Quarantine should be maintained from six to eight weeks. Even this is probably too short a period. A sheet saturated with carbolic acid or formaldehyde, and hung before the door, is of no special value. Formaldehyde fumigation has been discussed under smallpox. Care in surgical and puerperal cases is important, but the danger has been overestimated. All discharges should be disinfected. Bichloride wrappings should be placed about the dead body, and the funeral should be private.

Schools should be closed during epidemics. In hospitals the cases of scarlatina with streptococcus infection should be separated from ases of simple scarlatina. Sound children should be sent away from the house and isolated lest they spread the disease. If they remain at home, about one-quarter of them will contract scarlatina (Chapin).

II. General Treatment. 1. POSITION.—The prone position in bed is imperative in all cases, both during the disease and in the first two weeks of apyrexia.

2. ROOM.—The room should be light and well ventilated, though somewhat warm, especially in convalescence. A grate fire is advisable, and the windows should be open at the top. The air should be kept dry, since dampness tends to promote nephritis. During the fever the skin should be covered with linen, and in convalescence with wool.

3. SKIN.—The eruption should be brought out, when necessary, by pilocarpine (Demma) or with mustard baths. During the fever, the patient should be washed daily with warm water and soap, though unnecessary exposure should be avoided. The tension of the skin is thus lessened, the fever decreased, and toxæmic symptoms, such as insomnia and restlessness, are allayed. During desquamation, oil-rubs may be employed to decrease the risk of infection by dissemination of dry scales. They are, however, now considered deleterious, as they decrease the functions of the skin, which are of great importance when the kidneys are involved. Besides, infection is more probably carried by means of throat secretions, than by desquamation. Soap and warm water serve equally well. The author is opposed to antiseptic soaps because of the chance of absorption which may result in renal injury.

4. FEVER.—The following is a harmless fever mixture:

℞

Liquoris ammonii acetatis		℥iss.
Tincturæ aconiti		♏xxiv.
Syrupi limonis		℥ss.
Aquæ	q. s. ad	℥vj.

Misce, fiat mistura.

S.—One to three teaspoonfuls every four or five hours, according to age.

This mixture tranquilizes, promotes the rash, and allays fever. Hydrotherapy is useful, such as the warm sponge or full warm bath. Special care is indicated (*a*) in adynamic cases, because of danger of heart-failure; therefore the full cold bath should always be avoided and the moderately warm bath should be used; (*b*) in pericarditis; (*c*) in stenosis of the air-passages, or dyspnœa; (*d*) in hemorrhagic forms; (*e*) in arthritis; and (*f*) in nephritis.

5. DIET.—Water should be given very freely. A *fluid diet* is indicated by the fever, the percentage of renal complications, and the dysphagia. This may consist of concentrated foods, milk, soups, and jellies. In defervescence, milk-toast, custard, puddings, and bread andbutter, may be given. Eggs may cause albuminuria, and should therefore be avoided. After defervescence, meat, chicken, and fish, may be allowed. Meat is given by some clinicians because the urea is usually eliminated easily. Fruit and vegetables may be given freely

at all stages, if this can be done without injuring the throat. In severe throat complications, rectal feeding is indicated. (See GASTRIC ULCER.)

6. SCARLET RHEUMATISM.—The A.B.C. liniment (aconite, belladonna, and chloroform), warm wool and flannel bandages, are helpful.

℞

Tr. opii	ʒiij.
Acidi carbolici	ʒj.
Tr. belladonnæ	ʒiij.
Ol. camphorati	ʒiv.

M. et S.—May be applied on muslin covered by cotton

Salicylates are beneficial in some cases, but unless given in careful doses may injure the kidneys. If the arthritis is obstinate, aspiration with all surgical care and even drainage may be indicated.

7. ANGINA.—Ice in the mouth, ice-cravats, hot fomentations especially for the enlarged glands, and Dobell's solution as a nasal douche should be given. Care should be exercised in the use of antiseptics lest children swallow them. A 10-per-cent. peroxide of hydrogen, 2-per-cent. chlorate of potash, or 2-per-cent. carbolic gargle, may be used. Formalin gargles are too irritating. Caustics are useless.

For the nose the following formula is excellent:

℞

Acidi carbolici	ʒss.
Acidi borici	ʒss.
Glycerini	℥j.
Tr. myrrhæ	℥j.
Aquæ	q.s. ad ℥v.

M. et S.—Locally, in such dilution that no irritation is felt.

Huebner injects 3-per-cent. carbolic solution into the tonsils (ʒss b.i.d. on each side). A 10-per-cent. nitrate-of-silver solution is used with great advantage in the early stages of the angina. Coincident diphtheria is treated by means of local measures and antitoxin (described under diphtheria), and the antistreptococcus serum may be tried in streptococcic angina.

8. LYMPH GLANDS.—Secondary infection may occur, against which prophylactic care of the mouth and throat is important. Ice should be applied in the beginning only, and later hot boric-acid dressings when the blood- and lymph-streams become stagnant. Iodine and ichthyol salves and surgical intervention are indicated in the order named.

9. OTITIS.—Otitis is treated by copious boric irrigation, given quite hot and at a slight elevation, and followed by equal parts of glycerine and laudanum, 5-per-cent. carbolic acid, or 5-per-cent. cocaine in olive oil. Leeches over the mastoid often relieve the pain. Examination of the drum is rather impracticable in children with small sensitive meatus. There is said to be no harm in awaiting spontaneous rupture, but early puncture under cocaine is much safer. After rupture cotton with a small amount of iodoform or boric powder should be applied.

10. NEPHRITIS.—Nephritis is treated (*a*) according to the old prophylactic rule, by which all cases, severe or light, are confined to bed for at least four weeks. The urine must be watched. An albuminous diet is said not to be contraindicated, since the urea is usually well eliminated, but, according to Jaccoud, the milk diet is valuable in the prevention of nephritis. Irritants, like cantharides, are always to be avoided. Tepid baths lasting a quarter of an hour should be given daily, especially in convalescence. Watch the temperature and the urine in convalescence. Wiodowitz in 102 cases used urotropin successfully as a prophylactic. The number of his cases is too small for generalization, but his results are at least suggestive. (*b*) Treatment of established nephritis. Hot baths increase the sweat and the flow of urine. They should be given twice daily, and may last for even an hour, but the patient must be kept afterward between blankets. Alkaline diuretics, such as potas. acet., pot. bicarb., p. citrat., and infus. tritici repentis, may be given; pilocarpine for a child two years old, $\frac{1}{40}$ to $\frac{1}{30}$ gr., will often relax the skin. (See TREATMENT OF NEPHRITIS.) Calomel must be used with great care as a laxative and diuretic. Digitalis in moderate doses aids diuresis.

℞

Infusi digitalis	ʒv.
Potassii acetatis	ʒss.
Syrupi simplicis	ʒij.
Aquæ	q.s. ad ℥ij.

M. et S.—One teaspoonful every two hours.

High arterial tension should be treated with nitroglycerine. For convulsions, chloroform, chloral, and bromides are valuable. For vomiting, iodine is most efficacious (see VOMITING TYPHOID). Venesection and the administration of digitalis are necessary in severe pulmonary œdema. For marked uræmia, two drops of croton oil in milk or placed undiluted on the tongue, should produce free catharsis in two hours. If not successful by that time it should be repeated. Powdered jalap (gr. xv. to ʒj.) will unload the bowels in less urgent cases. Early evacuation is indicated in pleurisy, if chain cocci are found by puncture.

11. TONICS.—Such tonics as iron, arsenic, and cod-liver oil, are indicated in convalescence. The heart must be watched for weeks, because the heart is very often degenerated.

12. SERUM.—Marmoreck's and other streptococcic sera have given no definite general results, although in four instances the author has seen good results. Everything depends on whether the streptococci are the primary cause or only secondary symbiotic organisms. Again, immunization against one variety of streptococcus does not produce immunity against other varieties. In Vienna, Moser's serum has apparently been efficacious. In the St. Anna Children's Hospital, the mortality was 8 per cent., and in other institutions it amounted to 14.5 per cent. without serotherapy.

MEASLES (MORBILLI, RUBEOLA).

Definition.—An acute, highly infectious disease, characterized b an initial coryza and other inflammations of the air-passages, by rapidly spreading eruption, a fever-curve with a double summit, an by toxæmia.

History.—Its first clear description dates from Rhazes (980–103 A.D.). It is a very old disease and was widely epidemic in the eighteenth century.

Etiology.—(*a*) *Susceptibility* is almost universal; few individual escape (14 per cent. only [Madsen]), and one attack generally confer immunity, though this is sometimes congenital. A second attack i relatively rare, although third and fourth attacks are known to hav occurred. (*b*) *Age*—It is rare during the first five months of life and it occurs most frequently after the first year. Twenty instance of placental transmission to the child are reported. It is more commo in adults than is scarlet fever. In Schwalbe's collection of 16,98 cases, 50.4 per cent. occurred in the first five years; 42 per cent. betwee five and ten years; 5.2 per cent. between ten and twenty years, and 2. per cent. after twenty years of age. (*c*) *Season*—Measles occur mostly in the winter and in the cooler spring months. (*d*) The actua *virus* is unknown. Protozoa are said to have been found by Doehle and bacilli by Canon and Piclicke. The longevity of the virus withou the body is not great. It exists in the blood, tears, skin, and in th bronchial and nasal secretions. Its activity is greatest during the catarrhal stage, but it is also dangerous during the eruption, and possibl even during incubation and desquamation. Hektoen twice succeede in inoculating the disease from the blood of measles patients.

Propagation usually occurs by personal contact, and rarely throug a third person. Aerial transmission is frequent, whence isolation i the home is not always prophylactic. For this reason the diseas usually runs through the family. The most frequent means of dissemination is the school-room. It is more often epidemic than i scarlatina, and epidemics range more widely through the countr They last from four to six months.

General Clinical Course.—The stages are: (I) Incubation of nin or ten days; (II) initial, catarrhal, or prodromal stage of three o four days, with fever, catarrhal symptoms in the eyes, nose, etc., an an enanthem; (III) the eruptive stage, lasting four or five days with renewed fever, intoxication, eruption, and inflamed mucosæ (IV) the desquamative period of variable duration.

I. **Incubation.**—The incubation lasts nine or ten (or fourteen days.

II. Then the **catarrhal** (initial, prodromal, invasion) **symptom** appear. These are chilly sensations, malaise, frontal headache, redness of the eyes, lachrymation, photophobia, coryza, and sneezing; dryness, redness, and pain in the nose, in the throat, and in the bronch

PLATE II.

FIG. I.

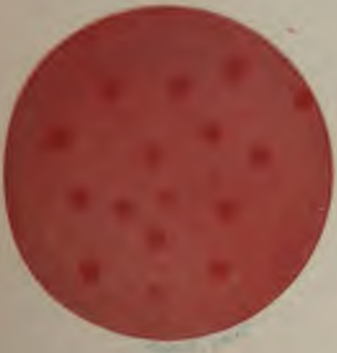


FIG. 2.

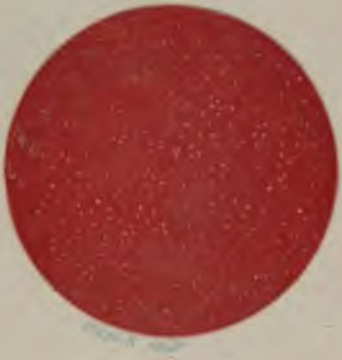


FIG. 3.

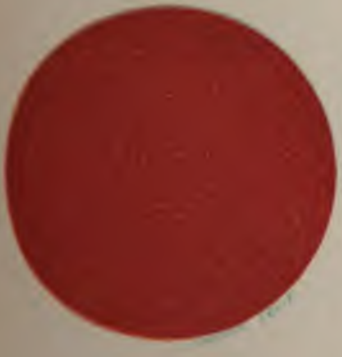
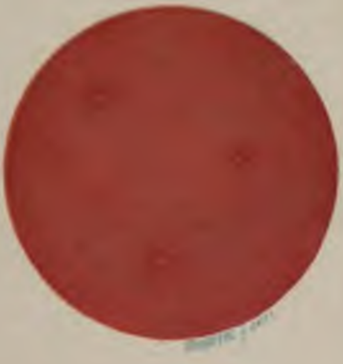


FIG. 4



The Pathognomonic Sign of Measles (Koplik's Spots).

Fig. 1.—The discrete measles spots on the buccal or labial mucous membrane, showing the isolated rose-red spot, with the minute bluish-white centre, on the normally colored mucous membrane.

Fig. 2.—Shows the partially diffuse eruption on the mucous membrane of the cheeks and lips; patches of pale pink interspersed among rose-red patches, the latter showing numerous pale bluish-white spots.

Fig. 3.—The appearance of the buccal or labial mucous membrane when the measles spots completely coalesce and give a diffuse redness, with the myriads of bluish-white specks. The exanthem on the skin is at this time generally fully developed.

Fig. 4.—Aphthous stomatitis apt to be mistaken for measles spots. Mucous membrane normal in hue. Minute *yellow points* are surrounded by a red area. Always discrete.

and cough, which develops after one day. Vomiting may occur. The respiratory symptoms are due to a mucous-membrane enanthem (Rehn) which is said to be seen at autopsy on other mucosæ. Examination of the throat reveals Koplik's spots (Plate II) in 86 to 97 per cent. of cases, described by him in 1886, and by Filatow in 1885. They are practically constant on the buccal mucosa near the molars, and are found on the first day. They are round, of a bluish-white color, and are surrounded by a reddish areola. Early catarrhal symptoms are more common in measles than in any other acute infection of childhood. The initial fever reaches 102° or 103°, and declines gradually during the third and fourth days. Occasionally there is a prodromal rash, which is mostly hyperæmic, though rarely macular or papular.

III. **The eruption** begins on the fourth day with papules on the temples, cheeks, forehead, and then on the back, wrists, forearms, and on the anterior surface of the body and legs, in order of progression as named; with the appearance of the eruption, the fever again rises. The eruption disappears on pressure or on lateral stretching of the skin, and is palpable and superficial. There are always islets of normal skin, even in the thickest eruption. The glands are usually enlarged and there is more or less marked sweating.

The general and local symptoms usually abate on the sixth day, and the fever falls by crisis. In severe cases, epistaxis, diarrhœa, cerebral intoxication, bronchiolitis, pneumonia, hypostasis, cyanosis, and asphyxia, may be observed, and death may result from lung involvement, mixed infection, or decreased physiological resistance.

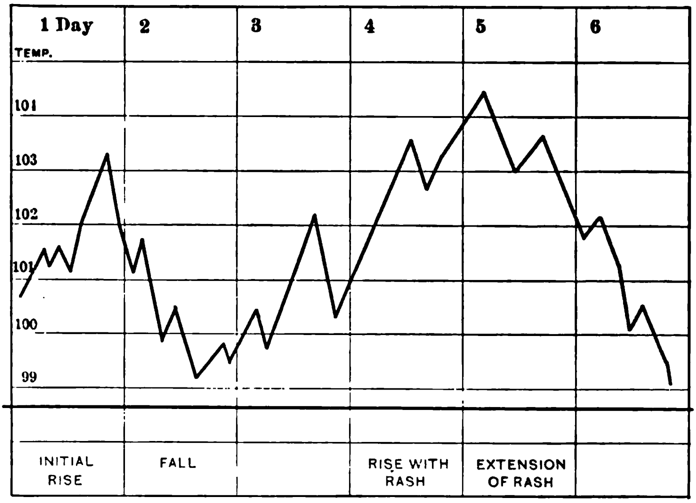
IV. **Desquamation.**—In the desquamative stage the skin pales, becomes slightly pigmented, and scales form. Convalescence is established in eight or ten days.

Individual Symptoms in Detail.—I. **The Eruption.**—It is essentially the same on the skin and on the mucosæ. In the mouth the mucosa is red on the first day of fever, and Koplik's spots are found; on the second day redness without swelling is especially noted on the tonsils and palate. The mucous eruption may reach the larynx, intestines, and genitalia. Though the enanthem tends to spread, the skin eruption (exanthem) is circumscribed. The rash is due to hyperæmia rather than to actual exudation. It fades on pressure; in some cases the roseolas do not fade on pressure, but leave a brown-colored area, especially in the nodular forms of measles. The roseolæ usually are discrete, are slightly elevated, especially in the centre, from spastic contraction of the bloodvessels and œdema, are light to dark red in color, are round or crescentic in shape, and average 2 to 6 millimeters in diameter. They always leave islets of normal skin between the independent spots of eruption, even in the most diffuse types. The eruption follows a descending order and reaches the lower parts of the body last. When the eruption develops on the body first, the course is usually anomalous. Itching may be noted, and sweating may be marked. Desquamation follows the same order as the eruption, and is usually fine. Scars never result. The eruption is rudimentary in those who are debilitated by chronic diseases, and is exuberant in the healthy.

The varieties are: (1) *Morbilli læves:* the usual form, with small elevations. (2) *M. vesiculosi seu miliares.* (3) *M. papulosi,* nodular measles: the eruption may feel slightly "shotty," but such induration is always superficial. (4) *M. confluentes seu conferti.* (5) *M. hemorrhagicæ,* benign or malignant (black measles; typhoid, septic, adynamic types). (6) *M. sine exanthemate; sine enanthemate.* "Recession" of the eruption (fading or "striking in") is usually a sign of heart-failure.

II. **The Fever.**—The curve is not absolutely typical. It begins suddenly without a chill, and falls on the second day, perhaps even to normal, rises again with the eruption, and reaches its maximum in thirty-six hours, thus giving a curve with a double summit. It usually falls by crisis before the eruption disappears. Fever, after the eruption begins to fade, indicates complications. In rare cases fever is absent. The temperature is normal, even subnormal during desquamation.

Fig. 9.



Fever curve in measles.

III. **The Respiratory Tract.**—Nasal catarrh is of special momen in sucklings. Epistaxis, fibrinous rhinitis, and diphtheria, are uncommon complications. Catarrhal laryngitis may occur, accompanied b hoarseness and false croup; 50 per cent. of the cases suffer from erosions or ulcerations of the larynx (Rilliet and Barthez), 16 per cen from pseudomembranous laryngitis, and 15 per cent. from simple ir flammation. Laryngeal complications are not necessarily febril Bronchial catarrh cannot be excluded by the absence of rales.] may reach the smaller tubules and usually, but not always, occurs lat in the disease. It produces a clear, stringy, viscid sputum whic may later become purulent and nummular. The bronchial glanc are always swollen. Simple atelectasis may occur, and may occasion indeterminate breathing and dulness, which decreases with change of posture or deep breathing. Bronchopneumonia occurs in 6 to 12 per cent. of the cases and is the most frequent fatal complication. It differs from other lobular pneumonias in its rapid development, and its greater tendency to pus formation, which result from the fact that the measles-catarrh promotes the action of the pneumococcus and pyogenic cocci. The temperature is often no higher than in bronchitis. There are often bronchial breathing and other physical signs. In some cases they may not be detected clinically. Toxæmia and carbon-dioxide narcosis accentuate the patient's distress. Bronchopneumonia is rapid in development, but slow or sometimes incomplete in resolution. It may result in interstitial pneumonia, gangrene, or tuberculosis. The latter is a dreaded sequel of measles. It occurs (*a*) as a miliary invasion, and develops in the brain much more frequently than in the lungs. It is caused by the bacilli from old caseated bronchial glands which are released by solvent substances which reach them from the lungs by way of the lymph-vessels. It also occurs (*b*) as a fresh tuberculosis developing upon a bronchopneumonia. This variety may heal or apparently recover, to break out anew at a later time. Croupous pneumonia is as infrequent as pleurisy. Pleurisy is in most cases secondary to lobular pneumonic foci and may cause effusion.

IV. **Cardiac Symptoms.**—These are rare. Accidental bruits may be heard. Toxæmia may, in exceptional cases, cause myocardial degeneration.

V. **The Digestive Tract.**—The tongue is coated. There is thirst and aphthæ, and stomatitis may be observed. As a result of an intestinal enanthem, diarrhœa (which is important prognostically), mucus in the stools, and (anatomically) swelling of Peyer's patches, have been described. The liver is seldom swollen.

VI. **The Nervous System.**—Nervous symtoms, such as delirium, sopor, coma, or convulsions, may, in rare cases, result from severe toxæmia, even though the delicate nervous system of children is seldom involved in measles. Nervous symptoms may occur in protracted pneumonia, and doubt may exist as to whether they are caused by the measles, miliary tuberculosis, tuberculous meningitis, or carbon-dioxide narcosis. Paralyses, myelitis, muscular atrophy, polyneuritis, and polymyositis, are most infrequent.

VII. **Genito-Urinary Tract.**—Acetonuria and albuminuria are frequent. Ehrlich's diazo reaction occurs in nearly 100 per cent. of the cases. Nephritis is very uncommon. It may occur during desquamation, but is more often due to complicating diphtheria, or tuberculosis. Gangrene of the genitalia has been observed.

VIII. **Other Symptoms.**—The spleen and lymph-nodes are but slightly swollen; bone and joint complications are rare; keratitis is infrequent; otitis media occurs in a small percentage of cases, although some observers have found it in as high as 50 to 60 per cent. It is said that streptococci in the conjunctivæ are much more frequent in severe than in mild cases. The red cells are little altered, while the leukocytes show a relative lymphocytosis, with ana bsolute leukopenia.

DIFFERENTIATION FROM GERMAN MEASLES AND SCARLATINA.

GERMAN MEASLES	SCARLATINA	MEASLES
Incubation: 14 to 21 days.	2 (1 to 7) days.	9 to 10 days.
Prodromes: absent, short, or light.	Brief, a few hours to 1 or 1½ days.	3 to 4 days, generally markedly catarrhal.
Invasion: very mild.	Sudden, stormy, vomiting, convulsions, angina, and adenitis.	Catarrhal symptoms.
Exanthem: the rash is the first symptom, usually appearing on the first or second day on the face; as small red dots, rosy and brighter than in measles; small, little elevated, and thick about the mouth. They do not fuse. Less crescentic than in measles.	Appears on first or second day, reaching a maximum on second to fourth day, first below clavicles, involving face later, dusky-red, diffuse, burning, leaving mouth and eyes free.	Occurs on fourth day, greatest on fifth or sixth day, on the face, forehead, and about the mouth; brick-red elevated pappules or crescentic islets of eruption, always with areas of healthy skin between.
Enanthem, etc.: may be diffuse redness of throat and conjunctivæ.	Angina proportionate to skin eruptions; dusky-red; tonsillar plugs; conjunctivæ, bronchi, intestines usually intact.	Koplik's spots; patchy redness of throat; photophobia; conjunctivitis, marked bronchitis, very often bronchopneumonia; diarrhœa in some cases.
Glands: usually enlarged, hard, tender, especially posterior cervical, also axillary and inguinal. Spleen somewhat enlarged.	Proportionate to angina, hence largely cervical. Enlarged spleen.	Mostly at angle of jaw; rarely systematic enlargement. No enlargement of spleen.
Constitutional symptoms: few; no depression; no leukocytosis; little pulse increase and fever.	Much depression with rash, coated tongue with peeling on the fourth day (Strawberry tongue); fever 105° to 106°; pulse increased more than fever; lysis on seventh day; leukocytosis.	Depression, coated tongue, anorexia; fever-curve with two elevations; crisis on seventh day; no leukocytosis.
Complications: practically none.	Nephritis, arthritis, endocarditis, sepsis, periadenitis.	Catarrhal pneumonia, pleurisy, tuberculosis.
Convalescence: rapid; desquamation rarely copious, and always fine.	Prolonged, tardy; desquamation copious and usually in shreds (hands and feet).	Often prolonged; desquamation rarely copious and always fine.

Diagnosis.—The diagnosis depends upon the existence of an epidemic, and upon consideration of all the symptoms rather than of the eruption alone. Differentiation from *typhus*, where there is also catarrh, is made by the greater general disturbance in typhus, its more sudden onset, higher fever, greater swelling of the spleen, and by the fact that the roseolous eruption becomes petechial and is least marked on the face.

Sepsis with a morbilliform exanthem, the early stage of *influenza*, *syphilis* (in which other secondary symptoms appear with the eruption), and *variola* (*q. v.*), may sometimes delay the early diagnosis. In *drug-eruptions* (those of turpentine, iodides, copaiba, and antipyrin), the diagnosis may be most difficult. The fever, Koplik's spots, catarrh, and central elevations in the eruption, are indications of measles.

Prognosis.—The mortality is very great among aboriginal races. In the Fiji Islands the death-rate was very high. In the Samoan Islands 4,000 deaths followed the introduction of measles, in 1893. The usual important factors are: (*a*) The *genius epidemicus:* Henoch records 30 per cent. and Heubner 37 per cent. in some epidemics. The mortality in a German epidemic of 2,881 cases was only 1 per cent. (Heissler). (*b*) *Individual resistance:* in private practice 3 per cent. is the average death-rate. It is higher in institutional practice and in the army. It is 16 per cent. in the children's hospitals in Paris. Measles ranks third among the exanthemata in mortality. (*c*) *Age:* The death-rate is highest at the second year; 55 per cent. of fatal cases occur in the first two years of life (Henoch). (*d*) *Complications* are more important than the disease itself; *e. g.*, tuberculosis and bronchopneumonia, which is the worst complication, with a mortality of 33 per cent.

Treatment.—1. **Prophylaxis.**—Prevention is important, especially in children under two years, in severe epidemics with pneumonia, in the weakly, and in cases of pertussis. Though the mortality and complications are greater in adults, it is of no advantage to contract measles, and the too prevalent practice of allowing the disease to run through the family must be condemned. In all cases, the secretions from the nose, throat, and bronchi, must be disinfected, and general sanitary care should be exercised as in smallpox and scarlatina.

2. **Hygiene.**—The patient should have fresh air, a warm room, rest in bed until after desquamation, and a liquid diet. A mustard bath may be employed in order to bring out the rash.

3. **Catarrhal Symptoms.**—Photophobia does not necessitate a dark room. Some sunlight should be admitted. A boric-acid solution should be dropped frequently into the eyes and vaseline applied to the lids. Prophylactic care of the mouth is necessary. To relieve pain in the ears, belladonna, opium, and a 1-per-cent. cocaine solution may be used; paracentesis is indicated for gathering. The nose should be douched with Dobell's solution. Tr. belladonnæ, ♏ i.–iii. may be given every two hours for coryza until flushing of the face, increase of pulse-rate, or a dry, red throat, develops.

4. Antipyresis.—Antipyretics are indicated (*a*) in sudden high elevation with severe cerebral toxæmia; and (*b*) in high fever with diffuse bronchitis or pneumonia. They are given most efficaciously in the form of baths, with cold affusions to the head and neck. Wine, leeches to the mastoids, mustard plasters, and laxatives, may also be indicated in toxæmia.

5. The Circulation.—The heart rarely requires treatment, except in septic types or in pulmonary complications, when camphor, ammonia (aromatic spirits), strychnia, wine, or coffee, are indicated.

6. Respiratory Symptoms.—Care of the mouth, a soft or liquid diet, inhalations of steam, hot fomentations over the larynx, warm baths, emetics (such as apomorphine or ipecac), and possibly intubation or tracheotomy, are indicated for laryngeal complications. Bronchitis should be treated with ipecac, codeine (morphine), emetics, inhalations of steam, massive hot packs to the chest, and cold affusions over the nape of the neck (see ACUTE BRONCHITIS); stimulation for pneumonia (*q.v.*) may be indicated.

7. Alimentary Tract.—Enemata relieve constipation. When diarrhœa is excessive, opium, hot wine, or cognac, demulcents, hot applications, and physiological salt-solution by rectum, are indicated.

8. The Skin.—The skin should be rubbed with olive oil; a 2-per-cent. menthol salve, or 1-per-cent. carbolic solution, relieves itching.

9. Complications.—The treatment of complications, such as tuberculosis, pertussis, etc., will be described under these topics.

RUBELLA.

Synonyms.—*Rubeola notha, German measles, Rötheln.*

History.—Rubella was first described by Bergen (1752) in Germany, and its separate existence was established by Maton (1815) in England, though it is still called by some writers a hybrid form of measles and scarlatina. It is more often confused with atypical scarlatina than with atypical measles. By the Germans it is called rubeola, though we employ rubeola as synonymous with measles.

Definition.—An independent infection, which occurs often in those who have had measles and scarlatina. One attack confers immunity.

Etiology.—Its etiology is not known. Most cases occur during the first half of the year. The disease most frequently attacks children.

Symptoms.—I. **The Incubation.**—The incubation lasts from ten to twelve (or perhaps fourteen to twenty-one) days.

II. **Prodromes.**—Prodromes may be lacking, or for a period of from two hours to a day, there may be cough, slight photophobia, or sneezing. Slight temperature and a few general symptoms, such as chilliness or headache, usually develop.

III. **The Eruption.**—The eruption occurs on the first day, usually in the palate and throat. It resembles measles, and consists of hyperæmic punctate red spots (Forscheimer's spots). The skin is red, and the eruption disappears at first under pressure; then the macules become darker and more circumscribed; later, the eruption does not entirely disappear under pressure. There are two forms, the circumscribed and the diffuse. The eruption becomes slightly elevated, does not fuse as it does in measles, and erythema is not present about the points of eruption as it is in scarlatina. Anatomically, it is a capillary hyperæmia of the papillary and upper layers of the corium, accompanied by slight inflammation and exudation between the corium and epidermis. It advances in "jumps," and fades in one place while it blossoms in another. It appears first on the scalp and face, and progresses downward to the neck, body, arms, and lastly to the legs and feet. Unlike scarlatina, spots may be observed about the lips. The eruption is greatest on the upper parts of the body, and is increased by constriction or by pressure of the clothing. Its average duration is from two to four days. The papillæ of the tongue show slightly through the fur. Desquamation is minimal and fine. Tracheitis, or, rarely, bronchitis, may be observed. Almost characteristic is the swelling of the posterior cervical and post-auricular glands. There may be some angina, dysphagia, or anorexia.

IV. **The Constitutional Symptoms.**—The constitutional symptoms are few. The fever is usually slight. Its duration averages three days, and it falls by crisis or lysis when the eruption has matured. Except in a few American epidemics, urgent nervous and other complications, such as icterus, albuminuria, nephritis, or colitis, have rarely been observed.

Diagnosis.—The diagnosis is often very difficult and the unity of the disease is sometimes questioned (see MEASLES). The "*fourth disease*," described by Dukes, is said to present a longer incubation and more diffuse rash. Erythema infectiosum, emphasized particularly by Escherich, is a weakly, contagious, maculo-papular erythema, prevalent particularly in the spring and fall, and occurring chiefly on the extensor surface of the extremities, and rarely on the trunk.

Prognosis.—The prognosis is good, and therapy is symptomatic. Isolation is theoretically advisable.

ERYSIPELAS.

Synonym.—*St. Anthony's Fire.*

Definition.—An acute, febrile, contagious disease caused by Fehleissen's *Streptococcus erysipelatos*. It is characterized by a peculiar circumscribed inflammation of the skin and adjacent mucosæ, which tends to rapid and broad extension. It usually runs a self-limited course, with slight, if any, visceral involvement.

History.—Erysipelas was known to Hippocrates and Galen. Trousseau (1848), in France, maintained that it was always a wound infection. Werner, in England, maintained that it was an infectious disease, and Koch (1880) found the streptococcus, which was more fully described by Fehleissen (1881) and which bears his name.

Etiology.—It is wide-spread, and sporadic cases are seen in almost every centre of population. It occasionally breaks out epidemically. It prevails largely in the early spring months; 60 per cent. of cases occur between January and May (Anders). In the preantiseptic era it was the dread of hospital physicians, and even at this day it occasionally visits the most sanitary institutions. It is conveyed by a third person and by fomites; and, infrequently, by unclean instruments, and vaccine. Cases of placental transmission to the fœtus are known. The virus is not particularly penetrating but may adhere to clothing and furniture with great tenacity. Individual predisposition varies, but many persons have, apparently, a natural, or perhaps a hereditary, susceptibility to the disease. It is like rheumatism or acute angina, in that it often recurs repeatedly. It occurs mostly in the female sex (73 per cent. of the cases). Alcoholism, debility, and chronic nephritis, are predisposing factors. Any distinction between "idiopathic" and "wound" erysipelas is artificial, for in practically every instance, some abrasion, though perhaps too small for the eye to detect, occurs in the skin or mucosæ.

Bacteriology.—The Streptococcus erysipelatos (*seu* pyogenes longus) was found in the lymph-vessels by Fehleissen, who first drew the distinction between erysipelas and phlegmon. Though it strictly coincides with Koch's criteria, its absolute differentiation from the Streptococcus pyogenes is impracticable. Inoculations from cases of erysipelas sometimes produce suppuration. Pus from abscesses has caused erysipelas, and, in certain cases, erysipelas and suppuration have developed simultaneously. This has given some weight to the theory of the bacteriological unity of erysipelas, suppuration, and septicæmia.

Symptoms.—I. **Incubation.**—Incubation lasts from three to seven days (fifteen to seventy-two hours in direct inoculations).

II. **The Eruptive Stage.**—(*a*) This stage usually begins with vomiting, fever, chill, and the eruption, and is sometimes preceded by general symptoms, such as fatigue, anorexia, or pains in the joints. The fever rises rapidly to 103°. The chill, occurring in 75 per cent. of the cases, is usually preceded by a few hours of general discomfort. It is absent chiefly in erysipelas which is secondary to other diseases. (*b*) The eruption, in typical cases, first appears on the face (95 per cent. of the cases), near the inner canthus of the eye, near the nose, or on the cheek. It consists of a hard, bright-red, slightly elevated, burning, and painful area of inflammation, which pits but little on pressure, though its color may disappear. Pathologically, erysipelas is simple inflammation; in and under the skin are found œdema, hyperæmia, white- and red-cell extravasation, and streptococci which advance in the lymph-clefts, and are most abundant in the spreading edge, but are also found in the lymph-vessels beyond it. Its elevated red edge is sharply marked, or "wall-like." As the area spreads, the skin becomes much swollen, œdematous, smooth, tense, red, and hot, causing a shapeless swelling of the eyes, nose, lips, and ears, and obliterating the facial lines and expression. The extent of this area is indicated by tenderness, and is therefore more accurately measured with the finger than with the eye. Under the lens, small vesicles are always found. The prong-like advance of the eruption is similar to that of spilt alcohol, or, as Volkmann puts it, like burning paper; *i. e.* it disappears behind as it advances forward. Its extension is irregular, because the streptococci follow the line of least resistance. If obstructed by dense tissue, deep attachments, or the so-called ligamenta cutis, its course becomes circuitous, asymmetrical, and tardy in one area though rapid in another. It is limited at the base of the skull, and rarely affects the chin, or passes over it to the opposite side. The neck resists its advance, often successfully, but erysipelas may course over the entire body. The sternum, condyles, ilium, and Poupart's ligament, check or divert its progress. When apparently obstructed, new islets of eruption appear near by, which are nearly always consecutive, as may be seen by the faint lymphatic involvement between the foci. The scalp is often unaffected, but if invaded it becomes swollen, tense, and pale or blue rather than red, and the hair falls out. The genitalia may swell enormously. Necrosis of these tissues, and also of the elbow and knee, may result from excessive tension. The first area pales, and a coarse desquamation results, while the eruption at more remote points advances. The lymph-glands are almost constantly swollen, tender, and are palpable, unless hidden by œdema. They become enlarged proportionately to the dermatitis. The eruption presents few variations: *erysipelas miliare* (*vesiculosum*); less frequently *erysipelas bullosum; erysipelas squamosum*, or, in old and cachectic subjects, *erysipelas hemorrhagicum.* (*c*) The *fever* rises suddenly, and is highest on the third or fourth day (103° to 104° or higher). It is continuous, then becomes remittent or irregular, and finally, after seven to nine days, resolves by crisis, or less often by lysis. Fever is almost always present; in very rare cases is it wholly absent. In attenuated erysipelas there is little fever and there are few general symptoms, though the rash may be well developed. In general the intensity and the progress of the infection are marked by the degree of fever.

Other General Symptoms.—The pulse is soft, and ranges between 100 and 120; albuminuria occurs in 38 to 67 per cent. of the cases; moderate leukocytosis is the rule, and there may be some pain and swelling of the liver and spleen. Nervous toxæmia, with the exception of headache, is uncommon.

Course.—When limited to the face and head, eight to ten days is the usual duration, and a course longer than two weeks is uncommon. *Erysipelas migrans* may last weeks or months, travel from limb to limb, and recur again and again. In one of the author's cases it lasted six months.

Complications and Sequels.—1. **Nervous.**—Toxæmic symptoms are infrequent except in aged, debilitated, or alcoholic subjects.

Delirium is toxæmic, but was considered reflex, resulting from trigeminous involvement, by Traube and Jaccoud. It is an unfavorable development and occurs especially when the scalp is invaded. A profound typhoid condition may occur in debilitated, or alcoholic cases. Meningitis, formerly confused with toxæmia, infrequently follows orbital cellulitis or extension along the fifth nerve. Post-febrile psychoses, multiple neuritis, and degeneration of the cord, are exceptional. Eye inflammation, ulceration, and blindness, are very infrequent.

2. **Circulatory.**—Endocarditis is infrequent though simple and ulcerative types may develop. Pericarditis, myocarditis, acute dilatation, and arterial or venous thrombosis, are infrequent. Leukocytosis is usually moderate, and is generally parallel with the fever. High counts usually indicate concomitant suppuration. Streptococci in the blood are usually, but not necessarily, ominous.

3. **Respiratory.**—Erysipelas may invade the nose, throat, ear, larynx, and bronchi, and begins with local symptoms or with chill and fever. Erysipelatous coryza, otitis, and stomatitis, are infrequent and are difficult of recognition. Erysipelatous angina is characterized by much pain, sharply marked redness or lividity, a glazed varnished surface, marked œdema of the uvula and epiglottis, and the formation of vesicles or bullæ. Sometimes a wall-like margin is seen. Gangrene may develop in severe instances. The neck glands may be swollen. For the recognition of erysipelatous angina, coincident facial erysipelas is necessary.

Laryngeal erysipelas is more often secondary (to pharyngeal erysipelas) than it is primary. It is rare, severe, is easily or necessarily confused with glottis œdema, and is marked by great turgescence, rapid spreading and swelling, and high, early fever. Without tracheotomy, fatal stenosis and extension to the lower air-passages may occur. The rare so-called "erysipela spneumonia" is especially described by French writers. Though the existence of this type cannot be denied, because any infection may travel downward from the throat, it is more probably a septic localization than "internal" erysipelas. In streptococcus pneumonia, the spleen is enlarged (sepsis) more frequently than it is in ordinary pneumonia. The advance is rapid, the extension is wide, the gray hepatization is great, and fibrin formation is scant or lacking. Nevertheless, in pneumococcus infection the same "wandering pneumonia" may be seen. Pleurisy is usually metastatic (septic).

4. **Alimentary.**—Vomiting is often cerebral. Exceptional complications are parotitis, œsophagitis, gastritis, enteritis, intestinal ulceration, hemorrhage, pain, icterus, and pylethrombosis.

5. **Genito-Urinary.**—Albuminuria (67 per cent. of the cases) is the most constant visceral finding. Nephritis (4 per cent.) is often of the acute glomerular type, and rarely becomes chronic. Streptococci have been seen in the urine and kidneys, especially in septic types. Typical erysipelas may begin in the external genitalia, especially in puerperæ, or may first infect the internal mucosa which has been abraded dur-

ing labor. There may be great pain, local necrosis, and imminent risk of higher or peritoneal invasion. Accurate diagnosis of the internal form of erysipelas (*E. puerperale grave internum*, Virchow) can seldom be made.

6. **Septic**—Subcutaneous abscesses, which are usually small, and which resemble cold abscesses (Tillmann, Alcalme), appear on the head or body, occur in 6 per cent. of the cases, especially in alcoholic, nephritic, diabetic, or debilitated patients. Phlegmons may develop, usually on the legs, and are known to occur even without mixed infection. Gangrene is fortunately rare (½ of 1 per cent.). It may develop early, in the eye-lids, in the genitalia, over the lean tissue covering the joints, or it may develop later in the extremities. Herpes is not infrequent. Baldness may also result. The joints may be painful or swollen. Suppuration is metastatic. Suppurative adenitis may occur with or without mixed infection. General fatal sepsis, aside from the puerperal form, is infrequent. It bears the same relation to local erysipelas as does miliary tuberculosis to a local tuberculous focus (Ponfick).

Diagnosis.—Diagnosis is easy, when the area is the size of a dollar, (*a*) because of its very location; (*b*) because of the early involvement of the lymphatics and of the lachrymal sac; (*c*) because in most cases the point of entrance can be found; it may have healed; it may be concealed by the swelling, or lie an inch or more away from the first eruption; (*d*) because of the redness, smoothness, swelling, and œdema; (*e*) because of the elevated, wall-like margin; and (*f*) because of the prong-like, irregular progression.

In *weeping eczema*, the edges are not raised, the skin is not smooth, there is less fever, and the development is slower than in the ordinary type, and itching is present. *Malignant pustule* may cause redness and swelling, but anthrax bacilli are found under the primary papule and crust. *Dermatitis venemata*, or *medicamentosa*, and pemphigus, are unlike erysipelas. *Erythema nodosum* is discrete, though diffuse, and does not spread. Diffuse simple erythema is a febrile affection, and of short duration. *Dermatitis exfoliativa* (Ritter) in the new-born is afebrile, develops first about the mouth and chin, and reaches the body and limbs later. Its diffuseness and redness are characteristic. In *lymphangitis* the swelling of the lymphatics advances toward the centre and presents cord-like or net-like areas, which are neither confluent, vesiculated, uniformly red, nor evenly edged. In *phlegmon* and deep *cellulitis*, the process is deeper and harder, and the blisters and margin of erysipelas are lacking.

Erysipelatous angina may not be diagnosed without coincident facial erysipelas. The local findings, such as great swelling, dysphagia and lymphatic enlargement, are not pathognomonic; neither are the general findings, such as high fever, albuminuria, nephritis and splenic tumor.

Erysipeloid, a zoönotic affection described first by Rosenbach, occurs especially in persons whose occupation leads them to handle meat. It is recognized by the "butterfly" eruption which develops on both cheeks or on the finger-ends, and is unaccompanied by fever or general symptoms. The edge of the eruption is clear-cut, its advancement slow, its color a dark livid-red, and its course lasts one to three weeks with spontaneous recovery. Gilchrist (1904) reported 329 cases, of which 323 were caused by crab-bites.

Prognosis.—(*a*) In uncomplicated facial erysipelas the outlook is good. In general the mortality is 3 to 4 per cent. in private practice, and double that figure in hospitals. (*b*) In *erysipelas migrans* the outlook is less favorable. (*c*) In *erysipelas neonatorum* (Trousseau, Lorain) the outlook is almost invariably fatal on the fifth to seventh day. It begins on the navel, and less often on the vulva, and on circumcision-wounds. Vomiting, diarrhœa from toxæmia, peritonitis, icterus, pneumonia, necrosis, gangrene, suppuration, and sepsis, are much more common than in adult forms. Fortunately, like puerperal fever, this form is now infrequent. (*d*) Erysipelas secondary to other infections is serious, especially in chronic maladies, such as alcoholism, diabetes, nephritis, and tuberculosis, and also in bed-ridden, or senile subjects, in whom the asthenic, hemorrhagic, or gangrenous, types are chiefly observed. In acute diseases such as typhoid, diphtheria, and pneumonia, this complication is dreaded. (*e*) Puerperal forms or involvement of the mucosa, especially that of the larynx, are unfavorable (Hippocrates). (*f*) Recurrence is more frequent than in any other infection, which shows that no permanent immunity is conferred. Relapses (10 to 15 per cent. of the cases) are attacks recurring within a few days. Recurrences (20 to 50 per cent.), properly speaking, return after months or years; indeed, in some individuals, erysipelas may be called habitual. It is observed most frequently in the young and in women (75 per cent.), and mostly on the face. It may result in elephantiasis. Five to fifteen attacks are not uncommon, and Roger has seen forty in one person.

Treatment.—**1. Prophylaxis.**—In this bacteriological era, the disease has been decreased by means of asepsis and isolation, though in regard to the latter, many physicians are still remiss. Chronic rhinitis, rhagades, decayed teeth, and neglected ulcers, must be considered in cases of habitual erysipelas.

2. Mechanical Means.—Mechanical efforts to limit the extension of the disease are still used, but unsuccessfully. These consist of binding with elastic strips, elastic constriction, or heavy applications of collodion.

3. Local Applications.—Iodine, creolin, bichloride of mercury, carbolic acid, alcohol, iodoform, boric acid, and numerous other topical applications, may be recommended, but the water they contain is the best ingredient, and was recommended by Hippocrates himself. Mechanical and local applications cannot reach the deeply-situated cocci. Cold compresses afford a sure relief for pain and tension. Ichthyol ointment, 10 to 20 per cent., is soothing but in no sense curative.

℞

Creolin	1 part.
Iodoform	4 parts.
Lanolin	10 parts.

M. f. ung. (Koch.)

4. **Drugs.**—Erysipelas is a self-limited disease, therapeutically unapproachable. Tincture of the chloride of iron (Hamilton Bell, 1861) is well tolerated in large doses (♏-x-xxx) but neither shortens nor mitigates the disease.

℞

Tincturæ ferri perchloridi	ʒiiss.
Quininæ sulphatis	gr. xxiv.
Spiritus chloroformi	ʒij.
Aquæ	q. s. ad ℥vj.

M. f. mist.

S.—Two tablespoonfuls every three or four hours. (J. Burney Yeo.)

5. **General Treatment.**—The symptoms are managed expectantly,—*i.e.*, as they arise. Headache, delirium, pain, vomiting, and cardiac weakness, are treated as in typhoid. *Rest in bed* and as *generous nourishment* as the stomach will tolerate, are indicated.

6. **Marmorek's Serum.**—Chantemesse reduced the death-rate of his cases to 2½ per cent. by serum treatment, a figure often reached by more simple measures (Roger and Botognesi).

Erysipelas as a *curative agent* (*erysipelas salutare*): Permanent cure of lupus has followed a complicating erysipelas. Syphilis quickly subsides after this infection, and its beneficial effects have been observed in leukæmia, Hodgkin's disease, carcinoma, sarcoma, chronic arthritis, rheumatism, iritis, tuberculous adenitis, neuralgia, diphtheria, and tuberculosis.

MALARIA.

Definition.—An acute infection caused by the Plasmodium malariæ, inoculated in man by the mosquito and characterized clinically by (1) paroxysms of intermittent, quartan, tertian, or quotidian fever, or by (2) remittent or subcontinuous fever, and sometimes by (3) pernicious, or (4) chronic cachectic forms.

History.—Malaria, known to Hippocrates and Celsus, is the disease most completely investigated, in that (*a*) its cause is exactly known; (*b*) its prophylaxis has become known since the discovery of infection by the mosquito; (*c*) the disease can be watched in different stages in the blood; and (*d*) there is a specific treatment.

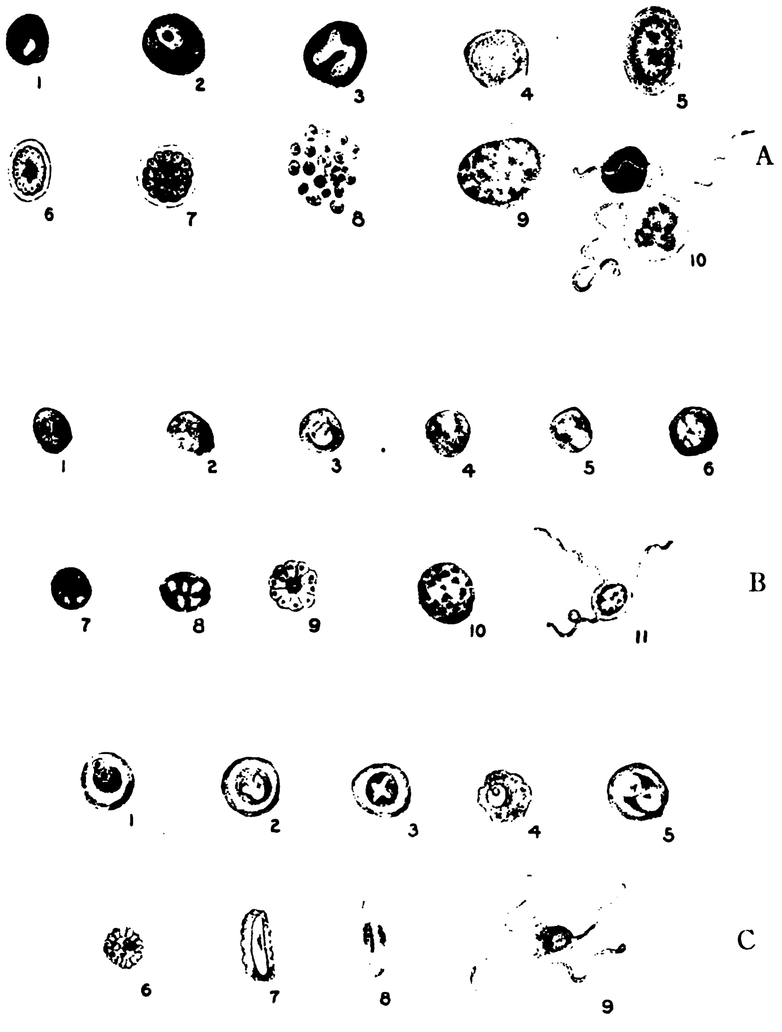
Etiology.—All ages and both sexes are affected equally. Men are more exposed to infection, and children are readily infected on account of their thin skin. Such occupations as sewer and railroad building, ditching, and night work, especially, are predisposing factors; 32 per cent. of the French soldiers in Madagascar in 1895 acquired malaria; reduced physical resistance, exposure, excesses, and alcoholism, also predispose to the disease. Negroes have a relative immunity Instances of familial or individual immunity have been noted. Epidemics may occur, as happened during the building of the Pari boulevards. Indirect causes, such as warmth of climate, the warm season, moisture, rain, swampy localities, telluric influences, bad ai (mal-aria), and impure drinking-water, are now proved to be cause only in so far as they relate to the breeding of the mosquito. In Africa severe forms of malaria are found everywhere except in the lower par and in the Sahara. In the far East its chief seats are India, Burmah Assam, Faral, and the East Asiatic coast; Asia Minor, Japan, and th East Indies. In Europe, lower Russia, Italy, Hungary, the Balkans and Greece, are most affected. Malaria also abounds in South America in the northern three-fourths of the continent. In the West Indies severe types prevail. In the United States, malaria is slowly movin southward, and cases seldom develop on the Pacific coast, in th Northwest, in the dry West and, in the Lake region. Even in th South it is becoming less frequent.

Direct Cause.—The direct cause is the malarial parasite, describe by Laveran, in 1880, and also by Marchiafava and Celli, in 1885. I this country Councilman, Abbott, Osler, James, Dock, Koplik Thayer, Hewetson, and others, have studied the disease. It is a red cell parasite, a hæmocytozoön. The biology of the Plasmodium malariæ concerns (*A*) its life in the human blood, and (*B*) its life in th mosquito. The unity of all species was maintained by Laveran, bu various species must be recognized because (1) their morpholog varies; (2) their geography, seasonal prevalence, and malignanc also vary; (3) on inoculation, the species and fever type are maintained; (4) the type remains the same in the same individual.

(A). **The Plasmodium in Man.**—Special forms of the parasite:

1. The *quartan parasite* has a life-cycle of three days. In the fir twelve hours the young form is without pigment. A small amorphous hyaline bright spot lies in the red cell, whose form flattens th parasite. There is some amœboid movement upon warming the slid It gains little in size for one day, when some pigment or melanin appears in the form of coarse dark rods and granules with little movement. Melanin is the hæmoglobin digested by the parasite. It i usually dark or black and resembles the melanin of malignant growth It is dissolved by alkalies and ammonium sulphide, but not by acid Movement of the parasite ceases as pigment increases. The parasite occupies one-half to two-thirds of the red cell after forty-eigh hours, and almost the entire cell after sixty hours. There is rarel more than one parasite in each cell. Flagella (*v.i.*) are not frequen During growth the red cell on which the plasmodium feeds eith undergoes no change in size or becomes smaller and darker peripherally. As sporulation occurs, the pigment gathers centrally an the spores, usually not numbering more than ten, develop into th "daisy" form. Vacuoles are seen in some cases. After sixty-ni hours, mature segments finally separate, each with a bright spot

PLATE III.



Malarial Parasites.

A. Tertian parasite. 1, young form; 2-4, stages of growth and pigmentation;
3-7, segmentation; 8, rupture of spores from corpuscle; 9, extracellular form;
0. flagellate form.

B. Quartan parasite. 1-6, development and pigmentation; 7, 8, central group-
ng of pigment and spore formation; 9, rosette of spores with nuclei; 10, extra-
ellular form; 11, flagellate form.

C. Æstivo-autumnal parasite. 1, ring-like body; 2, 3, amœboid forms; 4,
rasite in brassy degenerated red cell; 5, pigmentation; 6, segmentation; 7, 8,
lf-moon bodies; 9, flagellate forms.

nucleolus; *i. e.*, segmentation occurs in the peripheral blood three hours before the paroxysm, which corresponds with their rupture from the red corpuscle into the plasma. Sporulation means death of the adult parasite, or its "death in childbed," as Mannaberg well describes it. The spores rupture through the mother cell and the red cell, and escape into the plasma, whence they attack and enter other red cells, to repeat their life-cycle. The pigment and fragments of red cells are picked up by the leukocytes and are carried to the liver and spleen. Some forms (gametocytes) do not mature nor sporulate. They are taken up by the mosquito, to complete in it another cycle. (Plate III.)

2. The *tertian organism* has a forty-eight-hour cycle. In its first day it is small (1μ to 2μ), and appears as a bright, unpigmented, disk-like or annular spot in the red cell, thrusting out pseudopodia, with most active amœboid movement, and possessing a relatively large nucleus,--partly clear chromatin, and partly chromatin which may be deeply stained by the Romanowsky method. It fills about one-third of the cell. In its second day, pigment rapidly accumulates in the form of fine granules or rods, which are usually peripherally located, and which have an actively swarming movement, compared by Laveran to that of boiling water. This is due to currents in the protoplasm of the parasite. The amœboid movement of the parasite decreases in twenty-four to thirty-six hours, as it grows and as the pigment increases, but some movement remains for a long time. Vacuoles are frequent. The parasite occupies two-thirds to three-fourths of the red cell, which becomes distended and chlorotic. The most common form of the sporulation is the concentric accumulation of pigment and the development of fifteen to twenty rough, refractile spores, which form in two concentric circles ("sunflower" arrangement), or are more frequently irregular, like a bunch of grapes. The spores are small and round. Sporulation occurs three hours before the chill and the rise of temperature, and is completed in forty-eight hours, when the toxins secreted produce the rigor and fever. Most spores are seen at the time of the chills, and even then few can be seen, because sporulation occurs to a greater extent in the internal organs than in the peripheral blood-stream. When spores are seen between paroxysms they represent the sporulation of another generation of parasites, too weak or too few to induce a paroxysm. The generations are never absolute as they are in the quartan type, and the parasites of the same generation vary six or eight hours in their cycle. This explains the clinical variations in periodicity. Flagellated forms may be seen a few minutes after withdrawal of the blood. The nodulated and clubbed flagella lash about and indent the red cells. They may break off and swim about. They occur in numerous parasites which never sporulate in human blood, and remain as large bodies with vibrating pigment. They were formerly regarded as degenerated parasites, but really represent gametocytes or the "sexual forms," which develop later in the mosquito.

3. The *æstivo-autumnal parasite* has a life-cycle of from twenty-four to forty-eight hours. It has a very small, unpigmented body, and possesses most active amœboid movement. It thus attracts early attention but is easily overlooked, as it possesses almost the same refraction as the red cell. It becomes quiet after drawing the blood from the body, when it appears characteristically ring-like, often with a central vacuole. The shape may be successively amœboid, diskoid, annular, cruciform, or often may have a slight protuberance at one side—the seal-ring form. Mature forms contain particles of hæmoglobin. The pigment is extremely fine, powder-like, dark, sometimes reddish, peripherally located, and has some movement. When the parasite reaches one-third the size of the corpuscle, it is usually motionless, and the pigment becomes concentrated centrally or sometimes peripherally. Motion in the parasite has been seen in the cadaver by Ziemann. The spores number six to fourteen (or sixteen), are very small, round, and without movement or vacuoles. Sporulation occurs as in the other forms. Fever, pigment concentration, and sporulation, coincide (Golgi). If large parasites with much pigment are seen in the peripheral blood, they denote the onset of a paroxysm, and after the paroxysm, ring-forms without pigment may be observed. The young forms circulate in the general blood-stream, though sporulation usually occurs in the deep viscera. The *half-moon bodies*, or crescents, are delicate, slender, highly refractile bodies, measuring eight to ten microns by two to three microns, are developed in the bone-marrow or spleen, and possess considerable pigment, which is usually mesially located and has little, if any, motion. They have no amœboid movement but may slowly change form. In shape they are spindle, oval, or spherical. They occur singly in the red cell. They do not multiply in the human blood. The young crescents develop in the viscera or bone, and their adult forms are seen in the peripheral blood, for the periphery offers the best opportunity for leaving the body—through the bites of mosquitos. The male form of the crescent has flagella (microgametes) which penetrate the sexual elements of the female crescents (macrogametes). This occurs outside of the body, either on the slide or in the body of the mosquito. After several days crescents, spherules, or spindles, appear. Crescents indicate that the fever has just begun. Fever rarely occurs when crescents alone are found in the blood. If fever develops with the presence of crescents, amœboid bodies, also, are nearly always found. The red blood-cell becomes copper-colored or brassy.

This knowledge of the parasite explains experimental inoculation from individual to individual as well as accidental infection during operations. Transmission to the fœtus is unknown. The parasite may be stained with methylene blue or hæmatoxylin, and may be counterstained with eosin. For the beginner, at least, study of the fresh blood under an oil-immersion is the best method.

The Nocht-Romanowsky staining fluid, according to Ewing, is made as follows: (1) Polychrome methylene blue solution: To 1 ounce of polychrome methylene blue (Grübler) add 5 drops of 3-per-cent. solution of acetic acid (U. S. P. 33 per cent), to neutralize the undue alkalinity. (2) Ordinary methylene blue solution: Make a 1-per-cent. (saturated) watery solution of methylene blue, preferably Ehrlich's rectified, or Koch's, by aid of gentle heat. This solution improves with age and should be at least a week old. (3) Eosin solution: A 1-per-cent. solution in water of Grübler's aqueous eosin. Then to 10 cc. of water add 4 drops of (3), 6 drops of (1) and 2 drops of (2), mixing well. To use, the specimens, fixed by alcohol or heat, should be immersed for two hours, specimen-side downward; they will not overstain by twenty-four hours' immersion. They should then be washed in distilled water, dried slowly over the flame, and mounted in Canada balsam. The density of the blue may be varied to suit individual fancy, nor need the proportions be rigidly followed, but the accurate neutralization of the polychrome solution is most important.

MANNABERG'S TABLE: CHARACTERISTICS OF THE VARIETIES OF THE PARASITE.

(NOTE.—Forms 3, 4, and 5, are usually grouped as one, æstivo-autumnal.)

	Life-cycle.	Motility.	Pigment.	Maximum size.	Form of sporulation.	Number of spores.	Half-moon bodies.	Changes in the infected red corpuscle.
1. Quartan parasite.	72 hours.	Little movement in the young forms.	Coarse, with little movement.	Size of red blood corpuscle.	Daisy form; single spores elongated and nucleated.	6 to 12.	None.	Red cells little changed in size or color.
2. Ordinary tertian parasite.	48 hours (or less)	Active movement in the youngest and middle-aged forms.	Fine granules; very active, "swarming" movement.	Size of red cell, or sometimes larger.	Sunflower or grape form; single spores, small, round, and seldom visible when unstained.	15 to 20 (or less).	None.	Often hypertrophic. Chlorotic.
3. Pigmented quotidian.	24 hours (or less)	In the young unpigmented forms very active amœboid movement, growing less.	Very fine, no swarming movement, forming in clumps.	$\frac{1}{3}$ to $\frac{1}{2}$ of red cell.	Irregular particles.	6 to 8 (or more).	Present	Brassy, shrunken, or sometimes wholly decolorized.
4. Unpigmented quotidian.	24 hours.	Very active amœboid movement.	None.	$\frac{1}{6}$ to $\frac{1}{4}$ of red cell.	Stellate or irregular.	6 to 8.	Present	Shrunken and brassy.
5. Malignant tertian.	48 hours.	Active, persisting also in pigmented bodies.	Moderately fine, often shows oscillating movement.	$\frac{1}{2}$ to $\frac{2}{3}$ of red cell.	Irregular forms.	10 to 12 (rarer 15 to 16).	Present	Shrunken and brassy.

(*B*). **The Life of the Organism Without the Human Body.**—King (1883) first suggested that the mosquito was the intermediate host and sole source of infection. It takes the plasmodium from man and reinoculates him with it after the life-cycle within its body. Other analogous infections are known, such as the hæmosporidia of birds, frogs, bats, dogs, sheep, monkeys; *e. g.*, Texas cattle-fever inoculated by the tick, and the African tsetse disease (*trypanosoma*) in animals and man, which is inoculated by the tsetse-fly. Manson, especially, then Ross, Grassi, Bastianelli, Bignami, Daniels, Stephens, Christopher, and others, have developed this most interesting subject. The Plasmodium malariæ has not been found except in man and in the mosquito. The mosquito is the primary and higher host, since the sexual process of reproduction in its stomach is biologically higher than the simple sporulation in man. The culex and the anopheles are the two chief genera. The culex does not contain nor convey the parasite. Its palpi are very short and its wings generally have no mottling. When sitting, its hind legs are carried above the body and its body lies parallel with the wall, or may sag, from the blood it has sucked. Its eggs are laid in tanks or puddles. It is the ordinary house mosquito and is found chiefly in cities. The anopheles conveys malaria. Its palpi are almost as long as the proboscis; the wings are mottled; the rear legs usually depend, or touch the wall, but sometimes are carried over the body. Its body makes an angle of 45° with the wall. The eggs are laid in pools or sluggish streams with algæ growth, and the open country is its preferred habitat. In America, four species exist: (1) The most important is the Anopheles maculopennis (the Anopheles claviger of Europe); (2) Anopheles crucians; (3) Anopheles punctipennis; (4) Anopheles argyritarsis. Wher the anopheles bites persons whose blood contains sexually mature forms of the plasmodium (gametocytes), flagellation in the male form occurs and the flagella enter the female cells. After fecundation in the insect's stomach, the impregnated cell enters the muscular wall of the middle intestine, and, after two days, small, round, refractile pigmented bodies appear, which, after a week, grow to 60 or 70μ in diameter. They develop into myriads of sporoblasts which reach the salivary glands of the insect, whence they are again inoculated into man by biting. These sporozoids thus resulting from sexua conjugation in the mosquito, are equivalent to spores formed by asexual sporulation in the human blood. Entering the red cells in man they follow the cycle of their type as above described, and produc either sporulating forms or sterile forms. The sterile forms in th human blood—*i. e.*, the forms which do not sporulate—linger in th peripheral blood with the teleologic purpose of preserving and continuing their life in the mosquito, and, on reaching its body, develop again the agents of reinfection. This knowledge concerning the mosquito explains many interesting and formerly incomprehensible details. We now know why those living in the first story of houses ar more likely to be infected than those living in the upper floors, an why those who slept in houses facing inland were infected while those in houses exposed to the sea air escaped, etc. In Rome, the fever prevailed just outside the Porta del Populo, while the Corso, three hundred feet distant in the city, escaped.

Symptomatology.—(*A*). **Fever.**—Fever is, next to the finding of the melanin and the plasmodium, the most constant symptom, though, strictly speaking, malaria should not be divided into types by the fever alone. It comes on after an incubation varying irregularly from one and a half to fifteen days. Fever bears a close relation to sporulation, as was demonstrated by Golgi, in 1885. The fever-curve is most variable, being very irregular in some types or almost mathematically regular in others. It is usually irregular at first even in regular types. It may be remittent (subcontinuous), intermittent (tertian, quartan, quotidian), or continuous for a few days, when it ends by crisis or becomes remittent or intermittent before recovery. The paroxysm may be delayed beyond the time when it is due (*postponens*), or may anticipate this time (*anteponens*). There may be hyperpyrexia, or normal or subnormal temperature. This occurs in the algid variety.

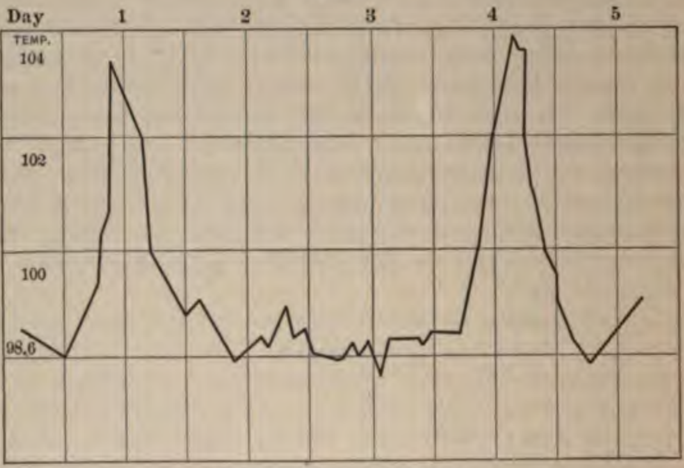
The classical divisions of the paroxysm are (1) chill, (2) fever, and (3) sweating, whose respective duration, according to Lorain, is in the proportion of 3–2–7. Any element of the paroxysm may be absent. Thus the fever or the rigor is absent in some virulent varieties and in persons partly immunized, and the sweating may be slight or absent. The paroxysm lasts ten (four to forty-eight) hours, and in 91 per cent. of the cases occurs between 10 A. M. and 3 P. M. Prodromal symptoms are fatigue, and anorexia. (1) The *chill* then occurs. It is rather sudden in its onset, often very severe, with headache, nausea, emesis, coldness, contraction and lividity of the skin (*cutis anserina*); the temperature by rectum is elevated, the pulse rapid, small, and hard; the respiration is rapid or even dyspnœic, and the urine is increased. The parasites are in the peripheral blood in the tertian and quartan types, but largely in the internal organs in the æstivo-autumnal type. (2) The *fever* is high, perhaps not higher by rectum than during the chill, but is more evidenced by the red, burning skin, thirst, delirium, and headache, soft, full, or dicrotic pulse, and other fever symptoms. In this stage the young parasites are free in the blood and begin to attack new red cells. After their entrance into these cells the fever ceases. (3) In the *sweating stage* the young parasites are found in the red cells, and phagocytes take up the detritus and pigment. The sweat is usually profuse and somewhat characteristic, giving off an odor like that of semen. The patient may be exhausted, but post-febrile euphoria is usual.

SPECIAL FEBRILE TYPES OF MALARIA.—The quartan and tertian forms are regular and intermittent, and the æstivo-autumnal forms are more irregular and remittent.

1. The *quartan fever* is the rarest of all types ($\frac{8}{10}$ of 1 per cent.), is found in the temperate zones, and occupies a position geographically

intermediate between those occupied by the tertian and æstivo-a tumnal varieties. It is caused by the quartan parasite, is the m regular of all types, and is called quartan because the simple fo causes paroxysms every fourth day. The parasitic cycles are read followed in the circulating blood, and the organisms do not accum late in the internal organs. Sometimes fission bodies may be fou without fever. The fever is rather obstinate and recurrence is f

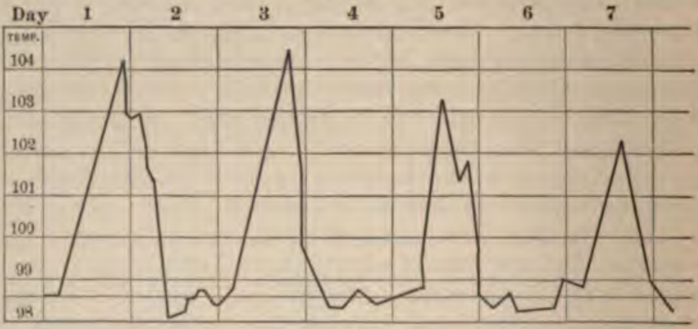
Fig. 10.



Quartan malaria (Silvestrini).

quent, but quinine exerts its specific action in a relatively short ti The type may be single, from one generation of parasites; doub from two generations; or triple, from three generations, thus prod ing, respectively, temperature every fourth, every first and second, finally, every day of the cycle.

Fig. 11.



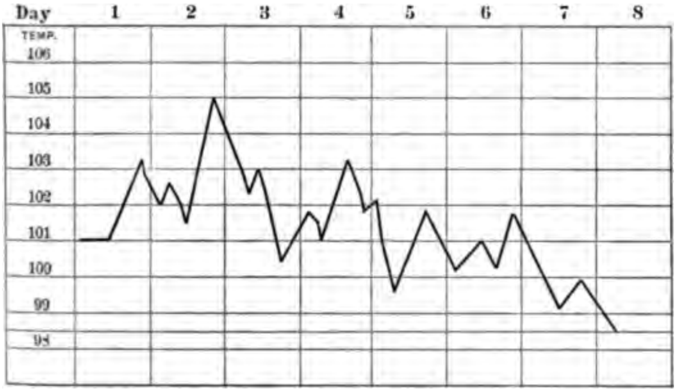
Tertian malaria.

2. *Tertian fever* is most frequent in the temperate zones and is most widely distributed variety. It is found in all malarial countr and constitutes 50 to 65 per cent. of all malaria. The organisms te

to accumulate in the internal organs. Fission bodies are rarely found without fever. It is rarely pernicious. The type may be single, or, more frequently, double from two generations of parasites. Fever which is almost continuous may sometimes be observed (Thayer and Hewetson). It responds very readily to quinine or, indeed, to simple rest in bed (spontaneous recovery).

3. *Æstivo-autumnal fever,* caused by the æstivo-autumnal parasite, is often irregular, remittent, intermittent, or continuous (subcontinuous). It is characterized clinically by severe and recurrent fever, marked anæmia, frequently by absence of paroxysms (chills occurring in only 71 per cent. of the cases), and sometimes by pernicious symptoms. It was first called æstivo-autumnal by Marchiafava and Bignami, in contradistinction to the vernal or milder spring forms. *Subtypes:* (*a*) Regular intermittent æstivo-autumnal fever. Its two varieties are: (i) The æstivo-autumnal tertian, in which the parasite is different from that of the ordinary tertian. The temperature is apt to be high, and the paroxysm doubly long. The typical curve may be modified by the absence of a distinct initial elevation, by an exaggerated pseudo-crisis, by a briefer or longer duration of the attack, by anticipation or procrastination, by double attacks, and by marked oscillations. (ii) The æstivo-autumnal quotidian, in which the attacks are usually short, but have an abrupt elevation of temperature. They often become irregular, frequently fuse, and have fewer symptoms than in the other variety. This form is often mild and the patient may recover spontaneously. It may, however, become pernicious. This form is readily confused with typhoid. (*b*) The irregular intermittent æstivo-autumnal fever, which has marked irregular intermittency or remittency, or subcontinuous fever, because the attacks are duplicated, anticipated, or prolonged. The paroxysm may last twenty hours. The life-cycle of the parasite is often difficult to follow. The symptoms may be mild or very severe.

FIG. 12.



Æstivo-autumnal malaria.

(*B*). **Blood-Findings.**—The blood-findings are pathognomonic; *viz.*, parasites, anæmia, absence of leukocytosis, and melanæmia are found.

(*a*) RED CELLS.—Anæmia is always present during the attack, from direct deglobulization by the parasite, and possibly also from liberated toxins. Acute anæmia is naturally more marked and rapid than in any other acute infection. The loss is greatest in the first attack, especially in pernicious cases, and sometimes reaches half a million cells. Reduction to 500,000 red cells (Kelsch), thus giving the picture of pernicious anæmia, has been observed especially in cases of pregnancy, and in children (Frayer and Ewart).

(*b*) WHITE CELLS.—In the first attack absolute and relative leukopenia is observed, which is due to the collection of the white cells in the liver and spleen, to the destruction of the phagocytes, and, in cachexia, to lesions of the blood-making organs. As in other anæmias caused by diseases of the bone-marrow and spleen, an increase of lymphocytes or large, mononuclear cells, with decrease of the polymorphonuclear cells, may be observed, especially in the apyretic intervals. Genuine leukocytosis in malaria denotes a complication such as pneumonia or sepsis. An absolute increase is sometimes observed in pernicious malaria, such as the "black-water fever," but even here the increase is in the lymphocytes. Cell division and degeneration in the leukocytes may be observed.

(*c*) MELANÆMIA.—Melanæmia (first observed by Virchow and Heschl, 1847–50) is, next to the presence of the plasmodium, the most constant change in malaria. It probably occurs in every instance, though an unpigmented æstivo-autumnal form is believed by some to exist. Melanæmia is present at all times during fresh infection and may occur shortly after the fever has disappeared, as in the half-moon forms. After infection it usually disappears within forty-eight hours. It is practically pathognomonic of malaria, though it is said to occur in recurrent fever, in melanotic new growths, and in Addison's disease. It is found in the polymorphonuclears and sometimes in the lymphocytes. It is seen in the smaller capillary walls where their diameter suddenly decreases (lung alveoli, intestinal villi, appendices epiploicæ, renal glomeruli, dura mater, cerebral convolutions, spleen, liver, and bone-marrow); the capillaries may be occluded by pigment. The lymph-glands are usually free of pigment, with the possible exception of the portal glands. Besides this pigment there is also the copper-colored hæmosiderin. This may be found not only in the vessels but in the parenchyma of the liver.

(*C*). **Spleen.**—Enlargement of the spleen (first described by Andouard, 1803), is the third important finding. It is palpable in 88 per cent. of the cases and is rarely anatomically absent (Laveran, Plehn, Kiener, and Kelsch) in tropical forms. Its size, hardness, and plumpness, are proportionate to the length of the fever. They increase with its rise, and remit between attacks. In acute infections the spleen is increased three to five times its normal weight (five ounces); it is soft and friable; the veins are dilated, and the parenchyma is flooded with infected cells and macrophagic cells; and there are sometimes areas of focal necrosis. The Malpighian bodies contrast strongly with the pulp. There is often a sticking pain over the organ, which is increased by moving the diaphragm. Pain is due to capsular tension, or perisplenitis, and is often associated with pain and tenderness in the bones. As the disease subsides the splenic intumescence disappears but may occasionally remain (fever-cake). In chronic infections the spleen is permanently enlarged, reaching even into the pelvis. It may weigh two to ten pounds. The capsule is thickened, even calcified. Perisplenic adhesions occur and friction is frequent. Sometimes also a systolic murmur is heard (Griesinger). The organ is plump, the veins are patent, and the acute hyperæmia and focal necrosis lead to increase of the connective tissue. The pigment, enormous at first, gradually decreases. Ultimately little remains of the pulp, follicles, and splenic function.

Rare complications are pressure on the intestines, nerves, and ureters, torsion of the pedicle of a wandering spleen, rupture (of which Choux has collated 147 cases), abscess, or perisplenic abscess.

(*D*). **Skin.**—The skin is congested during the chill, and the vasomotor constriction gives way, during the febrile rise, to hyperalgesia and vaso-dilatation. Miliaria appear in the sweating stage. Herpes is very frequent, mostly nasal or labial; erythema is not common except when it results from quinine or antipyrin. Erythema nodosum has been observed in children. Roseolæ are most rare. Œdema occurs chiefly in chronic, and ambulant cases, and an earth-colored cachectic hue appears in chronic cases. The mucosæ are gray but not pigmented. Slight icterus is not infrequent (urobilin icterus), and sometimes it is intense. Infrequent complications are urticaria, pemphigus, petechiæ, pigmentation, a measles-like eruption, furuncles, phlegmon, noma, gangrene, or erysipelas.

(*E*). **Circulatory Symptoms.**—The heart is little involved, though sometimes myocardial degeneration and dilatation may occur during the paroxysm. The pulse rises parallel with the fever, small at first from vaso-constriction, later dicrotic and full from vaso-dilatation. Endocarditis is a rare development, as are angina pectoris, aortitis, or phlebitis. The author observed one case complicated with ulcerative endocarditis and aortitis.

(*F*). **Respiratory Symptoms.**—Dyspnœa and pulmonary congestion may occur during the paroxysm. These are due to parasites and phagocytes accumulating in or even occluding the capillaries of the lung. Chronic bronchitis, emphysema, and chronic interstitial pneumonia, occur as complications. Bronchopneumonia is frequent in malarial cachexia. Genuine lobar pneumonia occurs only as a mixed infection, since the malarial parasite possesses no phlogogenic properties. Plasmodia have been seen in the sputum. Pneumonia is atypical, is often present without rusty sputum and without chill, and is especially dangerous in malarial cachexia, the mortality ranging from 60 to 78 per cent. Pulmonary tuberculosis and pleurisy are rare.

(*G*). **Digestive Symptoms.**—The oral mucous membrane is dry and is sometimes the seat of hemorrhage, or gangrene; the tongue is heavily coated and the breath is foul. The parotid gland is sometimes swollen. The stomach may be the seat of erosions, catarrh, hyperæmia, or of infarction with plasmodia. Anorexia, pyrosis, vomiting, and cardialgia, are common symptoms. Ulceration from amyloid degeneration is observed in only a few chronic cases. Hæmatemesis from chronic splenic tumor has proved fatal.

THE INTESTINES.—Swelling of the lymphatic structures, erosions, catarrh, hyperæmia, thrombosis from parasite aggregation, meteorism, constipation, and rarely diarrhœa, are observed. Acute abdominal pain is not uncommon, but occurs without obvious cause. Pain may simulate peritonitis. Hemorrhage is rare. The disease may resemble dysentery, with ulcers in the large and small gut. Acute ascites may result from pylethrombosis, from plugging of the capillaries by melaniferous cells, or from congestion of the peritoneum.

THE LIVER.—In acute infections the liver is swollen, hyperæmic, or is the seat of focal necrosis. Its endothelial cells are swollen, it becomes macrophagic, and its veins are full of pigment, though as a rule few parasites are present. Icterus may develop from sepsis or red-cell destruction; it is usually polycholic. Pigmentation from degenerated red cells is more common than actual melanosis. In chronic infections the liver may weigh eight or ten pounds, is hard and smooth, and has a thick capsule. The changes, *seriatim,* are focal necrosis, atrophy or partial regeneration, and pigmentation with hyperæmia. As a result of atrophy and regeneration, angioma, lymph-cysts, or abnormal lobules develop; hyperplasia of the perilobular connective tissue occurs with ultimate cirrhosis. Atrophic and also hypertrophic cirrhosis may follow chronic malaria (Frerichs, Bamberger), but Marchiafava, Bignami, and Osler, rather question its malarial origin, because other causes are also present. Amyloid degeneration (Rokitansky), and simple atrophy from pylethrombosis and marasmus, are rather uncommon sequels.

(*H*). **Genito-Urinary Symptoms.**—Polyuria is often observed during the paroxysm, which sometimes reaches two to four quarts, despite the severe sweating. The urine is usually decreased in the æstivo-autumnal type. The post-malarial polyuria of convalescence is more frequent. In one instance the author observed a veritable diabetes insipidus as described by Sydenham and Mosse.

The urea, iron, chlorides, and specific gravity, of the urine are increased. Phosphates are decreased during the paroxysm, and increased afterwards. Albuminuria is present in 25 to 40 per cent. of cases, and usually disappears with the paroxysm, but sometimes lasts two to three weeks, or longer. Nucleoalbumin has been found in the urine, besides peptone, acetone, and indican. Ehrlich's diazo reaction may often be obtained, in 5 per cent. (Thayer and Hewitson) to 40 per cent. (in the author's experience). Glycosuria is occasional. The toxic coefficient is increased. Nephritis occurs in 4.5 per cent. of the cases but is not often fatal. The parasites are rarely found in the glomeruli or veins but frequently abound in the intertubular capillaries. Glomerular desquamation and epithelial exfoliation, endocapsular exudation, necrosis, and cast formation, develop in the convoluted tubules. Chronic parenchymatous and interstitial nephritis may result from previous malarial infection. The large white kidney, followed by secondary contraction, is considered by some to be a frequent sequel. Hæmoglobinuria, anuria, and uræmia, occur in blackwater fever (See PERNICIOUS FORMS). Gangrene of the genitalia is an uncommon sequel. Orchitis is usually due to other diseases. Metrorrhagia and abortions occur frequently.

(*I*). **Nervous Symptoms.**—The parasite and its presumed toxin affect especially the nervous system, and for this reason von Swieten has called malaria a neurosis. There is scarcely a nervous symptom which is not occasionally observed in malaria. The sensorium, which is involved slightly in mild cases, may be more seriously affected in the more severe types, the symptoms ranging from simple exaltation to coma and paralysis. Headache is usual, and neuralgias are frequent, especially in the first and second branches of the fifth nerve. Cerebral confusion was noted by the writer in two ordinary tertian cases. In pernicious types especially, it is not always easy to determine whether nervous symptoms are due to simple toxæmia or to cerebral thrombosis. The central nervous tissues are usually distinctly hyperæmic, and are anæmic only in the algid or choleraic types. The endothelial cells of the vessels are fatty, swollen, and pigmented. Thrombosis in grave cases may result from swelling of the endothelium, though it is usually caused by the rough infected red cells. Thrombosis may lead to multiple small extravasations of blood. The infected erythrocytes never leave the vessels.

Symptoms of paralysis (aphasia, hemiplegia, paraplegia, and monoplegia), or of motor irritation (tremor, chorea, tetany, athetosis, contractures, epileptic attacks, or reflex spasms, such as sneezing or vomiting), may possibly occur in urgent types. Neuritis, neuroses, psychoses, spinal paraplegia with atrophy and bladder disturbance, ascending spinal paralysis, bulbar symptoms, symptoms resembling multiple sclerosis (Spiller), tabes, acute ataxia (Kahler and Pick), and paralysis agitans, are rare sequences.

SPECIAL SENSES.—The eyes are reddened. Herpes may, in rare cases, affect the cornea. Retinal hyperæmia, hemorrhage or atrophy, neuroretinitis, or choroiditis, may occur (especially in pernicious cases). Otalgia, otitis media, labyrinthine vertigo, and deafness, are very exceptional.

(*J*). **Muscles and Bones.**—The muscles are rarely infiltrated or degenerated, but are often painful. Rheumatism is infrequent except in India. The bone-marrow is at first fatty and pigmented; the fat and pigment then disappear with marked vascularization, and, finally, with proliferation of the marrow-cells and connective-tissue thickening. Bone tenderness is often noted.

Relapses.—Relapses caused by the awakening of an old infection almost invariably occur in all forms, and are most obstinate in the æstivo-autumnal type; the blood-findings are almost always positive. Reinfection is also frequent.

Chronic Malaria.—Untreated infection, or reinfection, persists for months, especially in the æstivo-autumnal variety. It is accompanied by irregular fever, marked anæmia, the parasite in the blood, and splenic and hepatic swelling; repeated attacks always occur. In severe chronic cases, the *malarial cachexia* develops, usually after years of infection. It is most marked in intensely malarial regions, in the æstivo-autumnal form of the infection, and in reduced subjects. The fever is either slight, or entirely absent; sometimes it is irregular and reaches 102° to 103°. The essential features of malarial cachexia are (1) anæmia from defective blood regeneration, with its general symptoms, such as œdema, rapid pulse, venous thrombosis, and hemorrhages in the skin, retinæ, and stomach; (2) vascular changes, possibly due to pigment deposit, and (3) enlarged spleen and liver. Gangrene, chronic nervous symptoms, alimentary and respiratory catarrh, amyloid disease, liver hypertrophy, and extreme malnutrition, may occur.

Relation to the Other Diseases.—Malaria and typhoid do not positively exclude each other; however, they rarely co-exist in this country. The detection of the plasmodia on the one hand, and typhoid bacillæmia or the response of the blood to the Widal test, on the other hand, are necessary for diagnosis. *Surgical* operations may arouse a latent malaria. Sunstroke may initiate a pernicious attack. Erysipelas, septicæmia, suppuration, dysentery, cholera, smallpox, scarlatina, recurrent fever, scurvy, and tuberculosis, may very infrequently co-exist with malaria.

Pernicious Malaria.—Pernicious malaria, in the widest sense, is malaria which threatens life. It is characterized by severe, obstinately recurring fever, pronounced intractable anæmia, pernicious visceral symptoms, frequently by abortive paroxysms without chills, by considerable depression, tendency to recurrence, and the presence in the blood of the æstivo-autumnal plasmodium. Pernicious malaria constitutes but a small proportion of cases of æstivo-autumnal malaria. Very few pernicious cases have occurred from infection with the ordinary benign tertian parasite (Ziemann, French, and one case observed by the author). The pathogenesis of the pernicious type lies in the inelastic, irregular, infected red cells which cannot pass the visceral capillaries, and therefore slow the blood current. In cases of capillary hemorrhages, only the normal red cells escape from the vessels, and the parasite-laden red corpuscles always remain in the vessels. Regressional organic changes may occur in the cerebrum, gastrointestinal mucosa, spleen, liver, and kidney. The melanosis, phagocyte aggregations, and endothelial swelling are of wholly secondary importance. Accessory factors are (*a*) arteriosclerosis, renal disease, cardiac dilatation (from toxæmia), alcoholism, sunstroke, overwork, malnutrition, and lack of acclimatization; (*b*) the number of parasites and their abundance in internal organs. Parasites of the æstivo-autumnal group, with large blocks of central pigment, usually indicate many plasmodia in the viscera. The parasites are often few in the peripheral blood or, indeed, in the blood of a splenic puncture, though they may be found in very great numbers in the brain at autopsy (Bastianelli, Marchiafava, and Bignami); (*c*) great activity in multiplication may be observed even in the peripheral blood where they are usually less abundant during the sporulating stage. Several plasmodia are sometimes found in a single corpuscle; the spores may number ten, twenty, or even thirty; (*d*) increased toxicity of the malarial parasites is purely hypothetical, as their toxin has not yet been isolated; but the necroses in the spleen, liver, and kidney, and the great morphologic alteration in the "brassy" red cells, are at least suggestive.

1. The pernicious *typhoid form* (*perniciosa typhosa*), is inappropriately called typhomalaria. Its clinical picture is variable; chills are often absent; the sensorium is involved to a variable extent, and depression, delirium, insomnia, headache, and backache, occur. The fever is atypical, intermittent, or remittent (subcontinuous), resolves by crisis or by lysis, and is sometimes hyperpyretic or subnormal in register. Quinine disturbs or breaks the type. The pulse and respiration are increased. Gastro-intestinal symptoms may be marked; vomiting sometimes of bile or blood, epigastric pain and tenderness, frequently tympany, and thin, dark movements of the bowels, occur. The spleen and liver are usually tender and swollen. An irregular bronchitis (sometimes dyspnœa), hæmoglobinuria, albuminuria, or urobilinuria, are frequent. The course is usually benign, and responds to quinine; the mortality is 10 per cent., and, in adynamic forms, 25 per cent. Fatal cases usually terminate with nervous symptoms, and especially with coma. The differential diagnosis was impossible before the blood-findings were known, but was often made *ex juvantibus* by administration of quinine. Without blood examination malaria and typhoid may be confused; they very rarely co-exist; benign or malign malaria may resemble typhoid closely, with slow, dicrotic pulse, splenic tumor, headache, fever-curve, cerebral and abdominal symptoms, and even roseolæ and intestinal hemorrhage; and, on the other hand, erratic typhoid with remittent temperature, chills, and sweats, or typhoid in the latter part of its normal course, may simulate malaria.

Baccelli gives the following differentiation:

MALARIA (Subcontinua typhosa)——*vs.*	——TYPHOID.
Begins intermittently.	Progressively remittent.
Remissions very irregular.	Very regular, and in the morning; step-like.
Temperature may be high at first.	Considerable fever, reached only on the third or fourth day, or later.

(Concluded on next page)

MALARIA (Subcontinua typhosia)——*vs.*——TYPHOID.

Headache in beginning is rare, variable in site and intensity, pulsating and neuralgic.	Early, permanent, frontal, oppressive, characteristic.
Eyes dull, subicteric from beginning.	In early stages, glistening eyes; no icterus.
Stuporose facies, dry tongue, and sordes, less.	Marked.
Breath nauseating.	Mouse-like.
Delirium present from beginning, recurs with temperature and other symptoms.	Occurs first with advanced stage, is constant and varies only in intensity.
If lung congestion occurs, it begins suddenly; foci change from lobe to lobe, recurrently; dyspnœa from deranged circulation.	Slow development, hypostatic; dyspnœa may rarely result from tympany, etc.
Nervous restlessness, jactitation.	Stupor, sopor, prostration.
Meteorism can occur, but is not characteristic as in typhoid; stools darker.	Meteorism and diarrhœa begin gradually, and develop to a high degree; ochre stools.
Liver congestion frequent; subicterus, even icterus.	Less congestion, rarely icterus.
Herpes is frequent.	Rare; roseolæ.
Cycle, none.	Very typical.
Blood: plasmodia and melanæmia.	Widal reaction and typhoid bacillæmia.
Recurrence.	Relapses less frequent.
Convalescence more rapid and easier.	Tardy.
In adynamic: icterus, hæmoglobinuria, leukocytosis, great anæmia.	Does not respond to quinine, which practically always reduces fever and other malarial symptoms.

2. Of *cerebral forms,* the comatose (*perniciosa comatosa*) is the most frequent. Physical depression, sopor, melancholia, and impaired memory, may exist in the preliminary stage; later, contracted pupils, involuntary evacuations, lessened reflexes, or paralysis, may develop. The coma may be intermittent. Its second appearance is longer and more dangerous than the first. If the patient recovers, bradylalia, scanning speech, and insufficient motility of the lips or tongue, may be observed. The pulse at first is generally slow and regular, but as death approaches it becomes soft, thready and rapid. Death is usually cardiac. The respirations vary, being quiet or noisy, hurried, or of the Cheyne-Stokes type. If fever is present it is irregular. The author has seen four such cases in Chicago. Apoplexy, uræmia, and sunstroke, must be considered. The splenic tumor *per se* suggests a blood examination. The "meningeal type" occurs especially in chil-

dren, and in these cases the slow pulse, headache, rigid and tender neck, sopor, convulsions, involuntaries, hyperæsthesia, and ultimate coma, can be correctly interpreted only after a blood examination. Delirium may be the main symptom, and may appear as a mania, as fixed ideas, hallucinations, or as melancholia. The rare convulsive type may be (*a*) generalized, and may simulate tetanus, hysteria, uræmia, or epilepsy especially in children, or (*b*) localized, with such symptoms as contractions, trismus, eye deviations, or opisthotonos. The paralytic form is the least frequent type. Hemiplegia with aphasia is more common than monoplegia, focal paralysis, or the bulbar form.

3. The *algid pernicious type* occurs with cyanosis, collapse, Hippocratic expression, and marked alimentary symptoms, early death being the rule.

4. The *pernicious gastro-intestinal* variety is most frequent next to the cerebral type, and occurs with vomiting, pain, epigastric distress, cardialgia, icterus, and dysenteric manifestations. In a soldier from Cuba, the author observed profuse hemorrhages from the bowel. A prompt response to quinine followed the detection of the æstivo-autumnal plasmodium.

5. The *pernicious biliary fever* ("jungle fever") occurs with vomiting, polycholia, icterus, nervous symptoms, hemorrhages, adynamia, œdema, and dark albuminous urine. The course is usually ten to twelve days, but even after the administration of quinine this type may be lethal. The blood examination differentiates it from yellow fever.

6. The *hemorrhagic types* resemble Werlhoff's disease, and scurvy.

7. The *febris biliaris hæmoglobinurica* ("black-water fever") occurs in South America, in Africa, and in our Southern States. The factors in its production are syphilis, alcoholism, repeated malarial attacks, exposure to changes in climate, undue exertion, and cold, as well as local influences, and in some instances the use of quinine. Stephens (1903) in 95 cases found the parasite in 96 per cent. before, and in 62 per cent. at the onset of the attack. The etiological role of quinine is questioned, but some other factor is apparently more potent. The cardinal symptoms of this type are (*a*) hæmoglobinuria; the urine is always albuminous, neutral, or alkaline, in reaction; hæmoglobin is detected only after the destruction of at least one-sixth of the circulating red blood corpuscles; (*b*) icterus, invariable except in the mildest cases; (*c*) irregular fever; (*d*) the detection of the plasmodia. The mortality (40 to 50 per cent. of the cases) is due to syncope, hemorrhages, anuria, and coma.

Diagnosis.—1. From Symptoms.—A probable diagnosis can often be made, as, for instance, from typical intermittent fever which occurs early in the day and is distinct from the vesperal hectic or septic fever. The three elements of the paroxysm are most marked in the benign malaria. In the æstivo-autumnal form the chills may often be absent. Splenic enlargement, pain, and herpes, are always suggestive, but they also occur frequently in ephemeral fevers, acute gastritis, pneumonia, meningitis, rheumatism, and influenza, and more rarely in typhoid, tuberculosis, and tuberculous meningitis. Much emphasis was once placed on various intermittent manifestations, such as neuralgia, vertigo, herpes zoster, and alimentary and respiratory catarrh. To-day equal skepticism is properly entertained in regard to their malarial origin.

2. The Diagnosis ex Juvantibus.—Laveran excluded malaria, when the fever persisted for more than four days, with the daily administration of 20 to 30 grains of quinine. In tropical forms more quinine may be necessary for this purpose. It must be remembered, however, that other diseases than malaria may subside under the administration of quinine.

3. The Plasmodium.—Mistakes in interpretation are possible. The plasmodium may be mistaken for vacuoles resulting from mechanical injury to the red cells; it may be mistaken for particles of immersion oil which may reach the specimen; or for retraction of the hæmoglobin, which may show the stroma of the corpuscle, and for poikilocytosis. Possibly less mistakes are made when the inexperienced observer examines the fresh blood. The plasmodium may also be confused with swarming particles from the red blood-cells, or with blood plates which are sometimes mistaken for spores. The differentiation from these is easily made in the stained specimen. Leukocytes containing pigment are distinguished by their large nucleus and by their amœboid movement, the latter of which is lacking in plasmodia sufficiently large to cause confusion.

POSITIVE FINDINGS.—The malarial parasite is found in the blood of malarial patients only. Osler found them in every instance, in 531 cases. It is usually stated that they occur in largest numbers before or at the time of the paroxysm, though it must be admitted that the first examinations in early fever are often negative. Previous administration of quinine may render the findings obscure. Melanæmia is practically pathognomonic. Pigment lying free in the plasma is of no importance, for even the greatest care will not eliminate the possibility of contamination.

NEGATIVE FINDINGS.—These are of relative value only, and depend upon the skill of the observer, and the time of the examination. In Baccelli's series there were negative findings in only 2 per cent. of the cases. If plasmodia are not found in the circulating blood, the spleen may be punctured—a proceeding, which is not without danger. Stevens regards an increase of the large mononuclears to 20 per cent. as a safe sign.

Differential Diagnosis.—1. From *typhoid* fever (see PERNICIOUS MALARIA AND TYPHOID).

2. In *sepsis* (see table under typhoid), pyæmia, ulcerative endocarditis, liver abscess, septicopyæmia, hectic pulmonary phthisis, suppuration in the intestinal and urinary tracts, and irregular fever, may be confounded with malaria. However, we find in malaria the plas-

modium, melanæmia, response to quinine, and absence of leukocytosis; whereas in sepsis, leukocytosis, positive blood cultures, and septic localizations, are very frequent.

3. *Pneumonia* with remittent curve, irregular grippal temperature, and hysterical fever; gastro-intestinal catarrh; Malta fever; recurrent fever; miliary tuberculosis; fever in malignant new growths and especially in sarcomatosis; filarial infection; pernicious anæmia; splenic anæmia; leukæmia; pseudoleukæmia; Weil's disease; and acute febrile jaundice, may be confused with malaria, if careful and repeated blood examinations are not made. In Weil's disease, muscular pains, especially in the calves, and decolorization of the stools, which are usually polycholic in malaria, are suggestive. Pernicious malaria (*v. s.*) may superficially simulate apoplexy, sunstroke, meningitis, cholera, dysentery, or purpura.

Prognosis.—The degree of infection, type of parasite, reinfection, health and habits of the individual, and antecedent renal or cardiac disease, are the most important considerations. The prognosis must be conservative in pernicious and cachectic forms. In Italy the yearly death-rate is 54 per 1,000, and the French lost 6,000 soldiers in Madagascar in three months. On the other hand, fully 20 per cent. recover when given rest in bed and a full diet, as is often shown in cases which are treated with placebos, and saved for clinical demonstrations. Spontaneous recovery is not yet fully understood. The factors are the phagocytes in the spleen, in the bone-marrow, and, to a less degree, in the endothelium of the splenic, hepatic, and cerebral vessels, Kupfer's cells in the liver, and possibly the leukocytes which are usually decreased in other than pernicious types.

Treatment.—1. **Prophylaxis.**—Prophylaxis now embraces more than personal prevention. In Italy and other countries it is a national economic problem. Drainage, the filling in of stagnant pools and swamps, and the planting of pines, eucalyptus trees and sugar-cane, have proved most successful, though sometimes accomplished at great sacrifice of life. The drainage of the great Bordeaux swamp in 1805 cost 3,000 lives. In digging canals or building roads, immunes should be employed, but whites may work successfully if they avoid excessive labor in the hot sun, alcohol, and exposure to the night air. Nocturnal infection by mosquitoes may be minimized by using nettings on the doors and windows. The pools where the insects breed should be filled in, or covered with crude petroleum to kill the larvæ. The stocking of semistagnant streams with fish is also of value. The prophylactic use of quinine in daily doses of 5 to 10 grains is effective.

2. **Specific Treatment.**—Quinine is one of our few specifics. It has been used for ages in Peru. The plant was known as the "tree of health" to Pizarro in the conquest of Peru. Quinine was introduced over one hundred years later into Europe by del Vego (1640). It acts on the malarial organism while it is developing (Marchiafava and Bignami); when the parasite has transformed all the hæmoglobin into melanin, and segmentation begins, quinine is ineffectual. All such

malarial manifestations as fever, acute splenic tumor, and anæmia, are cured by quinine except in pernicious forms. It acts as a direct protozoan poison. Binz thought it entered into such close union with the hæmoglobin that the parasite was suffocated. Just as syphilitics tolerate much mercury and iodide, so malarial subjects escape many or all of the symptoms of cinchonism, such as tinnitus aurium, cephalic distention, and slight deafness resulting from therapeutic doses (gr. x.); deafness, disordered vision, flushed face, and cerebral congestion (sometimes relieved by epistaxis), vomiting, staggering, giddiness, tremor, and twitchings, resulting from larger doses, and stupor, delirium, cardiac oppression, complete deafness, and amaurosis, dilated pupils, convulsions, paralysis, hæmoglobinuria, dyspnœa, coma, weak heart, or hemorrhages resulting from ordinarily toxic doses.

Administration.—If idiosyncrasy exists, cinchonism is lessened by ext. ergotæ fl. ʒss, or, better, by sodium bromide ʒss. Its antiperiodic action is enhanced by combining it with opium gr. ss. Such laxatives as cream of tartar and calomel are unnecessary. Pills are often insoluble, and the dose is therefore given in capsules, *in powders involved in starch wafers*, or with one drop of sulphuric acid to each grain of the sulphate. Chloroform, coffee, cognac, and extract of glycyrrhiza, but poorly disguise its bitterness. If the stomach rebels, a warm-water injection with equal parts of quinine and tinct. opii deodorat. may be used. In severe cases double the ordinary dose of the bimuriate of quinine and urea (Quininæ carbamas bichlorida) should be used hypodermatically, but, though every care be taken, abscesses develop too often. In comatose and other pernicious types, Baccelli injects quinine intravenously (sterilized solution of sodium chloride, quinine muriate, and water, in the proportion of 1, 10, and 100, parts respectively).

Time of Exhibition.—If the fever recurs regularly, grs. x. to xx. should be given in the interval to act on the growing young forms in the red cells, and this should be repeated in three to five hours before the paroxysm, in order to have the drug in the circulation when the spores are scattered (Torti and Golgi). If the fever is continuous or sub-continuous, or if the type is severe, doses of the same amount should be given daily. Large doses are recommended because they are more effectual, the symptoms are frequently severe, and small doses are quite useless and sometimes only serve to aggravate existing malaria and develop quiescent forms (Steudel-Kuchel). Opinions vary as to the treatment of black-water fever; one view maintains that it is caused by the parasite, and another that quinine is its cause. Quinine, however, should be given only when plasmodia are found, and should be withheld after they disappear.

3. Symptomatic Treatment of the Paroxysm.—During the vasoconstriction of the chill, morphia gr. ¼ and nitroglycerine gr. 1/50 hypodermatically, and spirits of chloroform ʒj., in hot whiskey and wate afford great relief. The fever is short-lived and requires little inter ference other than the ice-bag or cap, and cool sponging. The sweating requires no treatment. A weak heart should be supported by morphine, strychnine and camphor by the hypodermic needle, heat and saline enemata or infusions.

In chronic malaria, iron and full increasing doses of arsenic should be combined with quinine. Arsenic is said by Durham to act on the gametocytes.

The contraindications to the use of quinine usually listed, (gastro-intestinal inflammation or irritation, middle-ear, genito-urinary, and cerebral inflammation,) are negligible in the treatment of malaria.

SEPTIC AFFECTIONS.

Synonyms.—Sapræmia, septicæmia (sepsis), pyæmia, septicopyæmia.

Definition.—*Intoxications* are the result of the absorption of substances which are elaborated for the most part by microörganisms, but possibly also to a less degree by living cells. *Infections* are always due to microörganisms. *Sapræmia* is the absorption of putrescent substances but not of the germs which develop them. *Septicæmia* (sepsis) is the presence in the blood of microörganisms (bacteriæmia), which are usually pyogenic, without hæmatogenous suppuration or metastasis. *Pyæmia* is infection, caused by microörganisms, which leads to hæmatogenous metastasis or suppuration. Septicopyæmia is coexistent septicæmia (bacteriæmia) and pyæmia (metastatic suppuration).

General Groups.—1. Some infections—such as carbuncles, puerperal parametritis, diphtheria, and tetanus—are wholly *local* so far as the causal microbe is concerned, and their symptoms are due to absorbed toxins, toxinæmia (toxæmia).

2. Other infections—such as typhoid, pneumonia, carbuncles, puerperal sepsis, anthrax, and gonorrhœa, which cause *bacteriæmia* or *septicæmia—at first local,* may *become general* when their germs enter the blood. Some of these infections, especially typhoid and pneumonia, which were previously considered local, are always general.

3. Still other infections, often resulting from the same germs, cause *metastatic suppuration* (*pyæmia*). Among these are multiple abscesses from carbuncle, puerperal fever, typhoid osteomyelitis, pneumococcic arthritis, and gonorrhœal endocarditis. It is frequently difficult or impossible to preserve clinically these types which so often blend; (*a*) what is seemingly toxæmia may prove bacteriæmia on examination of the blood; (*b*) apparently pure bacteriæmia may prove pyæmia at necropsy; (*c*) all three grades may occur successively; (*d*) the primary infection—*e. g.*, with scarlatina, tuberculosis, and diphtheria—whether local or general, may be complicated by secondary, or mixed infection, especially by the streptococcus; (*e*) again the symptoms vary, first with the variety of microbe and its virulence;

second, with its localization (in the throat, lung, pelvis, etc.); and third, with the physiological resistance. Subjects of malignancy, cardiac, vascular, nephritic, cirrhotic, and other disease, readily succumb to an ultimate sepsis to which the name of *terminal infection* is applied. For these reasons we will discuss septic infections as a whole, and indicate such special etiological and clinical variations as seem necessary.

Etiology of Septicopyæmia.—The causal organisms of septic infections are numerous. The most common are the pyogenic cocci, the pneumococcus and the colon bacillus; less frequent are the gonococcus, anthrax bacillus, typhoid bacillus, Friedländer's pneumobacillus, meningococcus, bacillus pyocyaneus, influenza bacillus, gas bacilli (Fraenkel's bacillus phlegmones emphysematosæ; bacillus aërogenes encapsulatus), and possibly the micrococcus tetragenus, the spirilli and bacilli of Vincent, and the proteus group. Although puerperal fever, primary wound fever and severe late pyæmic infection were known to Hippocrates, and also to Willis (1682), Semmelweiss (1847), and others, it was reserved for the bacteriological era, building on Virchow's work, to clarify this group of affections. Though Wunderlich (1847) spoke of spontaneous pyæmia, and Leube (1878) coined the term cryptogenetic (occult) septicopyæmia, careful clinical and necropsy investigations have gradually revealed the *atrium of infection*. Although some cases are still obscure, the source of infection is now found in 90 to 94 per cent. of cases; (1) *skin* lesions, such as acne, furuncles, felons, and bites, (staphylococcus most frequent; less often streptococcus); (2) *throat* (streptococcus); (3) *nose* (not frequent in sepsis, though probably the atrium in meningitis for the meningococcus and pneumococcus); (4) *ear* (pneumococcus and streptococcus); (5) *lungs* (pneumococcus); (6) *intestines* and *bile tracts* (colon bacillus; typhoid bacillus, gall-stones with infection, appendicitis, dysenteric ulcers); (7) *urinary* tract (pyelitis from colon bacillus; vesical and urethral infections chiefly by the staphylococcus, far less by the streptococcus and gonococcus); (8) *vagina* and *uterus* (chiefly streptococcus).

General Pathology and Symptomatology.—(*A*.) **General Toxæmic Symptoms.**—1. *Fever.*—Fever is usually the first clinical symptom and occurs more often abruptly than gradually. The virulence of the germ and its toxin (rather than its variety) governs the intensity of the toxæmic symptoms. In general the fever tends to be remittent or intermittent. Lenhartz, in his work on septic infections, concludes that (*a*) the streptococcus infections usually produce irregular intermittent fever with moderate variations (sometimes remittent or in severe cases continuous fever); (*b*) the staphylococcus usually causes weakly remittent or continuous fever; (*c*) the pneumococcus produces remittent or continuous fever; (*d*) the colon bacillus and gonococcus cause intermittent fever with great variations. Fever may be absent in very severe forms, especially before death.

2. *Chills.*—Chills occur both in septicæmic and in pyæmic forms, are usually irregular, but may occur daily or two or more times a day.

3. *Nervous Toxæmia.*—Nervous toxæmia may be pronounced. It may appear as unrest, delirium, sopor, convulsions, dry tongue, and the status typhosus, or it may resemble meningitis. The brain may be clear, as it is in many puerperal infections.

4. *Vasomotor* and *cardiac weakness*, with increased, often irregular, pulse, may be present.

5. *Increased respiration* and bronchitis may be noted.

6. *Albuminuria* and acute nephritis are common.

7. *Septic diarrhœa*, in which the dark movements number six to ten daily, may be observed.

8. *Acute splenic tumor* is present.

9. *Anatomically* we find cloudy swelling of the heart, liver, and kidneys; a vascular, soft, moderately swollen spleen; often acute nephritis; hemorrhages into the skin, retina, and mucous and serous membranes, with or without bacteria; lung œdema, and, where there is marked anæmia, fatty heart, hemorrhage into the bone-marrow, and increased iron deposit in the liver.

(*B.*) **Blood-Findings.**—1. *Bacteriæmia* is found in 50 per cent. of the cases clinically, and in 95 per cent. at autopsy (Lenhartz). Some circulating bacteria are apparently killed by the blood. In most streptococcic and staphylococcic infections, these organisms are found in the blood.

2. *Polymorphonuclear leukocytosis* is frequent, though not invariable.

3. *Anæmia*—Marked anæmia is usual, and sometimes even resembles the pernicious form.

(*C.*) **Metastases.**—They are apparent clinically, or first found at necropsy. Metastases occur through the veins or lymph-vessels. Thrombo-phlebitis develops at the seat of primary infection, and disintegrated thrombi, containing bacteria, escape into the venous circulation. They lodge in the lungs or, passing them, reach the left heart and arterial system. Purulent lymphangitis may, in the same way, cause metastatic suppuration. If "end arteries" are plugged, infarcts result; if the arteries are not terminal, small foci develop. Not all the infarcts suppurate. This difference is explained by the fact that the germs found in them are less virulent, or that the embolic material is bland or free of microbes. Foci occur in organs in which there are end arteries and in which the circulation is slow, especially, therefore, in the liver, kidney, and spleen. (1) *Cerebral metastasis* may cause meningitis, brain abscess, or extensive softening (encephalomalacia with aphasia and hemiplegia). (2) *Cardiac* metastasis develops in 21 (even 63) per cent. of the metastatic group; endocarditis is localized chiefly on the mitral (62 per cent.) and aortic (22 per cent.) valves; it is attended by a heart murmur in 60 per cent., and often by pericarditis; the heart muscle is sometimes the seat of abscesses. (3) *Lung* localization may cause multiple or single abscesses, and often sero-fibrinous or purulent pleurisy. 4) *Renal* localization may result in nephritis or suppurating infarcts, with albuminuria, cylindruria, pyuria and bacteriuria. (5) *Splenic* localization usually results in enlargement, frequently in pain, and rarely in perisplenic friction. (6) Localization in the *bones*, may cause early fugitive bone pain, or later, fixed osteomyelitic tenderness over one or more bones, with inflammatory signs; in the *joints* (as in the post-febrile arthritides), it may cause simple pain, or serous or purulent synovitis; in the muscles it may produce abscesses, purulent œdema, or Wagner's polymyositis. (7) Localization in the *skin* may cause scarlatiniform, morbilliform, roseolous or urticarial eruptions, resembling the antitoxin rashes. Ecchymoses occur in 50 per cent. of fatal cases; they are usually late, often symmetrical, and sometimes occur on the tip of the nose, fingers, toes, or ears. Pustules and vesicles are less frequent; herpes occasionally develops. (8) Localization in the *eyes* may produce retinal ecchymoses (in at least 33 per cent.), which were first described by Litten. They are round, or irregular, or appear in streaks; they often have white centers (which are usually small), range in number from three to six, and appear late or just before death; the white spots of Roth (8 per cent.) were once considered inflammatory, and are still unexplained. Least frequently eye localization results in panophthalmitis, of which 33 per cent. is due to ulcerative endocarditis. (9) The *peritoneum* is chiefly involved in puerperal forms. (10) The slightly enlarged *liver* is more often due to toxic parenchymatous degeneration and cardiac weakness than to abscess, which occurs (Baerensprung) in only 15 per cent. of cases of surgical septicopyæmia. Intestinal infarction is rare.

Special Symptomatology.—This may be considered etiologically o topographically. To avoid repetition, only the leading characteristic are presented.

(*A*). **Etiological.**—1. *Streptococcic infection* is the most frequen primary and secondary septic infection. It constitutes 66 per cent of all forms (Mau), and is said to enter the blood before death in 3 per cent. of all fatal diseases. Its atrium is most often the throa and (in the female) the genitalia; it enters the veins (in puerpera infection) more often than the lymphatics (erysipelas and puerpera sepsis); it more often produces septicæmia (65 per cent.) than pyæmi (35 per cent.); the lungs are usually unaffected; endocarditis occur in 7 per cent.; its course is chiefly acute, though sometimes subacut (ulcerative endocarditis).

2. *Staphylococcic infection* usually enters by the skin and the fu runcle is the typical primary local lesion. On the eye-lids, nose, or lip this lesion is dangerous because infection may reach the facial vei then the ophthalmic vein and the cavernous sinus. Furuncle of the nec may reach the transverse sinus. Infections of the skin are the mo frequent cause of osteomyelitis. Meningitis is known to have followe a felon. The infection enters through the throat, first with difficul but then with increased malignancy (Kocher and Tavel). It trave by the veins or lymphatics; it produces metastasis in 95 per ce and endocarditis in 6 per cent. of the cases. Abscesses of the lu and kidneys are very frequent; the coccus is found very often in t

blood and urine. Pustular skin eruptions are frequent. Staphylococcic infection is usually acute.

3. *Pneumococcic Infections.*—In all cases of pneumonia (*q. v.*) the pneumococcus appears in the blood (pneumohæmia). In sepsis following pneumonia, it is assumed to enter the blood through the pulmonary veins. Infection through the ear or bile-tracts is less frequent; metastases occur in 25 per cent. and endocarditis in 6 per cent. Twenty-nine cases of meningitis were first reported by Nauwerk (1881), but it is yet unknown why lung infections so frequently cause brain metastases. The pneumococcus alone may produce pus in the joints, skin, thyroid, and serous membranes. Netter's statement that pneumococcic sepsis is less severe than other types may be true in some cases, but it is often malignant. It may be associated with the pyogenic cocci, or saprophytes.

4. *Colon sepsis,* which is less frequent, may follow wounds, suppuration in cholelithiasis, cystitis, pyelitis, or intestinal obstruction. Endocarditis is exceptional, and sepsis (78 per cent.) is more common than metastasis (22 per cent.). As the intestine is its normal host, it is sometimes regarded as an autoinfection.

5. *Gonococcic infection* is rather more benign than the other types. There may be gonorrhœal endocarditis (Bordoni, Uffreduzzi) (of which Loeb has collected 62 cases), pericarditis, and pleuritis, or the more common gonorrhœal "rheumatism." Thayer found the gonococcus in the blood (*v.* Gonorrhœal Infection).

Other forms are less frequent. These are the typhoid sepsis (typhoid suppuration in the pleura, thyroid, bones, meninges, muscles, ear, and parotids), sepsis from Friedländer's pneumobacillus, and the meningococcus. Pyocyaneus sepsis is attended by irregular or continuous fever, rapid pulse, vomiting, diarrhœa, and a hemorrhagic diathesis.

(*B*). **Topographical.**—(1) *Ulcerative endocarditis;* (2) *cholangitic sepsis;* (3) *otogenous sepsis* (*v.* Sinus Thrombosis, Meningitis, Brain Abscess) will be considered elsewhere. The two following surgical or obstetrical localizations are of importance to the general practitioner and internalist.

(4.) *Osteomyelitis* is either a solitary metastasis or part of a generalized sepsis. Even when a solitary bone is involved, the blood often contains the causal microörganism. It is due to the staphylococcus chiefly, then to mixed infection with the streptococcus, which is found in all fatal cases, or with the pneumococcus, and but rarely to the typhoid organism. Fraenkel has recently found that in most autopsies on typhoid and pneumonia cases, their causal bacteria are present in the bone marrow. Acute forms of osteomyelitis are most frequent in the growing bones of children. Trauma is a promoting element, and the onset occurs with chill, fever, vomiting, diarrhœa, headache, and bone pain. It is usually first seen (and often mistaken) by the general practitioner. Chronic forms are quite infrequent, but Kocher, Jordan, and others, have described cases, which, lasting one year or more (even thirty), may simulate sarcoma, or syphilis.

(5.) *Puerperal fever* is caused by the streptococcus (95 per cent.) or by the bacterium coli, staphylococcus, gonococcus, proteus, or anaërobic organisms. It is still disputed whether streptococci normally reside in the healthy vagina and whether autoinfection may occur from this source, but some cases seem clearly proved. It is sporadic or epidemic; it is promoted by anæmia; some women, however, seem immune, especially those in the lower classes (De Lee). Its varieties are (*a*) *toxæmia,* or sapræmia. The symptoms are caused by toxins or putrid decomposition products respectively. Even in cases apparently of this group, bacteriæmia may be found (colon bacillus, saprophytes, or streptococcus). The rare puerperal tetanus is wholly toxæmic. (*b*) *Lymphangitic form*—Lymphangitis, from infected vaginal or cervical wounds, may cause moderate parametritis, which is usually a benign local infection. The usual forms are: 1. General septicæmia, from vaginal or cervical wounds, which follows the lymph-vessels through the cellular pelvic tissue to the general circulation. The symptoms usually appear within a day or so after delivery. Death may result (in two to fourteen days), or encapsulation (the important prognostic element) may last over a month with massive cellulitis. 2. Lymphangitic *peritonitis* is more frequent (about 20 per cent. of fatal cases). It begins three or four days after delivery and is usually generalized and suppurative. Infection may travel directly to the peritoneum or, more often, by retrograde lymphatic routes. This form includes Virchow's "erysipelas grave internum puerperale." It is more common in labor at term than in abortion. The symptoms are chill, fever, tense pulse, vomiting, peritoneal pain, distention, and often effusion, and diarrhœa. In many apparently purely peritonitic cases bacteriæmia is found. Early death is the rule, commonly in five to six days, but encapsulation is possible. 3. The *thrombophlebitic* (pyæmic) type occurs in 50 per cent. of fatal cases. It usually begins in endometritic ulceration, diphtheria, or ichorous inflammation, in contradistinction to the lymphangitic form of vaginocervical origin Infective thrombophlebitis travels (*a*) from the upper veins by way of the spermatic veins, of which the right empties into the inferior cava and the left into the renal vein; the phlebitis may actually extend into the vena cava; (*b*) from the lower plexus, by way of the uterine veins into the hypogastric (less often the obturator) veins, and thence to the inferior cava. Bilateral venous thrombosis is common (25 to 66 per cent.) Metastases are common; 58 per cent. occur in the lungs, 25 per cent. in the kidneys, 20 per cent. in the joints and muscles, and 16 per cent. in the spleen. Ulcerative endocarditis panophthalmitis, and suppurative peritonitis, may be present. It i unknown why the brain is so often unaffected in this form. Th course may be stormy from predominating toxæmia, or slower with metastasis. The fever is irregular, the pulse rapid, the chill severe, the anæmia marked, and bacteriæmia is present (streptococcæmia in over 90 per cent.).

Diagnosis.—The differentiation from typhoid, malaria, and miliary tuberculosis, is fully considered under typhoid fever. In typhoid, the bacillæmia (85 to 95 per cent.) and the Widal test (95 per cent.) are decisive. In septicæmia, bacteriæmia is present in 50 per cent. of the cases. In malaria, the plasmodium is absent in less than 1 per cent., and quinine is successful at once. In miliary tuberculosis, the choroidal tubercles (possibly confused with Roth's white spots), and tubercle bacilli in the blood are the only positive criteria, though unfortunately they are infrequent. Retinal hemorrhages argue for septicopyæmia. Septic pseudorheumatism rarely needs to be confused with true multiple articular rheumatism. Bacteriæmia, or organisms in the fluid of the joints, is never genuine rheumatism. Uræmia; secondary (terminal) infections in cardiac, arterial, renal, and hepatic disease; mixed infection in tuberculosis; fever in Hodgkin's disease; carcinosis; or sarcomatosis, may sometimes cause confusion.

Prognosis.—The prognosis depends upon the organism, its virulence and dissemination, the patient's physiological resistance (whence the obviously poor outlook in terminal infections), and finally on the surgical accessibility of the primary lesion or metastatic foci. Only 17 per cent. of Lenhartz's cases with bacteriæmia recovered. A stormy onset and chills are ominous. A case of Lenhartz with empyema, lung abscess, panophthalmitis, diffuse venous thrombosis, and multiple osteomyelitis, recovered. Puerperal infections, with the large wound-surface and frequent hemorrhages, are severe, the mortality of all forms being 55 per cent., and of severe forms 65 per cent. (Curschmann). The streptococcus has been regarded with particular dread; however, Bertelsmann (1904) states that the prognosis is twice as favorable as in staphylococcic invasion.

Treatment.—Surgical prophylaxis, even in apparently insignificant wounds, and obstetrical asepsis, are important, especially in subjects reduced by general diseases. Treatment of existing sepsis consists in giving the largest possible amount of food which can be digested; in free administration of water-and-salt solution by rectum or by transfusion, in order to flush the toxins through the kidneys; and in the free use of alcohol, given as it is in typhoid or pneumonia, but in its maximum dosage because the patient exhibits an exceptional tolerance to the drug and unquestionable benefit results. The treatment of hypostasis, diarrhœa, vomiting, and chills, is symptomatic. The temperature is seldom influenced by drugs, though quinine (see PNEUMONIA) may most safely be given. The internal use of germicides is justly abandoned. We have not observed the slightest benefit from Credé's silver ointment, though others think it very beneficial; Credé's collargol has been used with benefit, intravenously, in doses of one to two and a half drams. Nuclein and ʒj to ʒij of fresh brewers' yeast are also advised. The use of Marmoreck's antistreptococcic serum has given no essential reduction in the death-rate, and its suggestive effects in isolated cases is easily misleading. Lenhartz has used a serum from the blood of convalescents from septic cases with some success. Surgical intervention is indicated whenever definite accessible foci are localized.

PNEUMONIA.

Synonyms.—Fibrinous pneumonia, croupous pneumonia, lobar pneumonia, pleuropneumonia, pneumonitis, lung fever.

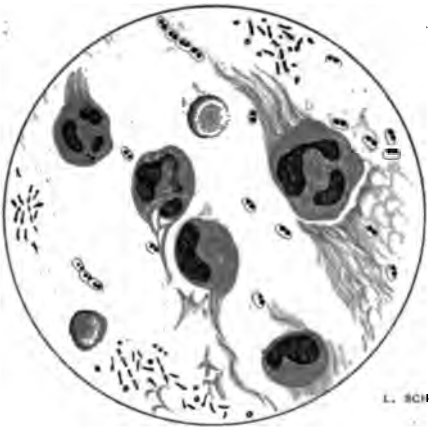
Definition.—An acute infectious disease caused by the pneumococcus (and occasionally by other bacteria), self-limited in its course, characterized by inflammation of the lung (filling of the alveoli, and often of the bronchioles, with a fibrinous exudate), by involvement of considerable areas (lobar pneumonia), by toxæmia, by pneumococcæmia (pneumococci in the blood), and by solution of the fever by crisis. It is not uncommonly complicated by secondary infective processes.

Frequency.—Pneumonia constitutes 6.5 per cent. of all internal diseases, 3 to 4 per cent. of all diseases, 6.6 per cent. of all deaths among medical cases (Liebermeister), and 8 per cent. of all deaths (E. F. Wells).

That it is a *specific infection* is proven (1) by its frequent endemic, epidemic, or even occasional pandemic, prevalence. It may haunt certain dwellings or break out in hospitals, asylums, or prisons; (2) by the great frequency or constancy of the pneumococcus in the sputum, blood, lung, and secondary metastases; (3) by its clinical course, its abrupt onset with general symptoms, followed by local signs, toxæmia and typical ending, and its cyclical self-limited course.

Bacteriology.—1. The *pneumococcus* or Diplococcus pneumoniæ (Plate IV.), was first recognized by Fraenkel (1884) and Weichselbaum (1886), though it was seen earlier by Sternberg, Pasteur, Eberth, Koch, Talamon, Leyden, and Guenther. Fraenkel and Weichselbaum first demonstrated its frequency and importance, and the diplococcus bears their name. It is an elliptical, lanceolate, encapsulated diplococcus; it is easily seen in cover slips, it readily stains by the Gram method, and presents many cultural variations. Its atrium in pneumonia is by way of the respiratory tract, although in sepsis (*q. v.*) it may enter the blood by other avenues. From the lung it may spread, chiefly through the bloodvessels (as it did in Fraenkel's case, where it was traced from a pneumonic focus through a thrombophlebitic pulmonary vein) to the blood and to other viscera. Weichselbaum believes that it may extend by way of the lymphatics. It is found in dust, in saliva, and in 30 per cent. of healthy noses and throats, though usually with attenuated virulence. Again, the organism is virulent, and experimental inoculation has produced pericarditis, endocarditis, and empyema. It has been found in the joints, the blood, the brain, the bone-marrow; in otitis media, endocarditis, peritonitis, cholangitis, and endometritis; in the urine and fæces; and it may pass from the mother to the fœtus. It may be a terminal septic infection or may produce pneumococcus sepsis, as the typhoid bacillus produces typhoid

PLATE IV.



L. SCHMIDT. FEC.

The Diplococcus Pneumoniæ, Stained with Methylene Blue and Fuchsin as a Counterstain. Taken from the Sputum of a Case of Acute Croupous Pneumonia. (Simon.)

bacillæmia. Until very recently it was thought that typhoid and pneumonia were local infections with general symptoms, and that the presence in the blood of their microbes was a serious and a true complication. The typhoid bacillus is found in the blood in 95 per cent. of typhoid cases, and Rosenow, of Chicago, has found the pneumococcus in the blood in about 93 per cent. of pneumonia patients (*v. i.*). Indeed Prochaska last year found the Fraenkel-Weichselbaum organism in all of fifty cases. The pneumococcus is probably the sole cause of typical pneumonia, but other microörganisms are sometimes found.

2. The pneumobacillus (Friedländer, 1883) is 1μ long, one-third as wide as long, with a capsule; it is found sometimes in the healthy nose, is larger than the pneumococcus, and does not stain by Gram's method. It has distinct biological and cultural characters, but its relation to pneumonia is not established. It is probably a mixed infection only.

3. Other bacteria, such as the typhoid, colon, diphtheria, proteus, influenza, plague, and pyogenic organisms, have been found. The existence of psittakosis pneumonia, supposed to be contracted from animals, is denied by Lichtenstern.

Indirect Causes.—(*a*) *Age.*—Pneumonia may be seen even in the new-born. Predisposition to infection is great up to the sixth year, and then much less until the fifteenth year. After the fifteenth year its frequency increases after each decade. Over half the cases of pneumonia occur between the twentieth and fortieth years, and over 80 per cent. between the tenth and fiftieth years. (*b*) *Sex.*—Eighty per cent. of cases are males. The relation to trauma is still in doubt, though contusions seem an occasional accessory factor. (*c*) *Cold.*—Cold and wet are predisposing factors. Pneumonia prevails especially in February, March, and April, when the weather varies, when the temperature is low, when the moisture is great, and when the winds are high. Three-quarters of all cases of pneumonia occur in cold months. It is possible that dust disseminates the dried sputum. Negroes are infected more frequently than whites. Pneumonia is half again as frequent in the cities as it is in the country. In the winter of 1903–4, when 4,000 persons died of pneumonia in Chicago, the author knew of practitioners in many villages and towns not one hundred miles distant, who did not see a single case. (*d*) *Individual predisposition* is said to be a more important factor than in any other acute infection. Susceptibility to pneumonia is increased by debility, overwork, and alcoholism, though the disease also attacks the most robust individuals.

Toxin.—The toxin is not isolated, though it is assumed that a pneumotoxin exists and produces the toxæmic symptoms; when an antitoxin develops, the crisis occurs.

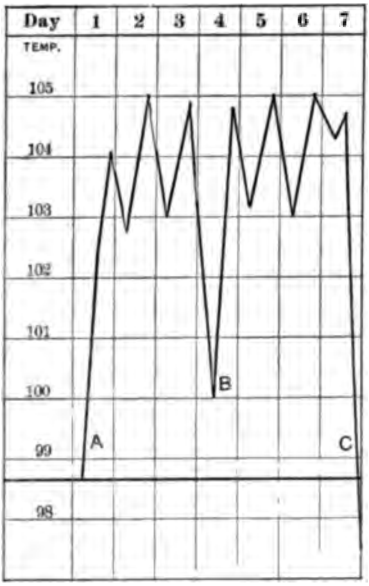
Immunity.—Immunity does not result from one attack; as in erysipelas, recurrence of the disease is likely (in 13 per cent., 33 per cent., even 50 per cent.); ten, sixteen, or even twenty-eight recurrences are known. Though immunity can be produced in animals, it rarely lasts longer than six months (Klemperer, Foa, Washbourne), and it is doubted whether the serum has antitoxic properties. It is thought to possess bactericidal qualities only.

General Clinical Course.—After a very *short incubation* of a few hours to a day, the disease begins suddenly. In children it often begins with convulsions—in the young with vomiting, and in older subjects with a severe chill, which often occurs in apparently perfect health, during work or sleep. On the *first day* there is a sudden rise of temperature; the face becomes red, even livid; the eyes are injected; the pulse and respiration are accelerated; the patient lies characteristically flat in bed; there is much depression; thirst, headache, and a sudden, sharp pain in the side develop, accompanied by cough and increased respiration. On the *second day*, the cough, pain, headache, and respiration-rate, increase, with dilatation of the alæ nasi; the expiration is short and grunting, and speaking is difficult; leukocytosis develops. The sputum appears, or, if already present, becomes bloody; and this, with the sudden fever, chill, pain, and increased breathing, practically establishes the diagnosis before signs of consolidation appear, if pulmonary embolism can be excluded. On the second or *third day* there are evidences of lung consolidation, such as the crepitant rale, bronchial breathing, and tympany which is soon followed by dulness; high temperature develops (with slight morning remissions); the skin becomes dry and red; there is increased pulse and respiration; herpes, anorexia, coated tongue, constipation, moderate delirium, febrile urine, and coughing attacks, develop. The coughing is frequent but short in duration, voids a viscid, red sputum, and causes catching respiration, pain, and broken sleep. The prognosis cannot be made from the symptoms up to this time, since the clinical course may be unfavorably modified (*a*) especially by collapse due to myocardial or vasomotor paralysis, or to embolism of the lung from right-heart dilatation; (*b*) by profound toxæmia; (*c*) by delirium tremens; or (*d*) by death, though at this stage it is rare. In favorable cases, when the toxæmia is spent, the crisis occurs; this usually happens on the uneven days (the fifth, seventh, or ninth) with gradually decreasing dulness and with disappearance of other local findings. A "critical" fall of temperature, pulse, and respiration-rate, and relief of nervous symptoms, and sweating, result. Lysis is less frequent. As a general statement, the toxæmia is the dominant feature of pneumonia, and the consolidation is decidedly secondary. Furthermore, the degree of toxæmia has no necessary or constant relation to the amount of consolidation, just as the toxæmia of typhoid does not depend on the presence or degree of the intestinal changes.

Analysis of Individual Symptoms.—**1. Onset.**—Prodromes occur in 25 per cent. of the cases (Grisolle), mostly in men between thirty and forty years, and rarely last more than five days; anorexia, depression, headache, bronchitis, backache, and low fever, may be noticed. In one of the author's cases a pneumococcus infection began in the throat, then invaded the larynx, and only after three weeks invaded the lung. The organism was obtained at post-mortem. In four cases, low fever and hæmatemesis for five to seven days suggested tuberculosis. In the two coming to autopsy, no tuberculous foci were found. In 75 per cent., however, the onset of pneumonia is sudden. *Chill*, convulsions, vomiting, and fever, after a short incubation, usually mark the brusque onset. Repeated chilly sensations, occurring when the heart is weak, do not as accurately designate the onset as does an initial rigor. A chill is present in 75 per cent. of the cases, and in no other acute disease except malaria is the chill so severe and constant; it lasts from a quarter to half an hour. The onset is atypical in senile pneumonia, and also when it occurs in the course of other diseases,—*e.g.*, delirium tremens, typhoid, variola, and psychoses,—and the chill, cough, dyspnœa, pain, and sputum, are often absent, so that the existence of pneumonia may not be suspected.

2. **Fever.**—The usual course of the fever is, first, a very sudden rise, especially in menstruating women (von Jürgensen); the fever is then continuous, often with not more than 1° or 2° variation for a few days; in three quarters of the cases the maximum fever is between 102° and 105°. It may then remit, which is of good prognostic import; remittent fever throughout the disease occurs in some cases. Recrudescences occur when consolidation passes from one lobe to another. The fever is lower or even absent in the aged, in alcoholics, and in cases of secondary pneumonia. Fever produces a marked loss of weight (twenty to thirty pounds). The rise preceding the crisis (*perturbatio critica*) may be mistaken for an ante-mortem rise. In

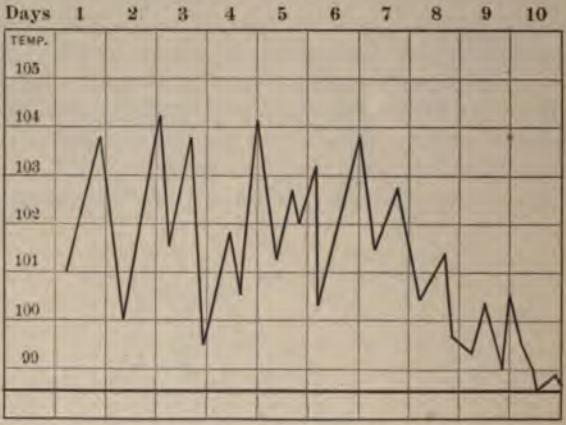
FIG. 13.



Fever-curve of typical case of pneumonia: A, sudden rise; B, pseudocrisis; C, crisis.

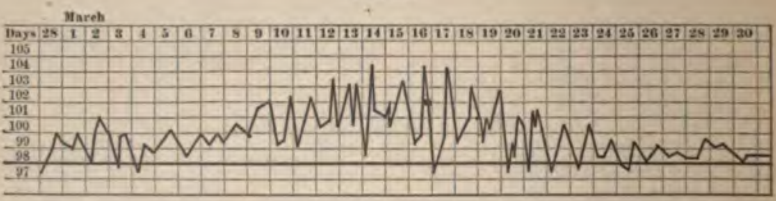
both conditions there may be delirium, convulsions, chill, or irregular pulse. A pseudocrisis may deceive the uninitiated physician, when the fever resumes its original elevation. Crisis occurs in 60 to 80 per cent. of cases, usually on uneven days, as noted by Hippocrates,—the fifth, seventh, ninth, or eleventh days,—and usually between evening and early morning. It very rarely occurs on or before the third day. It marks the end of the toxæmia but not of the consolidation. This requires several days more. In six to ten hours the temperature falls to (or below) normal, accompanied by sleep, euphoria, lessened pulse and respiration, lateritious deposits, and reappearance of the chlorides in the more abundant, limpid urine, and "critical" sweating, or even diarrhœa. The fall may measure even 10° or 11° and the pulse may reach 30. The precritical elevation and the beginning of the crisis may be attended by alarming symptoms, such as weak, irregular pulse and collapse. Lysis occurs in 20 per cent. of the cases. It is likely to occur when the fever lasts more than ten days, but lingering fever may indicate progression in other lobes and such complications as empyema or endocarditis. Lysis may be (1) gradual and continuous, or (2) slow and remittent.

Fig. 14.



Pneumonia; remittent (subcontinuous) fever.

Fig. 15.



Pneumonia fever-curve: exceptionally protracted though uncomplicated fever.

3. **Respiratory Findings.**—(*A*). PAIN.—Pain in the side is pleural, though sometimes muscular. Its presence in 90 per cent. of the cases makes it of diagnostic value, since it is the most frequent respiratory symptom. It appears early, is increased by coughing or motion, and is sharp and sometimes agonizing. It is usually well localized over the consolidated focus, but in 8 per cent. may be referred along the lower intercostal nerves to the abdomen, in children especially, and sometimes in adults. Appendicitis is thus simulated in 2 per cent. Sometimes the pain is located over the gall-bladder, or on the opposite side of the chest. Pain is absent in central, secondary, or upper-lobe pneumonias; in the aged, and often in children; and in cases with great delirium.

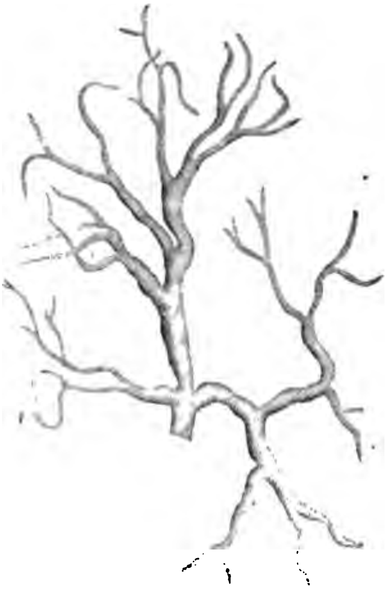
(*B*). DYSPNŒA.—Dyspnœa is frequent; it is caused by pain and especially toxæmia, but also by temperature, cyanosis, diminished respiratory area, bronchitis, and complicating pulmonary œdema, and scoliosis.

(*C*). THE RESPIRATION-RATE.—The respiration-rate is practically always increased to 24 or 40, (or more in children); and in two-thirds of all cases, it ranges between 30 and 50; it almost never rises to 60 or 80 except in the death agony, but such frequency is common in miliary tuberculosis. The author once saw a rate of 96 with recovery; the pulse was 90. Dilatation of the nares is said to be ominous, but seems to the author common in ordinary cases. Alteration of the pulse and respiration ratio was first noted by von Jürgensen; the normal ratio of 1 to 4 or 4.5 becomes 1 to 3 or 2. On very rare occasions the respiration-rate may be normal. Thus, in one instance with the entire right lung consolidated, respiration was 18 (without medication).

(*D*). THE COUGH.—The cough always irritates, is sometimes dangerous, and seldom relieves the patient. It is usually present from the beginning of the disease to the crisis, and almost invariably occurs in lower-lobe pneumonia, but is often absent in upper-lobe consolidation, in terminal or secondary pneumonia, in cases where there is much delirium, in the aged, and the young. It is sometimes pleuritic.

(*E*). THE SPUTUM.—The sputum is at first transparent, mucous, and very scanty. It soon becomes viscid, and is difficult to raise, and to expectorate, as it adheres to the mouth and lips. It is so viscid that the sputum-cup may be inverted without any flow of the sputum. In two days it becomes red, and rusty (80 per cent. of cases). Later, the sputum becomes more abundant, averaging between one and seven ounces daily, but is rarely very abundant or fluid. Increased fluidity may result from weak heart, or decreased coagulability of the blood. Rusty sputum is often absent in children who swallow their sputum; in cases of upper-lobe consolidation, in which there is less hemorrhage, less consolidation, and less cough to dislodge exudation; and, finally, it is often absent in the senile and secondary pneumonias, possibly because of very early gray hepatization. The cause of unusual color in the sputum, such as lemon- or brick-color, is unknown; it is sometimes observed in pulmonary œdema, or in incipient gangrene, and renders the prognosis uncertain. Safranin color (*sputum croceum*) indicates resolution. The rusty sputum is of great diagnostic value,—*e.g.*, in the beginning of the disease or in central pneumonia—and generally is of good import. Fibrin threads (or "casts") of the bronchioles are usually seen between the third and seventh days. On shaking the sputum in water and catching the shreds on a slide they appear clubbed at their alveolar ends. They are almost, but not wholly, pathognomonic, since Laennec found them in tuberculosis, and in fibrinous bronchitis. They consist of fibrin, white cells, fatty cells, and epithelium. Thinner casts, analogous to the cylindroids of the urine, and spirals, are sometimes found. The sputum is rich in chlorides, especially in calcium chloride. Microscopically the sputum contains blood cells, various epithelia, the pneumococci (found first by Wolff in sputum and by Weichselbaum in the lung), and often streptococci.

FIG. 16.



Fibrinous coagulum from a case of croupous pneumonia. (Bizzozero.)

(*F*). LUNG PATHOLOGY.—The lung pathology was developed in 1819, by Laennec, who was the first to accurately separate pneumonia from pleurisy. He described three stages. (1) The stage of *hyperæmia* is very exceptionally observed unless the subject dies in twenty-four to thirty-six hours, because hepatization (*v. i.*) develops rapidly; hyperæmia is most often seen at the edges of the consolidated lung where the pneumonia is beginning to extend. The lung crepitates and floats, though less than normal; it is red, voluminous, and tears readily,—[*i.e.*, it has lost its elasticity], and exudes a serosanguineous fluid on section. Under the microscope, congestion, serum, and diplococci in the alveoli, are observed. The alveolar epithelial cells swell and desquamate, which, according to some observers, are the primary changes. (2) In the stage of *red hepatization*, the lung is evenly red, friable, airless (not wholly so in pneumonia of an upper lobe), and sinks when put into water. Its weight is increased ten to fifty ounces. The lung is enlarged to the size of deepest inspiration (Rokitansky, who developed the gross anatomy of pneumonia), and has marks of the ribs on its fibrin-coated surface. On scraping the cut surface, fibrinous casts or plugs of the bronchioles are observed. The lung is granular on section (Laennec), which results from exudation into the alveoli. These granulations are smaller in the young and are larger in patients with emphysema. They are seen best with oblique illumination. Under the microscope, coagulated blood and exudate, alveolar epithelium, injected bloodvessels, emigration of leukocytes, diplococci, and meshes of fibrin, are noted. (3) In the stage of *gray hepatization*, the granulations are grayish-yellow, and are not red because the blood is absorbed; they are larger and looser than in the second stage. The connective tissue is grayer than the granulations, and is sharply defined by the lung pigment. It has been claimed that death always occurs if this stage is reached. If the patient lives, the granulations disappear; the lung exudate becomes semi-fluid (purulent infiltration); the pus can be washed out; there is a fatty emulsion, and resorption occurs by the lymphatic vessels and not by evacuation through the sputum. Enzymes (ferments) dissolve the exudate; the products of this autolysis are excreted by the kidneys.

Seat of pneumonia: The lobar process begins at the hilum and extends to the pleura, which is usually inflamed. If it remains at the hilum it is called central pneumonia (1.7 per cent.); if the entire lung is involved, it is termed pneumonia totalis or massiva; the terms "double pneumonia" and "crossed pneumonia" are self-explanatory. The lobes are involved in the following order of frequency: (1) Right lower lobe; (2) left lower; (3) right middle; (4) right upper; (5) left upper lobe. Jürgensen's collation of 16,614 cases of pneumonia shows that the right lung is affected in 53 per cent., the left in 37 per cent., and both lungs in 10 per cent. Fraenkel found the upper lobes involved in 16 per cent. and the lower in 84 per cent.

(G). PHYSICAL SIGNS.—The physical signs develop in the lungs only after twelve to twenty-four hours, or later.

1. *Inspection.*—The decubitus is variable. It is dorsal in lower-lobe, and erect in upper-lobe pneumonia; the patient at first lies on the sound side to avoid pain; on the diseased side later, for better expansion of the sound lung. The spinal column is concave toward the diseased lung for its better immobilization. The excursion is less on the diseased side because of pain. Litten's diaphragmatic sign is absent and the sound lung is vicariously active. The diseased side

is larger by one-half to one inch. The accessory muscles of respiration are active and dilatation of the nares is seen. Systolic pulsation of the lung occurs when the lingual lobe is consolidated, or, in the first stage, when the heart beats against semifluid exudation (Graves).

II. *Palpation.*—This detects the lessened excursion more readily than does inspection; vocal fremitus is usually increased because of better sound propagation, fremitus being normally greater on the right than on the left side. It is decreased (1) by occlusion of the bronchus by fibrinous plugs, which are, however, usually dislodged by having the patient cough; (2) by coincident pleural effusion; (3) by great consolidation (Gerhardt), in which the chest wall becomes tense. It is claimed that the consolidated side is the warmer. Pleural friction may be felt.

III. *Percussion.*—(1) A tympanitic note is obtained in the first stage (Skoda's resonance). This is caused by decreased elasticity of the lung and therefore decreased tension and amplitude of vibration, which is due to the swelling of the alveolar epithelium and turgescent capillaries. This condition may last as long as two days, and causes doubt as to the side involved, especially when pain is bilateral and breathing indeterminate (whence the value of crepitant rales). Tympany in the later stages is not uncommon in upper-lobe pneumonia where exudation is usually incomplete. It is rare in lower-lobe pneumonias but may occur over an upper lobe in total pneumonia from the air in the bronchus. It may be attributed to the stomach (Finckler) in cases of left lower-lobe pneumonia; to some moderate compression,—*e. g.*, of right upper lobe when right middle and lower are consolidated; to the normal lung lying over deeper consolidation or to resolution. Tympany may remain days or weeks after the crisis. Careful watching of the tympany, during the acute stage, often leads to detection of extension, which is shown by relaxation of the lung tissue, contiguous to areas already consolidated. (2) *Dulness* is usually found on the second day. It is the percussion sign of the second stage. When complete, it corresponds to the lobe confines, which are outlined in Fig. 33 under article, Pleurisy. The dulness is not absolute. It is usually first detected in the posterior axillary line in lower-lobe pneumonia and generally is more complete behind than in the front of the chest and more in a lower- than in an upper-lobe involvement. Dulness is difficult to detect if the area is not at least 1 by 2 inches, or if it is deeper than 2 inches from the surface. It is absent in central pneumonia. It disappears with resolution. Wintrich's palpatory percussion is most valuable. Wintrich's change of pitch (on opening the mouth) is heard in upper-lobe pneumonia, where the percussion wave is conveyed to the bronchus more often than in lower pneumonia. Cracked-pot resonance over the edge of the consolidation bordering on sound tissue is due to irregular tension.

IV. *Auscultation.*—Decreased breathing is often noticed at first (Grisolle). Stokes diagnosticated the first stage by sudden local puerile breathing with fever and by disordered breathing.

First Stage.—The *crepitant rale* of Laennec remains the auscultatory sign of the first stage; but, as Laennec stated, it is not absolutely specific, because it occurs also in tuberculosis, œdema, infarct, hypostasis, and bronchiolitis. It probably has its origin in the viscid exudate in the finest tubules and in the alveoli. Some consider it pleural. It is coarser in the aged and in children because the large tubes are more involved. It is heard at the height of inspiration as a succession or shower of delicate crackles. When not heard, a, coughing effort may elicit the crepitant rale. It often disappears after a number of deep inspirations; *e. g.*, when it is demonstrated to a class, only the first students hear it. Dislodged plugs may occasion expiratory rales. The crepitant rale disappears during the second, and reappears in the third stage.

Second Stage.—The distinct, intense *bronchial breathing* is due to better transmission of the bronchial sounds through the solidified lung. It sounds as though nothing intervened between the lung and the stethoscope and it is especially clear and near when the ear is placed directly on the chest. It is absent or partial in central pneumonia, especially anteriorly, in coincident pleurisy, and in obstruction of the bronchi by mucous or fibrinous casts. In the latter case, several coughs usually loosen the obstruction and restore the bronchial breathing. *Bronchophony,* due to the voice sounds being better transmitted through the consolidated area, is heard when the patient counts or speaks, but especially after each involuntary expiratory grunt. *Ægophony* is occasionally heard, as well as Baccelli's phenomenon (auscultatory whispering). The crepitant rale disappears in this stage, except at the edges of the hepatization where extension may be taking place.

Third Stage.—The crepitant rale reappears and is then known as the rale redux.

4. **Circulation.**—(*a*) The *pulse* rate is usullay 100 to 120; in children it may rise to 200. It is also rapid in the aged, and in cases with severe toxæmia. In adults, a rate of 130 to 140 makes the prognosis less favorable. If the rate is slow at the height of the disease the prognosis is grave, for heart or brain complications are indicated (Traube). Irregularity and gallop-rhythm (one systolic tone and two diastolic tones) are of poor prognostic import. Antecritical irregularity frequently occurs. The pulse is usually deceptive, because its fulness is of no value in prognosis. It is not often dicrotic. Extensive pneumonias may compress the subclavian artery, making the pulse smaller in one wrist than in the other. In the right *heart,* dilatation from toxæmia may occur; the second pulmonic tone is accentuated but this is not observed when the right heart becomes greatly weakened. There are functional bruits especially in children. In very rapid cardiac action, the two heart tones sound alike, resembling the fœtal heart tones (embryocardia). (*b*) *Collapse* is usually due to myocardial or to central vasomotor paralysis and may occur at the onset, acme, defervescence, or convalescence. (*c*) *The blood* shows anæmia,

and Bollinger suggests that the oligæmia, resulting from large exudates, may explain some cases of collapse. The fibrin increase is readily noted in fresh blood preparations. A polymorphonuclear *leukocytosis* of 15,000 to 30,000 (or even 100,000) appears early, and lasts slightly longer than the fever. In Cabot's counts, the leukocytes ran between 15,000 and 30,000 in 75 per cent. of his cases. The eosinophiles are decreased or wholly absent until the affection reaches its height, whence the favorable significance of their reappearance. The lymphocytes are decreased. During convalescence many myelocytes appear. The white count remains high in delayed resolution or with complications. A low count is generally unfavorable. *Pneumohæmia* (pneumococci in the blood) was, until recently, considered of bad prognostci import and it was once stated that but 15 per cent. of cases of pneumohæmia recover, but now it is not considered of great prognostic value. Rosenow found pneumococci in blood cultures in 93 per cent., and in smears in 42 per cent. They were most abundant on the fourth or fifth days, and few in number before or at the crisis. In making cultures, half a dram of blood is taken and mixed with 98 or 99 parts of bouillon. Prochaska found pneumococci in 100 per cent., when two or three drams (10 c.c.) of blood were taken for cultivation; they are probably present in all cases, though some think that they appear only when the protective resistance of the tissues is lost and death is imminent; pneumohæmia has been found before hepatization. Rosenow noted increased agglutinative power of the serum until the crisis occurs.

5. The Digestive System.—There is the febrile tongue, thirst, and anorexia. Vomiting results from cerebral toxæmia (especially in children), vagus irritation, coughing, or alcoholic pharyngitis. The distressing, sometimes dangerous, tympany and the occasional diarrhœa are usually toxæmic. Constipation is the rule. There is often some intumescence of the liver. Fraenkel found splenic tumor in 15 per cent. of all cases and in 40 per cent. of fatal cases.

6. The Skin.—The red cheek on the affected side was known to Aretæus. The skin is usually dry, but miliaria and sweating may be seen, especially in children. Herpes is found more often than in any other disease (30 to 50 per cent.) It appears from the third to fifth day or later, chiefly on the lips, nose, mouth, cheek, ear, or rarely on the anus or genitalia; Howard found causal changes in the Gasserian ganglion. Herpes is of diagnostic value, *e. g.*, from typhoid, and is generally of favorable import. Rarer eruptions are erythema, roseolæ, urticaria, purpura, or pemphigus. The lymph-glands are enlarged in 25 per cent. of the cases.

7. The Urine.—Characteristic fever urine is found, and the urea is increased threefold, especially after the crisis from over production and accumulation during the fever and from the resolving exudate. The uric acid, ammonium, and creatin, are increased. The chlorides are decreased from 225 grains daily to 30 grains, or they may be absent until the crisis or two days later, when they reappear in increased quantities (first observed by Rottenbacher); no satisfactory explanation for the decrease of the chlorides is known. The sulphates and phosphates are decreased. Pick notes that the urine often becomes neutral or alkaline after twenty-four to forty-eight hours; this is due to sodium deposit in the lungs, which condition lasts from twenty-four to thirty-six hours. Albuminuria and cylindruria appear in about 50 per cent. of the cases; peptonuria and acetonuria are not uncommon, and the diazo reaction appears in 20 per cent. Hæmaturia is found in 1.6 per cent. Nephritis seldom occurs (1 to 2 per cent.), and uræmia is very uncommon. Pneumococci have been found in the urine and in the glomeruli (Koch, Nauwerck).

8. **The Nervous System.**—Headache is almost constant. There may be insomnia, restlessness, or sopor, and the latter is always ominous. Delirium (22 per cent.) occurs in adults, especially in cases of upper-lobe pneumonia, alcoholism, or marked toxæmia. Delirium and other nervous symptoms resulting from exhaustion may occur at, or even for a week after, the crisis. Venesection in former times greatly increased the percentage of delirium. The patellar reflexes are often absent and the pupils may be dilated or sluggish. Convulsions, coma, and rigid neck, in children, result from toxæmia of the more sensitive young brain, from complicating otitis, from brain œdema, or from actual meningitis. Hemiplegia may result from (1) brain œdema or anæmia, or (2) arteriosclerosis and weakened heart.

Mechanism of Death in Pneumonia.—Death is rare in the first two days. If it occurs within this time, its cause is usually some antecedent malady such as nephritis or typhoid. (1) *Toxæmia* is all-important in its action on the heart, brain, and vasomotor nerves. Death results with a rapid pulse, rapid respirations, algid extremities, and obtunded sensorium. (2) The *extent of pneumonia* is the least important factor. A massive pneumonia in a robust person causes less disturbance than a very small patch in a nephritic patient. There is no parallelism between the area hepatized and the resulting toxæmia. (3) *Pulmonary œdema* is either mechanical (cardiac) or inflammatory—*i. e.*, the first stages of an incompletely developed pneumonia. (4) *Pneumococci in other tissues, e. g.*, pneumococcic meningitis, endocarditis, and pericarditis.

Complications.—These are fortunately few as compared with those of typhoid but they are severe.

1. **Pleurisy.**—Pleurisy is so constant that the name of pleuro-pneumonia is rather justified, the pleura being covered with a very thick, tenacious, curdy exudate. It is usually due to the pneumococcus; if due to the streptococcus, it is a complication of great danger. Effusive pleurisy is said to occur in 5 to 15 per cent., though Fraenkel saw serofibrinous pleurisy in but 1 per cent. in 1,000 cases. Pleuritis is para- or metapneumonic, serofibrinous (5 per cent.) or purulent (2 per cent.), febrile or afebrile. The presence of leukocytosis in a pleurisy may indicate a previous unobserved or overlooked pneumonia. Pneumonia may exist on one side and pleurisy on the other, —*e. g.*, an upper-lobe pneumonia with contralateral pleurisy (Traube).

The author has seen two such cases. The prognosis is not necessarily unfavorable, even when operation for pneumococcic empyema is refused. Interlobular empyema is most serious, because it so often escapes detection. Pneumococcic empyema occurs in 2 per cent. of cases of pneumonia and is notable since the pneumococcus so seldom provokes suppuration in the lung. Its symptoms are discussed under pleurisy.

2. **Endocarditis.**—Acute endocarditis complicates pneumonia in 1 per cent. (Chvostek) of all cases and in about 5 per cent. of fatal cases, though higher figures are given, 14 per cent. (Kanthack) and 16 per cent. (Osler); all autopsy figures are usually higher than figures in general practice. The pneumococcus is found in the vegetations. The valvular exudate is voluminous, though ulceration, valvular aneurysm and perforation are less common than in staphylo- and streptococcic forms. Endocarditis develops especially on old valvular lesions in the left heart and most frequently on the aortic valves (Netter, Heschl). The right heart is involved three or four times as frequently as in endocarditis due to other bacteria. Sixty per cent. of cases are associated with meningitis. Middle-aged adults are more often affected than the young, and women more often than men. Symptoms may be absent. Endocarditis is probable when a murmur, systolic or particularly diastolic, occurs with irregular and protracted fever, chills, sweats and embolism (50 per cent.). Symptoms may directly follow the pneumonia or develop after one-half to one week of normal temperature. The endocarditis is malignant in 80 per cent. of cases; in the balance, milder endocarditis occurs; 25 per cent. of cases of ulcerative endocarditis are pneumococcic. Fever is sometimes, and leukocytosis frequently, absent. (*Vide* chapters on ULCERATIVE ENDOCARDITIS and SEPTICOPYÆMIA.)

3. **Pericarditis.**—This is dangerous though not necessarily fatal. Some figures place its post-mortem frequency at 5 to 15 per cent. but Fraenkel estimates it at seven-tenths of 1 per cent. About 60 per cent. of cases are latent because the pericardial rub is frequently absent. Pericarditis occurs more often in children than in adults and, in all cases, chiefly at the height of the disease. Effusive pericarditis is less common than the fibrinous form. It is more common when the left lung is involved.

4. **Arteritis** and **thrombophlebitis** are far less frequent than in typhoid, but nearly every clinician encounters a few cases. Steiner collected forty-eight cases of venous thrombosis (1903); it most often develops in convalescence and usually (75 per cent.) heals, though early embolism or permanent venous obstruction may develop.

5. **Nervous Complications.**—The chief nervous complications are delirium tremens, meningitis, or, less frequently, Landry's ascending paralysis, cerebral thrombosis, embolism, encephalitis, and neuritis. *Meningitis*, first observed by Immermann and Heller, varies in frequency with the epidemic; it occurs in 5 per cent. of autopsies, and in three-tenths of 1 per cent. of all clinical cases. It occurs in asthenic

patients, in upper-lobe pneumonia, and is often accompanied by endocarditis. Symptoms are often wholly lacking. One of the author's cases died at the dinner-table a week after apparently complete convalescence, and the autopsy showed a quarter inch of pus over the convexity. The usual meningitic symptoms develop if infection reaches the base of the brain. This, however, is not usual. Lumbar puncture may determine the diagnosis, which is more easily made if meningitis occurs in convalescence. The prognosis is considered absolutely unfavorable, yet Achard and the author have each seen recovery in cases which were proven bacteriologically by lumbar puncture.

6. **Alimentary Complications.**—These are few. Croupous gastritis and enteritis, parotitis or peritonitis (*q. v.*), are not frequent. Dieulafoy described a hemorrhagic ulcerative gastritis, caused by pneumococcic embolism and attended by gastrorrhagia. Icterus is frequent in some epidemics. Its average frequency is about 1 per cent., though Chvostek saw it in 21 per cent. of his cases. Its causes are variable; (*a*) lessened excursion of the diaphragm in right lower-lobe pneumonia; (*b*) hepatic stasis; (*c*) duodenal catarrh; (*d*) hæmatogenous jaundice resulting from absorption of the hemorrhagic exudate in the lung or toxæmic solution of the red blood-cells (hæmolysis). Pneumococcic involvement of the peritoneum will be described under PERITONITIS.

7. **Lung Complications.**—Suppurative mediastinitis was first observed by Weichselbaum, who regards it as the connecting link between pneumonia, meningitis, and pericarditis. Pulmonary induration (cirrhosis of the lung) may follow incomplete resolution, especially after repeated attacks of pneumonia (Charcot); it is usually febrile. Abscess is not frequent (5 per cent., Laennec, or once in 1,200 cases, Fraenkel). Skoda emphasized the uncertainty of its signs and the difficulty in its diagnosis, for pus and elastic fibers in the sputum, and less frequently cholesterin and hæmatoidin crystals may be the sole sign, and continual coughing may be the chief symptom. Suppuration may result from the pneumococcus or from the pyogenic bacteria proper. *Gangrene* occurs in 1 to 2 per cent. of the cases. Clinically it is recognized by the horribly fetid sputum. Abscess and gangrene are more fully treated under DISEASES OF THE LUNGS.

8. **Complications in the Joints.**—Genuine rheumatism may occur with, before, or after a pneumonia. Sixty-eight cases of pneumococcic arthritis (pseudorheumatism) were reported by Slaughter (1903). The arthritis may be serous or purulent. The knee is affected in 30 per cent. Arthritis is monarticular in 62 per cent. and fatal in 65 per cent. of the cases (Herrick). Fraenkel found the pneumococcus constantly in the bone-marrow.

Clinical Types and Variations.—These are as follows:

1. **Abortive Pneumonia.**—This type is recognized by the rusty sputum, fever and respiratory disturbance, but produces no hepatization.

2. **Ephemeral Pneumonia.**—This type is characterized by one day of fever, and very rapid resolution.

3. **Relapsing Type.**—One attack occurs with fall of temperature, and then the entire cycle recurs. It occurs in 0.3 per cent. (Wagner). The recurrent type (*v. s.* IMMUNITY).

4. **Local Variations.**—Apex pneumonia is often more severe and more atypical in symptoms and signs; it is frequently adynamic, and pulmonary and cerebral complications are rather more common. Migratory pneumonia travels from one part to another (the so-called erysipelas-pneumonia). Central and massive pneumonia (pneumonia totalis, affecting an entire side) will be considered under diagnosis.

5. **In Children.**—An abrupt onset occurs without chill (before the seventh year) but with vomiting (50 per cent.) or convulsions (7 per cent.) and other cerebral or meningeal symptoms. Cough is often absent, no sputum appears, and the pain (third to fifth year) is referred to the abdomen but without tenderness there. Pain is a more important symptom than increased respiration, which occurs with any elevation of temperature. The respirations may number eighty in cases under two years of age; in older children, fifty. Crepitant rales are usually absent because of the superficial breathing caused by pain. Latency of physical signs, such as absence of bronchial breathing, bronchophony, or dulness, is sometimes noted. Localization is more often central, apical, or migratory. The fever may rise more slowly and lysis is more frequent than in adults. Rivière found croupous pneumonia as frequently (or more frequently) in children under two years as in those above two years. In 118 cases reported by Morse, 50 of them occurred in the first, and 68 in the second year of life.

6. **In the Aged.**—Pneumonia is frequently latent, insidious, and asthenic, with ill-marked signs, without chill, even without fever, and often without cough, pain, or sputum.

7. **In Alcoholics.**—The same latency or lack of type may be noted. In alcoholics the clinical picture of delirium tremens masks the lung lesion.

8. **Asthenic Pneumonia.**—In asthenic "typhoid," "biliary," or "toxic" pneumonia, toxæmia is dominant, there are high fever, enlarged spleen, albuminuria, often icterus, mixed infections, severe complications, such as meningitis or gangrene, and high mortality. These asthenic cases contrast strongly with what we might call "walking pneumonia." The author has had two patients who worked throughout the clinical course.

9. **Pneumonia Secondary to Other Diseases.**—Pneumonia may be secondary to tuberculosis, emphysema, arteriosclerosis, hepatic cirrhosis, nephritis, diabetes, typhoid, influenza, diphtheria, and plague. Anæsthesia by ether is thought to predispose toward pneumonia by irritation of the respiratory tract. The nose and mouth should be cleansed before anæsthetization to remove native germs. The frequency of ether pneumonia in abdominal operations suggests the possibility of embolism as a factor.

Various microörganisms are found—colon bacillus, streptococcus, etc. The physician may often fail to recognize this type because it is atypical, ill developed, or masked by the primary disease. Pathologically, the hepatization is rarely typical and is more lobular than lobar; the lung section is smooth and moist, and the exudation is more cellular than fibrinous.

10. **Delayed or Imperfect Resolution.**—Normal resolution is chiefly effected by lymphatic absorption, for the sputum voids but little of the exudate. (*a*) Slow resolution may require several weeks or even months, especially in apical involvement, and is more often seen after lysis than crisis. The toxæmia disappears but dulness and bronchial breathing persist. (*b*) Imperfect clearing up of the lung may be caused by abscess formation, old pleuritic adhesions, fresh pericarditis, or inflammatory involvement of the connective tissue and lymphatic vessels. Induration of the lung by organization of the exudate is usually febrile (Fraenkel). (*c*) The so-called pneumonia migrans is a pneumonia which slowly progresses from one part to another.

Diagnosis.—Mistakes are rare in the diagnosis of primary, frank pneumonia in adults—they occur mostly in cases of aged individuals, of alcoholics, of children, and in cases of pneumonia secondary to cancer, nephritis, diabetes, etc. Therefore, all cases with chronic disease must be closely examined when the respiration, pulse, and temperature rise, or when, even without these or other symptoms, the subject of chronic disease is not doing well.

1. **Onset Symptoms.**—The chill in adults, convulsions in children, which occur also in such other diseases as nephritis, infantile palsy, and alimentary catarrh, vomiting in the very young or in adolescents, suggest the possible onset of lobar pneumonia. Chill, pain in the side, sudden high fever, and rusty sputum, are almost sure symptoms, because pneumonia is practically the only disease in which they occur. Care must be taken to avoid the chief diagnostic confusion—infarct of the lung—in which the same symptoms may obtain, but in which the sputum is reddish, is not viscid, is usually darker, and far more abundant than in pneumonia. In 25 per cent. of cases the onset of pneumonia is not typically brusque, but the physical findings of pneumonia are present without a history suggestive of the disease; indeed it may rather be suggestive of simple pleurisy, grippe, etc. The profession regards pneumonia as a disease with a severe onset, but the laity, and with reason, emphasize the importance of antecedent exposure, colds, or bronchitis. The prodromes of pneumonia are too often disregarded by physicians, even when they carefully exclude pneumonia which is strictly secondary to other acute or chronic disease. In a very appreciable percentage of cases the prodromata are not brief and may last longer than two or three days, or even a week.

2. **Diagnosis from the Physical Findings.**—The physical findings are usually detected in the typical case upon thorough examination, and as a rule usually appear within two days after the characteristic symptoms of onset. They are the well known lessened expansion on the side of the lesion; increased vocal fremitus; distinct dulness; bron-

chial breathing; the crepitant rale; the grunting, abbreviated, expiratory "huh" sound, and a bronchial tone to the whispered or louder voice sounds. Typical as are these simple findings in their usual grouping, and easily recognized as they are when the student or practitioner has once heard and learned them, nevertheless many conditions modify their classical appearance and character. Thus, the fremitus is decreased in massive consolidations which render tense the chest wall, in coincident pleural effusions or in fibrinous plugging of the bronchi. The latter may, however, usually be dislodged by repeated coughing. The dulness is found only in hepatization which is nearly complete and is both preceded and followed by a tympanitic note. The crepitant rale is found only in the first and third stages, but in almost every case can be found in some part of the affected lung. Bronchial breathing is rudimentary or absent in incomplete hepatization, is often so in upper-lobe pneumonias, in complicating effusive pleurisy, and in obstructed bronchi. These physical symptoms are pathognomonic only in connection with the cyclic course of the disease, the symptoms of onset, the disturbed pulse and respiration ratio, the pneumohæmia, diminished chlorides, herpes, leukocytosis and crisis.

3. Localization.—Massive pneumonia (total pneumonia of one side) not infrequently simulates pleurisy, because fibrinous plugging of the bronchi is not uncommon, and prevents bronchial breathing, bronchophony, and the crepitant rale; and again, the fremitus may be absent together with some slight, although rarely essential, luxation of neighboring viscera. Consideration of the onset, the later cyclic course, and the dislodging of the plugs by forcible coughing, almost invariably determine the diagnosis. Too much is generally said about central pneumonia. Such cases exist beyond doubt, but considerable conservatism is necessary in their diagnosis. All pneumonias are central in their incipiency but rarely remain so. The author lately demonstrated a case in clinic in which everything pointed to pneumonia, but the local findings did not appear for a week, although they were carefully searched for twice a day.

4. In Children.—Pneumonia in children may be mistaken for abdominal disease, for pleurisy, or for brain affections. The "cerebral pneumonia" of Rilliet and Barthez may be (*a*) convulsive, occurring under two years, with fever, vomiting, and convulsions; but the evenly high fever is very suggestive even if the cough and dyspnœa are obscured by the brain symptoms; or (*b*) meningeal, occurring between the second and sixth years; but the pulse is not slow, and there is no deep breathing, nor change of color in the cheeks, as is observed in meningitis.

5. Differentiation from Pleurisy.—The differentiation is usually easy, but confusion is unfortunately common.

The history may be suggestive but it can be indefinite, especially in hospital cases.

PNEUMONIA——*vs.*——PLEURITIC EFFUSION.

Onset sudden, with chill, vomiting, herpes.	*Gradual*; chilly sensations rather than rigor.
Friction less common; may be present.	*Usually*, at some time in the clinical course.
Cyclical short course; high fever, crisis.	*Longer* duration, lower fever, lysis. Often tuberculous foci at apex, etc.
Sputum rusty.	*Mucous*, if any.
Leukocytosis.	Usually *absent*, unless purulent or complicating a pneumonia.
Edema absent.	*Present.*
Fremitus increased (coughing usually dislodging bronchial plugs).	*Absent* over effusion unless irregular adhesions bind the lung in places to the chest wall; increased above overcompressed lung or in the interscapular region.
Dulness, preceded and succeeded by tympany (tympany or partial dulness in upper-lobe pneumonia is not uncommon).	*Flatness*, increased downward; entering Traube's space on the left side, unless adhesions forbid. Level may occasionally shift with change of position. (See Fig. 33 under PLEURISY).
Crepitant rales, bronchial breathing (unless bronchus is plugged), and bronchophony.	*Decreased or absent respiratory murmur;* we may have bronchial breathing in interscapular region from compression, or even through the fluid, both in adults and children; ægophony.
No essential luxation of heart, liver or spleen (except the slightest in pneumonia massiva).	*Luxation* of viscera named, is usual but not invariable, even in large effusions.
Paracentesis negative.	Tapping nearly always decides doubtful cases; *fluid is found* over the flat area.

In *lung tumors*, bronchial breathing, bronchophony, dulness, red (or green) sputum, are also noted, but the history suffices for differentiation. In *acute tuberculous infiltration*, the course differentiates it from upper-lobe pneumonia by the gradual onset, in which cough sputum, and typical physical findings are absent. Many mistakes in both directions are made, simply by forgetting that pneumonia may occur in the apices, in which the incomplete exudation may impart a tympanitic note, and that the breath sounds are not always typically bronchial, at least anteriorly. In some cases tuberculous pneumonia may commence like lobar pneumonia (see ACUTE PNEUMONIC FORM, under COURSE OF PULMONARY TUBERCULOSIS).

Prognosis.—Pneumonia is the most fatal acute disease and even has a greater death-rate than tuberculosis. In 1903, 4,629 persons died of pneumonia, in Chicago, and, in 1904, 8,360 died of it in New York City. From 1851 to 1890, the mortality, in Chicago, from tuberculosis, was 35 per cent. more than from pneumonia. From 1891 to 1901, the pneumonia death-rate was 9 per cent. more than the tuberculosis mortality.

There are various prognostic factors: (*a*) **The virulence** of the pneumococcus, which varies from year to year (5 per cent. mortality in one year, 31 per cent. in another, Brandes). The mortality of 85,000 cases in the German army was $3\frac{6}{10}$ per cent., but these are necessarily selected cases. In hospitals the mortality ranges between 20 and 40 per cent., and is due to alcoholism, to excesses, and to overwork. In 465,400 collected cases, E. F. Wells found 20 per cent. the average death-rate.

(*b*) **Age.**—According to Fraenkel, the mortality is: one to five years, 30 per cent.; five to ten years, 3 per cent.; ten to twenty years, 5 per cent.; twenty to thirty years, 8 per cent.; thirty to forty years, 25 per cent.; forty to fifty years, 39 per cent.; fifty to sixty years, 43 per cent.; sixty to seventy years, 53 per cent.; seventy to eighty years, 87 per cent. The aged succumb readily; long ago Cruvelhier spoke of pneumonia, in this connection, as the most formidable scourge, and Peter characterized pneumonia as the natural end of old people. Morse, in 118 children, found the mortality 36 per cent. in the first year and 19 per cent. in the second; Revière, 25 per cent. in the first, 15 per cent. in the second year, and 2 per cent. in older children.

(*c*) **Sex.**—The mortality is 50 to 100 per cent. higher in women; in pregnancy the disease is always severe.

(*d*) **Location.**—Right-sided pneumonia is more fatal than left-sided pneumonia, and the upper- is more fatal than lower-lobe involvement. Extension from lobe to lobe is serious.

(*e*) **Unfavorable Conditions.**—These are hepatic cirrhosis and nephritis, in which the death-rate is almost 100 per cent.; delirium tremens and inveterate alcoholism, in which it is 45 to 50 per cent., cardiovascular disease, scoliosis, and high altitude, render the disease dangerous.

(*f*) **Unfavorable Symptoms.**—These are long prodromata, hyperpyrexia, enteritis, marked jaundice, albuminuria, severe pain, marked cyanosis, pulmonary œdema, diffuse bronchitis, and a white cell-count under 10,000. Sears and Larabee found the mortality 9 per cent. with the respirations under 30, 15 per cent. under 40, and 60 per cent. over 60 per minute; and 50 per cent. with the pulse over 130.

(*g*) **Unfavorable Complications.**—Endocarditis and meningitis are almost always fatal; abscess, gangrene, and arthritis are always serious; pericarditis is fatal in 50 to 75 per cent. of the cases.

Strictly speaking, death is due in almost all cases to *cardiac toxæmia.* Mechanical overwork of the heart or diminished respiratory space are of small importance.

Treatment.—(*A*). **Prophylaxis.**—Prophylaxis concerns disinfection of the sputum, cleansing of the room, isolation, and maintenance of the physiological resistance. Cleansing of the nose may be of value in prevention.

(*B*). **Serotherapy.**—Serotherapy has not proved successful, and pneumonia remains a self-limited disease.

(*C*). **Hygiene.**—The hygiene is the same as in typhoid. Quiet should be maintained, the mouth, especially, should be cared for, and the patient should be clothed with a light flannel jacket open down the back.

(*D*). **Diet.**—The diet consists of milk, eggs, and cereals.

(*E*). **Symptomatic Therapy.**—1. The Chill.—The chill is rarely seen by the physician, but the same measures are indicated as in malaria.

2. Cough.—For the cough, nauseating expectorants are to be avoided and codeine gr. $\frac{1}{4}$, and carbonate of ammonium gr. iij, should be given every two to four hours. If these are ineffectual, or disturb the stomach, morphine is indicated (*v. i.*).

3. Pain.—For pain, the chest should be firmly strapped with adhesive plaster, applied in several oblique directions so that the pieces cross each other. Morphine does not essentially depress respiration but rather lessens the irritability of the respiratory centre and relieves pain. Poultices can be more easily endured than ice, but neither are essential. Blisters, leeches, and the Paquelin cautery are superfluous. In pneumonia, general is far more important than local treatment.

4. Heart.—Care of the heart is the *main objective* in the treatment. Constant watching is imperative lest the delirious patient leave his bed, and it is a safe rule to consider all pneumonics delirious. Sitting up to drink, defecate, or urinate, should never be allowed, as pneumonia is a disease of sudden and alarming changes and there is constant danger of sudden death. No therapeutic measure should be employed which depresses the heart, and therefore antipyrin, antimony, aconite, and pilocarpine, are always to be avoided.

Cardiac Stimulation.—*Strychnia.*—Strychnia, grs. $\frac{1}{20}$ to $\frac{1}{30}$ every two, three, or four hours, is the best heart tonic, and should be given in every case because of the cardiac and vasomotor instability which often develops without warning. Vasomotor paralysis and collapse are attended by accumulation of blood in the abdominal vessels. Von Basch observed that pallor of the retinal vessels followed section of the splanchnic nerve. Gottlieb prefers caffeine and camphor to strychnine for vasoparesis. He holds that the dosage of strychnine necessary to constrict the vessels is too nearly the dose likely to produce convulsions. The opinion of the distinguished pharmacologist will not receive general support from clinicians and practitioners. In the collapse caused by acute toxæmia or other conditions, the vasodilatation is general, but particularly concerns the deep abdominal vessels. Strychnine is here indicated, not only because of its general effects but because of its powers of vasoconstriction on the deep vessels, since it is known that the drug tones the abdominal vessels, (while the vascular tonus in the skin and brain is not increased), and thereby flushes the brain vessels and stimulates the bulbar vasomotor and other vital centres. Cold has an analogous effect on the splanchnic nerve. The drug stimulates (1) the central nervous system; (2) the digestive tract, the secretions, and peristalsis; (3) the circulation, the heart-muscle, and chiefly the vasomotor centre, and (4) the respiratory centre.

Administration.—Some strychnine remains in the system from three to eight days, though some is destroyed at once by the liver, and some is quickly eliminated by the kidneys. If the kidneys or liver are diseased or functionally tardy, as they are in old age, the drug may accumulate. Full doses sometimes produce nervousness, irregular heart, palpitation, or renal-vessel spasm, as does digitalis. Strychnine is absorbed more quickly by rectum than by the stomach. Hypodermic administration gives less chance for the liver to destroy the drug than does administration by mouth.

Ammonia.—The preparations of ammonia are of the greatest clinical importance. They temporarily stimulate the heart-muscle, increase its frequency, and constrict the vasomotor system, thereby raising arterial tension. Its effects are soon observed in local flushing, subjective warmth, tension in the temples, a sense of exhilaration, and increase in the urine, sweat, and mucous secretion. The prime indication for ammonia is sudden and functional cardiac adynamia, which occurs at the crisis. It is a local irritant to the skin and to the subcutaneous tissues as well as to the respiratory and alimentary mucosæ, and therein lie its therapeutic value and its clinical disadvantages. Its irritant or stimulant properties upon the nose, mouth, and upper respiratory tubes cause a sudden reflex stimulation of the respiratory and circulatory centres, which follows its application to the nose, and its use as smelling-salts; and cause the centripetal stimulation, which travels from the throat, œsophagus, and stomach, to the brain, long before the drug is absorbed. Untoward action is frequently observed in gastric, intestinal, rênal and bronchial irritation resulting from overdoses, and also in undue irritation or necrosis following subcutaneous administration. The corrosiveness and fugitive action of ammonium carbonate are both very relative contra-indications, and are obviated by administration of small and frequent doses, gr. iij every two hours.

Digitalis.—Digitalis was recommended by Traube and Petrescu, who considered 75 to 150 (!) grains necessary for good results. The administration of these large doses simply proves, in the author's opinion, that the digitalis is not absorbed. It is claimed that exudation is modified and that leukocytosis is increased. Personally, the author has never seen digitalis produce such good result as strychnine does, but he has repeatedly noticed toxic symptoms appear at the crisis when the repeated doses were absorbed together. Collapse may occur. Digitalis should be used only as a last resort, ℳ ij to v of fluid extract (the most reliable preparation) should be given hypodermatically. The author believes digitalin is almost inert (see TREATMENT OF VALVULAR HEART DISEASE).

Alcohol.—The chief interest centres in its action upon the circulation. For years it has been called a heart stimulant, though important objections have been raised to this statement. The frequency of the heart's action is increased, but it is known beyond all doubt that the drug acts as a vasodilator or as a blood distributor. No pharmacologist now holds that alcohol stimulates the heart as do digitalis, strychnine, or camphor. Some clinicians use alcohol for weakened circulation in typhoid, pneumonia, or sepsis, and obtain good results. Other practitioners refuse to employ it because it is narcotic to the nervous system and to the vital respiratory, circulatory, and vasomotor centres in the lower brain. Some of the latter class refuse to use it on account of prejudice and others base their opinions on the laboratory results given above. It must be admitted, however, that alcohol has some good effects. Direct cardiac stimulation by alcohol *per se* is impossible, except in preparations like champagne which contain other ingredients. Alcohol was first recommended by Todd for fevers. It is effective in all varieties of sepsis. It protects tissues from excessive waste and is well tolerated in nearly all cases. There is no danger of cardiac collapse when it is withdrawn in convalescence. Jürgensen gives it in all cases, using red Bordeaux wine as a routine procedure, especially if cool baths are employed. If the heart becomes weaker, Bordeaux is replaced by the stronger sherry or port; then by rum, cognac, champagne, or whiskey in hot water, and tea or coffee, which can also be given by rectum. Alcohol is invaluable, whatever its action; it is second only to strychnine and is indicated by collapse or cardiac toxæmia in which hot whiskey and water unquestionably stimulate by reflex action on the throat and stomach.

Ether.—Dram doses may be injected for urgent cardiac symptoms, but it is apt to produce sloughing.

Opiates.—It is the author's personal belief that opium and morphine are not sufficiently recognized as vascular and cardiac tonics. Many clinicians withhold opiates in pneumonia even when the drug is indicated by maniacal outbursts, active delirium, and harrassing coughing or pain, because it depresses respiration, and ill effects upon the circulation are feared. When used freely, it decreases the irritability of the respiratory centres, and thereby lessens cough and dyspnœa, and the attendant headache and insomnia, and it almost invariably acts as a strong cardiovascular tonic. Its effects on diseased kidneys are not to be feared with ordinary care in administration. Morphine will of itself help to sustain life, and in several most critical cases, where the author has watched its effects for consecutive hours, its hypodermic use has strengthened and regulated respiration even when Cheyne-Stokes respiration was marked. When the heart weakens, its force will often be renewed by morphine. It is a most valuable adjunct to the true cardiovascular stimulants, either those which have continued action, as strychnine and digitalis, or those whose action is more fugitive, such as camphor, ammonia, or caffeine, which latter has an excellent effect, given by rectum, in neutralizing the effects of opiates.

Camphor.—Camphor in grains i to ij, hypodermatically in 10 parts of sterilized oil, every two to four hours, is an excellent diffusive stimulant for the heart and brain.

Saline Infusions.—These are indicated in profound toxæmia with weak heart. Two drams of salt in one quart of water are the rough proportions of the physiological salt solution. They are the "lavage of theblood" of French writers, who combine them with venesection. Although once violently abused, *venesection,* which has been employed since the time of Hippocrates, has again come more into vogue. It is valuable at the very onset in the robust for severe pain, active delirium, and urgent dyspnœa, and later when great engorgement of the right heart occurs. It rather promotes heart thrombosis and delayed resolution. Heart stimulants must be used with phlebotomy.

Oxygen.—Oxygen is said to relieve dyspnœa, and has been used as an adjunct to the heart stimulants, but recent studies have shown that it is often irritating and injurious. A mouth-piece should not be employed, or at least not held closely over the mouth, but merely near it.

Atropine.—Atropine is valuable in collapse with low temperature and clammy skin. The aggregate dosage should not exceed one-fortieth of a grain.

5. ANTIPYRETICS.—A high temperature is not *per se* injurious. Indeed, Cantani proved that cases with fever between 103° and 105° progress most favorably and that those with temperature above or below these limits have a more severe course. Von Jürgensen insists that all measures or remedies which *suddenly reduce* the temperature must be avoided. They are especially injurious at the crisis, when the heart sometimes staggers under the sudden fall of the fever.

Hydrotherapy is opposed by many. Marchand considers that it promotes chronic induration of the lung, though Liebermeister lessened the mortality in his cases by 9 per cent. The tonic vasomotor effects are thought to outweigh the element of shock. It is less indicated in children and is contraindicated in the very obese, the weak, or the aged. Cool sponging with water and alcohol is the safest method. *Quinine* gives no antipyretic effect except in doses of thirty grains or more. Its administration is advocated by Aufrecht, Jürgensen and Sée, who give thirty grains at 6 to 8 P.M., thereby bringing about a slow reduction of temperature without cardiac depression. Aufrecht combines it with iron.

6. NERVOUS SYMPTOMS.—These are toxæmic, but should be considered with regard to: (*a*) fever; the greater the temperature variation, the more marked are the nervous symptoms; febrile nervous symptoms are most efficaciously treated by baths or cool affusions; (*b*) anæmia, the delirium of which is relieved by morphine, coffee, alcoholic and saline infusions; (*c*) deficient oxygenation, in which cold baths, cardiants, and oxygen inhalations are indicated; (*d*) insomnia, which is treated by morphia, bromides, hyoscine hydrobromate, sulphonal, and cool sponging, but never by chloral; (*e*) delirium

tremens (*q. v.*), in which rest is absolutely necessary and is best secured by the drugs named under (*d*) and by alcohol.

7. Tympany.—(See Typhoid.)

8. Iodides and Cod-liver Oil.—These should be given in delayed resolution.

DIPHTHERIA.

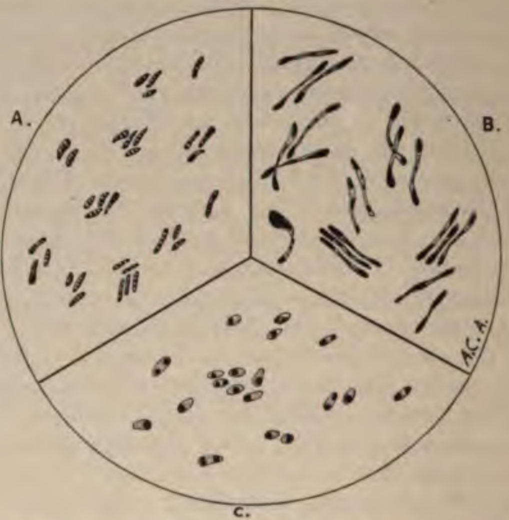
Definition.—An acute specific infective disease, which usually begins in the throat or upper air-passages, and is characterized by a local yellowish adherent membrane which is due to the Klebs-Loeffler bacillus, and by constitutional symptoms due to toxæmia.

History.—Diphtheria was known to Hippocrates. Asclepiades, 100 B.C., performed the first tracheotomy. The name diphtheria literally means membrane and originated with Bretonneau (1821).

Terms and Restrictions.—True diphtheria implies a membrane formed by the Klebs-Loeffler bacillus whose absorbed toxins usually also produce toxæmia. There are, however, membranes not formed by this bacillus but due to the scarlatinal virus, streptococci, pneumococci, staphylococci, caustics, etc. While these are diphtheritic membranes in the old anatomical sense, they are not diphtheritic in the bacteriological sense. We must then term them *pseudodiphtheritic* or necrotic inflammation, or *diphtheroid.* Therefore, clinically, there are some discrepancies: (1) what seems diphtheria, anatomically, may prove not due to the Klebs-Loeffler bacillus; (2) some apparently simple throat inflammations, resembling angina or tonsillitis, may show no membrane or atypical membrane, but bacteriologically are found due to the Klebs-Loeffler bacillus,—*i. e.*, are true diphtheria; (3) the diphtheria bacillus is found in 1 per cent. of perfectly healthy throats; (4) there are bacilli which closely simulate the diphtheria bacillus (*v. i.*). The pseudodiphtheritic or diphtheroid membrane is most often due to the streptococcus and is seen oftenest in scarlatina, but also in typhoid, measles, Vincent's angina, syphilis and pertussis. It develops not merely in the throat, but wherever the true diphtheritic membrane may occur,—*e. g.*, the larynx, bronchi, eyes, skin, etc. As a working statement, most cases clinically resembling diphtheria are proved diphtheria by close and repeated bacteriological tests. G. Smith found the bacillus in 72 per cent. of 27,000 collected cases, though Parks and Bebee find 40 per cent. of membranes diphtheroid—a high estimate.

Bacteriology.—The diphtheria bacillus (described by Klebs, 1883, and cultivated by Loeffler, 1884), is 1.2 to 2μ long and 0.3 to 0.5μ wide, with small round ends, or ends slightly enlarged. It stains readily by Gram's method, by Loeffler's methylene blue, and by carbol fuchsin. There are many irregular variations in form, such as branching types, but the serum culture always reveals the wedge or mallet-like forms. Some are very long, others short and parallel. Their grouping is characteristic, like Chinese letters, in lines set asymmetrically at various irregular or slight angles. They are immotile and sporeless. Cultivated in all media, the diphtheric bacillus overgrows other associated bacteria on blood serum. On blood serum mixed with glucose bouillon, the culture is visible in eight to ten hours as yellow streaks, whose surface is slightly granulated and whose edges are somewhat thick. In twenty-four hours the growths have spread everywhere, with a wavelike surface, high, undulating edges, and a succulent, glistening appearance. The diphtheria bacillus is very resistant, and though it usually disappears in two or three weeks after the patient has shed the membrane, it may live for weeks or months in the throat (even six to fifteen months) or for a long period in clothing. It is found chiefly in the throat membrane, and is less frequently detected in the submucosa, heart (endocarditis), blood, lungs (bronchopneumonic foci), spleen, liver, brain, cord, kidney or urine. In one report, it was said to have been found in the blood and viscera eleven times in fourteen cases. Its presence in these parts has been explained by what we may call diphtheriæmia, by post-mortem migration, or by association with other bacteria, of which the streptococcus is the most important, being found in septic types, in bronchopneumonia, and in glandular suppuration. Symbiosis with the streptococcus, which is the most common of all secondary infections and enters by the throat mucosa, increases the virulence of the diphtheria toxin (Roux and Yersin). Less important are the staphylococci—which rarely enter through the throat, but when they do, are extremely virulent—the proteus, colon bacillus, and pneumococcus.

Fig. 17.



Bacillus diphtheriæ: A, its morphology on glycerin-agar-agar; B, its morphology on Loeffler's blood-serum; C, its morphology on acid blood-serum mixture (Abbott).

The *action* of the bacillus is (1) *local*, disturbing the tissue, producing necrosis, and forming membrane; when inoculated in animals, local necrosis develops, whereon fibrin exudes and forms a membrane in which the bacillus is found; the contiguous tissues are œdematous; (2) *general*, forming toxins which are absorbed by the blood and lymph-vessels. Roux and Yersin (1888) found that the toxins injected alone, after killing the bacillus, produce a general intoxication, but no membrane or necrosis. They succeeded in producing a paralysis precisely similar to the post-diphtheritic paralysis. According to Ehrlich there are two toxins; one, the *toxin*, produces the ordinary acute phenomena of diphtheria; and the other, the *toxon*, produces the later anæmia and paralysis. The chief receptors of the virus are the nervous and lymphatic tissues.

The *virulence* of the germ varies. This is tested on the guinea-pig by inoculating it with $\frac{1}{100}$ of its body-weight of a forty-eight-hour bouillon culture, and by noting the results: (1) death in three days or less results from fully virulent cultures; (2) death in three to five days from one of medium virulence; (3) death after a longer time, or only local changes from weak cultures.

Some bacilli, known as the pseudodiphtheria bacillus, bacillus xerosis, etc., microscopically resemble the Klebs-Loeffler bacillus.

PSEUDODIPHTHERIA BACILLUS.——*vs.*	——DIPHTHERIA BACILLUS.
Luxuriates on gelatin and peptone agar.	Sparing growth.
Produces either an alkali or very little acid in culture.	Growth on bouillon should produce in twenty-four hours enough acid to neutralize 0.07 gm. of a 1-per-cent. sodium hydrate solution.
"Klatsch preparat" (adhesion preparation) does not develop in six or eight hours, and if it has, the bacilli are arranged irregularly.	Show the bacilli in colonies arranged in parallel or crossed lines within six or eight hours.
Are not pathogenic to guinea-pigs. (Some claim this type is only an attenuated form of the real bacillus.)	Kills (*v.s.*) in seventy-two hours. (Some apparently genuine forms may be nonvirulent.)
Smaller.	

In the pseudodiphtheria bacillus Neisser's stain does not bring out the granules which are present in the true diphtheria bacillus, in ox-serum growths under twenty hours old; the body of the diphtheria bacillus stains brown and its two to four polar granules stain a deep blue. (Stain preparation for one to three seconds in a solution of methylene blue 1.0 gm., 96 per cent. alcohol 20.0, glacial acetic acid 50.0, and water 950.0. Wash in water and stain again for from three to five seconds in solution of vesuvin 2.0 and 1000.0 gm. boiling water.) Another interesting and vital point is that pseudodiphtheria bacilli may become virulent and may kill; they are not neutralized by the diphtheria serum but only by the pseudodiphtheria serum (Alice Hamilton, Spronek, Ruediger).

Dissemination.—Diphtheria is disseminated (1) by patients acutely ill with the disease; the nasal, throat and laryngeal secretion or membrane may be coughed into the face or mouth of one of the family, of the nurse, or of the attending physician. Infections in which the patient is not kept in bed are especially dangerous, for children going to school may impart to others a most virulent diphtheria, though the same bacilli have produced in them little or no constitutional reaction; (2) by healthy individuals, in 1 per cent. of whom the germ is found without causing diphtheria, since individual predisposition is almost as necessary to infection as virulence in the microbe; (3) by fomites, especially damp cloths, in which the germ may live for months; cultures have been made from dry cloths after five months; the germ lives in gelatin from twelve to eighteen months; (4) as the germ may be cultivated in dust, it is probable that it may be spread by the wind. Although the bacillus has been cultivated from cheese, milk, and eating utensils, many question this mode of propagation. It is not demonstrated that domestic animals convey the disease. Diphtheria is endemic in all thickly populated districts, and from these, epidemics develop from time to time. Unlike scarlatina, which is growing milder, diphtheria epidemics are apparently growing in virulence, although they are far less malignant than in the middle ages and earlier modern times.

Age.—Most cases occur between the second and fifteenth years; and most deaths occur between the second and fourth years. Adults are frequently affected. Jacobi has seen three cases in the new-born, but infants at the breast are far less exposed. Creeping children are frequently attacked, because their hands and mouths come into close contact with dirt and dust (Jacobi). It develops more frequently in girls than in boys.

Season.—Most cases occur in the cold months. Predisposition, bad teeth, stomatitis, hypertrophied tonsils, nasopharyngeal and bronchial catarrh, measles, and nervous affections, are predisposing factors. Some individuals are temporarily, and some permanently, immune. One attack confers no immunity, and in this respect diphtheria resembles erysipelas, pneumonia, and rheumatism.

Symptoms.—Baginsky's classification of (1) local infection, (2) general infection, and (3) septic infection, is better than the old division into mild, moderate, and severe forms. Incubation lasts from two to seven days.

1. **The simple localized diphtheria** begins with fatigue, sopor, pallor, coated tongue, anorexia, dysphagia, pain beneath the angle of the jaw, and vomiting. These may be the only symptoms, but usually there are also chilly sensations, fever and thirst, dry tongue and lips, pharyngeal voice, sometimes constipation and abdominal distention. Infants sometimes refuse the bottle on account of nasal obstruction.

The *pharynx* is moist, glistening, œdematous, and reddish, with areas of gray, yellowish-gray, or dirty green color, which vary in extent from small white bands to irregular, gelatinous elevations or masses, and are located on the tonsils, epiglottis, palate, pillars, arch, or pharynx. They are adherent, and, on removal, leave red, bleeding spots which are soon covered again with membrane. The pharyngeal tonsil alone may be involved, and this infection may easily be overlooked without careful systematic examination.

The *typical membrane* is accompanied by fibrinoid swelling of the tissue, fibrin formation, emigration of cells, and necrosis. The Klebs-Loeffler bacillus is found on the surface in pure cultures, and streptococci and staphylococci may be found more deeply situated.

Wagner, Weigert, and Oertel, have shown that the diphtheritic membrane is composed: (*a*) of necrobiotic tissue. The superficial epithelium, and, later, the deeper structures, are necrosed; the bacilli are found in the superficial layers, and the necrobiosis may be also noticed in the connected lymph-glands, and in the internal organs (Weigert's coagulation necrosis, or hyaline transformation of the dead cells). (*b*) Somewhat deeper, the membrane is composed of more or less marked fibrin exudation, which is probably a conservative, walling-off process, which also pervades the tissue initially necrosed. (*c*) In the deepest part of the membrane, emigration of leukocytes occurs. In most adults the membrane is thrown off by the fourth or fifth day; in children by the seventh or eighth day.

The nose is not wholly free. The glands at the angle of the jaw are tender, and fever may reach 102° or 103°, may be rather irregular, or may be absent. The patient usually recovers in seven to ten days. Otitis, suppurative adenitis, vomiting, and albuminuria, are less common than in the more severe forms.

The form described is the classical type of mild local infection. Even though the bacilli may be virulent, the tissues and blood resist the infection. There are several deviations from this type which are often clinically misinterpreted: (I) In "catarrhal diphtheria" the membrane is often absent. This type is especially common in families where the more severe forms are seen. The bacilli, and redness, œdema, and swelling, are present. If a membrane develops, it may be atypical and pultaceous. (II) Diphtheria may resemble follicular pharyngitis, and between them there is no absolute distinction except a bacteriological one; in diphtheria, however, the red is brighter, the tendency to fuse is greater, and the spots are yellow, gray, or green, while in angina, the color is duskier, the areas more discrete, yellow, and pus-like. (III) Follicular tonsillitis may be very closely simulated, and may be only distinguished bacteriologically. (IV) In chronic diphtheria, there is a variable deposit upon a red, relaxed, œdematous pharynx. This deposit may spread over the tongue. The glands may be swollen, but usually there are no general symptoms. (V) The latent diphtheria of Huebner, which occurs in marantic subjects, may easily escape detection.

2. The diphtheritic general infection is difficult to describe in all its variations. It begins with high fever and such febrile toxæmic symptoms as headache, depression, insomnia, sopor, rapidly increasing anæmia, anorexia, dysphagia, and vomiting. Albuminuria and nephritis may appear later, with rapid respiration and the heart movement following the fever, the tones being dull and often accompanied by a systolic murmur. The pharynx is dark red, swollen, and glistening, and a membrane is seen on the tonsils or pharynx. This membrane is thin and gray; it begins in the form of streaks, or as a thin veil over the pharynx; it tends to spread very rapidly and sometimes results in a diffuse membrane with offensive secretion. It usually leaves exempt the hard palate as well as the superior part of the soft palate. The neck is tender from glandular intumescence. The nose is often closed by a purulent secretion and diffuse membrane, and oral breathing results. There is a type peculiar to young children who, with initial nasal symptoms and without appreciable throat symptoms, suddenly develop laryngeal stenosis, and, in the pre-antitoxin days, almost invariably died. In severe cases the heart is irregular with gallop-rhythm; there are throat paralysis, infiltration of the neck, dyspnœa, cyanosis, albuminous urine, tympany, swollen liver and spleen, ante-mortem fall of temperature, and death resulting from (1) pneumonia, (2) cardiac insufficiency, (3) nervous symptoms or paralysis or (4) nephritis.

3. **Septic diphtheria,** according to Baginsky, is due not to mixed infection but to the diphtheria toxins, and is therefore seldom observed since the introduction of antitoxin. Other writers maintain that streptococcus and staphylococcus infection determine this septic type. The yellow-gray exudate becomes a dirty green or brown from hemorrhage, is ichorous and horribly fetid, and may reach the nose or larynx, causing erosions, necroses, and stenosis. It often involves the lymph glands (adenitis) and connective tissue (periadenitis). The tongue is dry; respiration difficult; and nephritis, generalized hemorrhage, or local arterial ulceration, failing pulse, low temperature, swollen joints, and intumescence of the abdomen, liver, and spleen, usually result fatally. In 1884, 66 per cent. and, in 1881, 94 per cent. of cases of this type died (Glaeser).

Special Symptoms.—1. **Fever.**—The fever in diphtheria is not typical. It often rises to 103° and remains there for one or two days, when it falls by lysis, to rise moderately again from the fourth to the seventh day. It may be maintained by otitis, pneumonia, adenitis, and other complications, or by concomitant diseases, such as measles, scarlatina, or tuberculosis. The temperature of sepsis may develop. A preagonal perturbation may be observed, or there may be ante-mortem collapse. Algidity is due to febrifuge toxins, as it is in perforative peritonitis. These statements apply chiefly to cases untreated with antitoxin.

2. **The Blood.**—Leukocytosis develops in 90 per cent. of diphtheria cases, and is of the polymorphonuclear type. When absent, the infection is either very severe or very slight. It does not correspond to the severity of the disease nor to the fever curve. It disappears in favorable cases, and persists in those which are severe.

Special Localizations of Diphtheria.—1. **Diphtheritic Rhinitis.**—Diphtheritic rhinitis, catarrhal or croupous, due to the diphtheria bacillus, may usher in the disease or accompany the benign or more usually the severe forms. The serous and often sanious or bloody nasal discharge excoriates the alæ nasi, lips and cheeks; membrane is often expelled; infection may travel to the eyes, lachrymal sacs, antrum, or ears. Cervical adenitis is frequent, for the nasal lymph supply is most abundant and, as Jacobi pointed out, enlargement of the angular glands is always suspicious. The prognosis is nearly always grave, and convalescence very tardy. In contrast with this severe form, usually diphtheritic, though sometimes diphtheroid (streptococcic), there is a fibrinous rhinitis usually subacute or chronic and generally observed in children, which is due to the Klebs-Loeffler bacillus (82 per cent.), though sometimes to the streptococcus (18 per cent. of the cases); the membrane is abundant. This form is obviously dangerous to other children, since the remarkable absence of constitutional reaction does not prevent play or school-going. It responds readily to antiseptic douches.

2. **Diphtheritic Croup** (Diphtheritic laryngo-tracheo-bronchitis).—Most cases (85 per cent.) of croup are diphtheria, though some are due to streptococcic infection; their symptoms are identical. *Frequency:* Diphtheria involves the throat in 84 per cent. and the larynx in 16 per cent. of the cases. *Forms:* (*a*) Catarrhal laryngitis, due to the diphtheria bacillus, produces symptoms of short duration, usually two or three days; they may be severe or even stenotic. (*b*) Diphtheritic laryngitis (croup) is usually descending (97 per cent. of cases)—*i.e.*, secondary to throat or nasal diphtheria, but may on rare occasions be primary in the larynx; even in these cases there is usually some pharyngeal involvement. The ascending form is most exceptional. The child is usually hoarse at night or early in the morning, coughs the croupy cough, and then after twelve to twenty-four hours is distinctly dyspnœic. The accessory muscles of respiration come into play and there is inspiratory retraction of the thorax and epigastrium, especially in rhachitic children. Excursion of the larynx up and down, extreme restlessness, and temporary improvement after expectorating the membrane, may also be noted. The stenosis is due to membrane in the epiglottis, true cords, trachea, or even in the bronchi. The membrane is less adherent than when it is in the pharynx, and sometimes more pultaceous. The stenosis is accentuated by the central and immovable position of the vocal cords, by swelling in the subchordal tissue, and by muscular spasm. Aphonia is common but not invariable. The cough is usually rather hoarse than metallic, is often periodic, is increased after tracheotomy, or intubation, and often evacuates both the membrane and tube. Cough may be wholly absent because of decreased reflex bronchial excitability.

Temperature is usually absent though it may arise from complications. Fever after tracheotomy or intubation is ominous. The pulse rate increases with augmentation of the stenosis because the vessels contract from central stimulation. The respite following the raising of the membrane is usually transient, exacerbations occur, and finally, dyspnœa becomes continuous with stagnation of secretion in the tubes, absence of the vesicular murmur, stridor, extension downward of the membrane in some instances, loss of strength, increasing rapidity and weakness of the pulse, cold, clammy skin—in short, the complete picture of carbon dioxide poisoning and total asphyxia. In some instances, perichondritis, deep necrosis, or gangrene may develop. (In reading older medical works and pathologies, the student may be confused with the use of the terms "croup" and "diphtheria." Anatomically it was held that croupous and diphtheritic membranes differed chiefly in degree—croupous being more superficial and benign, diphtheritic being more deep and malign. In the present connection we use the term "croup" as synonymous with laryngeal diphtheria.)

3. Diphtheria of Skin.—True diphtheria caused by the diphtheria bacillus may produce a membrane on the eye, lips, nose, mouth, navel, neck, genitalia, and extremities. It is usually associated with the more common throat localization. The skin is hard and infiltrated, the secretion is corrosive, the contiguous lymphatics are involved, and sometimes cellulitis or phlegmon may result. Other cases of so-called "wound diphtheria" are due to the streptococcus, which is the most common cause of skin membranes. In tracheotomy, the membrane which forms in the wound is mostly streptococcic.

4. Diphtheritic Vulvo-vaginitis.—Genital involvement is rare (under 1 per cent.), is dangerous, and may be primary, but usually follows severe pharyngeal diphtheria; it consists of diffuse membrane, painful rhagades, ulcers, and foul secretion, with intumescence of the lymph glands. A diphtheroid (usually streptococcic) form is also encountered.

5. Diphtheritic Ophthalmia.—This localization is (*a*) false or diphtheroid (due to pus cocci) or (*b*) genuine or diphtheritic. It may here be noted that the old division into croupous (benign), and diphtheritic (malign) forms is no longer valid. Diphtheritic ophthalmia, chiefly in young children, occurs in 3 per cent. of cases. It may be primary in th eye, or secondary to nasopharyngeal diphtheria.

6. Diphtheritic Otitis.—This does not occur in over 5 or 6 per cent. o the cases (as against 33 per cent. of otitis in scarlatina and nearly 10 per cent. in measles). The orifice of the Eustachian tube or the middl ear may be reached by the diphtheria bacillus which usually travel from the throat.

Complications and Sequels.—The most important are diphtherit paralysis, heart paralysis, and nephritis.

1. Diphtheritic Paralysis.—The paralyses result clinically an experimentally from toxæmia, and occur in 3 to 10 per cent. (even per cent.) of cases. The percentage, apparently, has not been reduce by the use of antitoxin (*v. i.*). The frequency and intensity of t paralysis is usually directly proportional to the intensity of the dip theria but there is no invariable relation between the palsy and t severity of the infection. Indeed the causal diphtheria, in some case has apparently escaped detection.

(*a*) *Early Diphtheritic Paralysis.*—This occurs in septic cases at the acme. Dysphagia is its most common form. There is danger of inhalation pneumonia, especially in stupid cases. Other changes may occur, such as the disappearance of the patellar reflexes, and changes in sensation. Most cases die.

(*b*) *Post-diphtheritic Paralysis.*—This variety occurs after two or three weeks and usually follows throat lesions since laryngeal cases are likely to die early; Baginsky saw it principally in adults and Rolleston mostly between the ages of three and six years. Gowers claims that all throat troubles leading to paralysis are diphtheritic, though Bourges and others cite cases of paralysis after simple angina. The most common paralysis is that of the palate, which causes dysphagia, anæsthesia of the throat, and nasal speech. From central or peripheral change the pupils become wide and react to light but not to accommodation (Donders). The arms may be affected, but frequently escape. Ataxia may be the first symptom. Heart or central nervous symptoms may appear (*v. i.*). The reflexes disappear early whether actual paralysis is reached or not, the muscles react slowly, if at all, to the faradic current, although the reaction of degeneration runs by no means parallel to the degree of loss of voluntary motion. The muscles waste considerably. The sensory nerves suffer less, sensation being largely retained above the knees. The palsied limbs are sometimes painful and the muscles and nerve trunks are often tender. Sensory disturbance may be noted in the rectal, genital, and vesical twigs. The trunk, bladder, and rectum are more commonly involved than is usually admitted. The cranial nerves (strabismus, ptosis, diplopia), the phrenic nerves and the vagus (recurrent paralysis, superior laryngeal, cardiac branches), may be involved; fearful abdominal pains with threatening collapse attributable to sympathetic involvement, and sometimes psychical disturbance may be noted. Anatomically, disintegration of the medullary substance, multiplication of nuclei in Schwann's sheath, granular degeneration, and even total loss of the axis cylinder and sometimes foci of hemorrhage, are present, the changes being almost totally parenchymatous. Diphtheritic neuritis (actual nerve inflammation) is seen almost wholly in the palate and is due to local invasion from the inflamed throat. The patient recovers in two to three weeks if the pharynx alone is involved, and in three to four (even six to eight) months if the limbs are affected. Death results in 18 per cent. of the cases, chiefly from vagus involvement.

(*c*) *Organic Central Changes.*—The brain is slightly congested and œdematous. Degenerated areas may occur in the crura, medulla, pons or the motor nerves, and are less marked in the sensory nerves. Accidental findings are cerebral hemorrhage, embolism from heart thrombi, and acute encephalitis (possibly terminating in focal sclerosis). In the spine, hemorrhage, exudation into the nerve roots, meningitis, and degeneration of the motor cells, with disintegration of their roots, have been noted. The degenerative cerebrospinal lesions occur not infrequently with multiple neuritis.

II. **Cardiac Complications** (Diphtheritic Heart Paralysis).—Anatomically, the heart muscle is the seat of early changes. It is soft, thin, friable, light brown or yellow in color, and fatty; it is the occasional seat of endocardial and myocardial hemorrhages, acute interstitial myocarditis and fragmentation or myolysis, which Eppinger, in describing eighteen cases of sudden death, considers the usual cause of death. Pericarditis, with septic or hemorrhagic effusions, is not frequent. Permanent changes due to endocarditis (in 2 per cent. of fatal cases) or to myocarditis are uncommon.

Clinically, the tones are muffled. The first tone is impure or obscured by a systolic murmur (90 per cent. of cases); the second pulmonic is accentuated, the rhythm is disturbed (60 per cent.) and sometimes there is gallop-rhythm and embryocardia. The pulse is soft, dicrotic, increased from 80 to 120, or up to 150 if there is fever, but it may be slow. The heart-failure is more frequent than it is in any other acute infection, not even excepting pneumonia. Syncope on sitting up is fairly common. Diphtheritic heart paralysis occurs (1) early in severe or gangrenous types, or (2) late (in 10 to 20 per cent. of diphtheria cases) after three to four or six weeks, when convalescence is apparently established. It is marked by copious vomiting, pain in the chest and epigastrium, insufficient heart action, cardiac dilatation, cold extremities, pallor, dyspnœa, cyanosis and convulsions. The pulse is often irregular, fast, or with equally ominous significance, slow. Heart failure is explained in various ways (1) acute interstitial myocarditis (Rhomberg); (2) myolysis (Eppinger); (3) sudden centric vasomotor paralysis (Hasse); (4) neuritis of the cardiac plexus or of the vagus or phrenic nerves; (5) cardiac thrombosis causing cerebral or pulmonary embolism; 33 per cent. die.

III. **Diphtheritic Nephritis.**—(*a*) Albuminuria, resulting from renal degeneration, occurs in 50 per cent. of all cases and in 100 per cent. of severe cases. (*b*) In *light* forms of *nephritis* there is some albumin; the specific gravity and quantity of the urine are about normal, and there are leukocytes or epithelial cells with indistinct nuclei, the cells being highty refractile and coarsely granular. Blood is rarely found. There are some hyaline and granular casts. These findings are caused by the toxins and correspond in time and degree to the diphtheritic local process in the pharynx. (*c*) The *severe forms* of *nephritis* almost always occur in the severe general infection or in septic types and are characterized anatomically by diffuse parenchymatous changes, necrosis, hemorrhage, and exudation. Albumin, cells, casts, and cylindroids, are abundant. Hæmaturia is rare, although hemorrhage into the kidney substance is frequent anatomically. Although nephritis influences the prognosis, diphtheritic nephritis rarely produces hydrops or uræmia and "is not an independent factor as in scarlatina." It very rarely becomes chronic.

IV. **Respiratory Complications.**—The bronchi and trachea may remain free. There may be membrane formation (in 50 per cent. of fatal cases) or purulent secretion. Lobar pneumonia is a genuine complication, though *bronchopneumonia* is present in 50 to 80 per cent. of the fatal cases and is caused by the Klebs-Loeffler bacillus alone, or by the pus or pneumonia cocci, by the colon, proteus or pyocyaneus bacilli. Anatomically, some cases are due to downward extension of the bronchitis; some cases are hæmatogenous infarcts or abscesses; others are due to inhalation, and still others, seen after intubation or tracheotomy, rather resemble lobar pneumonia and are elevated and granular in appearance. Increase of pulse and respiration-rate, local consolidation, and accession of fever are observed. Pleurisy (serous, hemorrhagic or purulent) is not often observed.

V. **Alimentary Tract.**—MOUTH.—Fissures, sordes, membrane or ulcers on the lips, suppuration in the tonsils and in the retropharyngeal space, gangrene, and deforming cicatrices, sometimes occur. The œsophagus is usually intact, but the diphtheritic process, in septic cases, may reach downward and produce stenosis.

STOMACH.—Such symptoms as epigastric pain and vomiting are most often cardiac; such rare organic changes as hemorrhage and membrane formation usually escape clinical recognition.

INTESTINES.—Constipation is usual; in septic forms, thick, foul or even dysenteric evacuations may be exceptionally observed. Anatomically, there may be membrane formation and swelling of Peyer's patches. The liver is swollen from fatty or cloudy degeneration or from congestion, in which latter case it also becomes painful. Icterus is rare. Splenic tumor is found in septic types especially.

VI. **The Skin.**—The skin is usually normal, except for the febrile hyperæmia, cardiac pallor, or croup-cyanosis. The following alterations may be observed: Herpes, profuse sweats, petechiæ and the gray-blue color in septic types, symmetrical gangrene and dermatomyositis due to septicopyæmia; they usually occur with adenitis, nephritis, pneumonia, ulcerative endocarditis and scarlatiniform or morbilliform erythemata. Scarlatina or measles may actually complicate diphtheria.

VII. **The Glands.**—The anterior cervical glands are swollen, tender and painful. In the most severe forms periadenitis follows with diffuse involvement of the cellular tissue (angina Ludovici), section of which evacuates serum, though seldom pus. Glandular and articular changes are less frequent than in scarlatina. Muscular degeneration, interstitial infiltration, and tendency to separation of the muscle-fibers, are observed in the heart (myolysis or fragmentation) or even more clearly in the diaphragm.

Diagnosis.—To the naked eye a thick, adherent membrane, which is not removable without hemorrhage and which leaves ulceration (Bretonneau), is sufficiently characteristic. The enlarged glands are of diagnostic value. A positive bacteriological diagnosis is important, in such cases as have actual membrane, diffuse reddening, and tonsillitis; but three or four negative examinations are necessary before negative conclusions can certainly be drawn. The statistics of Heubner and of Baginsky show positive findings in from 92 to 98 per cent. of cases in which the clinical diagnosis of diphtheria has been made. These figures are probably most nearly correct, although others find the diphtheria bacillus in a notably lower percentage. The nose, nasopharynx and pharynx must always be examined in sick children and also in adults. In suspicious cases, particularly when cultures cause considerable delay, antitoxin should be given at once.

The health departments in our large cities provide apparatus for growing cultures, stations at which cultures and antitoxin may be obtained, and central and subsidiary laboratories for diagnosis. The Chicago Health Department has also a most able corps of physicians for free treatment, consultation and intubation. It is easy to obtain on sterile cotton on a probe a smear from the membrane or suspicious focus, to rub it on the serum without breaking its surface, and to put the tin box containing it in the incubator or under the axilla until the brood oven can be reached. In half a day characteristic cultures are obtained. Direct smears from the throat on a slide very often show the bacillus. In small places communal laboratories can easily be maintained, and competent young practitioners found to do this, and other clinical microscopy. In laryngeal diphtheria the bacillus is usually found on the pharynx, but not on the tonsils. Repeatedly negative findings are of value in the *pseudodiphtheritic* or diphtheroid pharyngitis, rhinitis, laryngitis, and bronchitis,—*e. g.*, membranes due to strepto-, staphylo-, and pneumococci, aphthous growth, syphilis, and the colon bacillus. The *scarlatinal* membrane has been considered. In *Vincent's angina* (*q. v.*), angina diphtheroides or angina ulceromembranosa, described by Vincent (1898), there is a whitish-yellow to grayish-brown membrane which may be confused with diphtheria and syphilis and which is caused by the symbiosis of two otherwise innocuous parasites; first the bacillus fusiformis (6 to 12 μ and staining readily with methylene blue and fuchsin), and the spirochæta darticola (twice as large, and not growing in cultures). The infection is usually mild and the ulcers heal slowly under chlorat. potash gargles or painting with pure iodine.

Course and Prognosis.—Aside from complications, the average course is one week, or possibly two. It is more favorable in diphtheria of the tonsils and skin than in that of the nose. The prognosis must always be guarded. (*a*) Without serotherapy the danger is always great, since light cases may become severe or extend to the larynx. (*b*) Epidemics vary in virulence, 30 to 50 per cent. mortality having been observed twelve to fifteen years ago. (*c*) According to Glaeser, in the first year the mortality is 77 per cent., and sinks with each year until it is, at ten years, 20 to 25 per cent.; after ten, 2 to 5½ per cent.; after thirty and forty, 1 to 3 per cent. (*d*) Other factors are debility, wide extension of the infection from part to part, adenitis, and croup; the general diphtheritic infection or septic forms with hemorrhage or gangrene; heart and renal complications; rapid and weak or slow pulse; involvement of the brain or nerves. (*e*) In 946 fatalities studied by White and Smith, about 50 per cent. died of bronchopneumonia, usually after intubation or tracheotomy; 25 per cent. died of cardiac complications, and 25 per cent. of early severe toxæmia, though some succumbed to asphyxia or late paralysis. (*f*) Coincidence with other acute infections is ominous, such as measles, in which the exanthem may become hemorrhagic, and respiratory catarrh, may result in a wide Klebs-Loeffler infection, the combination being usually fatal; coincidence with typhoid, frequently with gangrene or laryngeal stenosis; and with scarlatina, summer diarrhœa, and tuberculosis. The disease is less severe in conjunction with varicella and pertussis. Diphtheria belongs to the class of infections which are likely to recur.

Treatment.—First Indication—Prophylaxis.—For the physician, this begins with the case in hand. Isolation must be absolute. In needy families the isolation hospital offers the best solution. In Boston it has reduced the mortality of diphtheria to one-third or one-sixth of its former rate. All suspicious throat cases should be isolated pending their immediate clinical and cultural evolution. Not only should a sick child be isolated, but other children in the family should be kept from school and away from playmates. Of course, the sick one must be isolated until its throat gives negative cultures. The physician may protect himself by examining the throat through a piece of glass, and may avoid carrying the germs by slipping on a surgical gown and washing both face and hands with bichloride of mercury or similar solution. In fatal cases the body should be wrapped in a sheet saturated with bichloride, and buried privately in a tightly closed casket. . Those exposed to the infection may receive antitoxin, and should gargle the throat once with $\frac{1}{2}$ of 1-per-cent. solution of silver nitrate, or often, with bichloride of mercury ($\frac{1}{5000}$). The clothing, blankets, rugs, etc., must be steamed. After convalescence, the walls of the room should be rubbed with bread which is then burned, the woodwork should be washed with 2-per-cent. lysol solution or 1-per-cent. bichloride of mercury and the room should be fumigated with formaldehyde. Under prophylaxis may be included the building up of the general physiological resistance and the treatment of chronic catarrh of the nose, throat, and bronchi.

Second Indication—Local Treatment.—Though local treatment of the throat is less important than before the days of serotherapy, the antitoxin does not always kill the bacilli, and does not affect the associated streptococci. Caustics are never necessary. In children such mild antiseptics as 3-per-cent. boric solution and 3 to 10-per-cent. potassium permanganate solution, should be used lest they be swallowed. The child is held in the nurse's arms with its arms and body firmly wrapped in a sheet. The mouth is made to open by closing the nostrils, and a piece of wood or cork is inserted between the teeth. The throat is then swabbed. It is a harsh procedure and the effects are apparently more than offset by the resultant exhaustion. In adults 1 to 1000 bichloride, 25-per-cent. hydrogen peroxide, or 1-per-cent. carbolic solutions, 5-per-cent. ammonium sulphocarbolate, 5-per-cent.

ferric chloride or iodoform salve, may be used, or Loeffler's solution, which is as follows:

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Menthol	10 parts.
Toluol	30 parts.
Tr. ferri sesquichloridi	4 parts.
Alcohol absoluti	60 parts.

Loeffler's solution should be kept well corked in a dark bottle. Local injury, such as opening up new wound surfaces for infection, must be avoided. In nasal irrigation the tube is held horizontally, so that fluid may return by the other nostril. A kettle containing a very weak carbolic solution should be kept steaming in the room.

Third Indication—Serotherapy.—Gscheidlen and Traube showed that the blood possessed antibacterial action, both within and without the body, and immunity was produced by Behring against anthrax and by Bouchard against the Bacillus pyocyaneus. Behring demonstrated also that the serum in diphtheria and tetanus could overcome bacteria without destroying them. In 1888, Richet and Hericourt made animals immune to the streptococcus, and Behring and Kitasato produced immunity to tetanus. Behring (1890) borrowed Ehrlich's results with ricin and abrin to establish the units of immunization, and (1893) used the diphtheria antitoxin on diphtheria patients. The method of action of an antitoxin is vital or active (Buchner, Roux, Martin) though some, (Behring and Kitasato) consider that it is chemical or passive.

1. *Technique.*—A syringe with an asbestos plunger should be used. The syringe need not be boiled each time, but should be cleaned after each injection with water until the serum is washed out; it should then be washed with carbolic acid or alcohol, which should be thoroughly removed each time lest it coagulate the antitoxin at the next injection. The needle must be boiled each time. With ordinary surgical antisepsis, inject the antitoxin into the subcutaneous tissue of the thigh in adults, and into the interscapular region in children. In very severe infections, intravenous injections should be employed as results may be thus obtained eight hours earlier. The unit is 1 c.c., which counteracts ten times the minimum dose of diphtheria poison fatal to a 300 gm. guinea-pig.

2. The *dosage* is rather empirical, depending on the localization, age, and severity of the disease and the effects produced. The usual classification is as follows: (*a*) *Single:* 600 units, used in cases under two years, without croup; for immunization, a somewhat smaller dose is required than for cure (200 to 500 c.c.). (*b*) *Double:* 1,000 units, in children under two years when croup, fetor or necrosis occur; in older children with simple symptoms. (*c*) *Triple:* 1,500 units, in severer cases. On the whole, dosage is governed by results (*v. i.*). After administration the discharge should lessen, the membrane should exfoliate, the fever should fall, and toxæmia decrease. At least twice as much antitoxin is necessary for cure as for immunization. After

eight hours, three times as much is necessary as at the beginning; after twenty-four to thirty-six hours, eight times as much. As we cannot measure the toxins, so we cannot exactly specify regarding the antitoxin dose. Personally, the author prefers an initial dosage of 3,000 to 5,000 units to insure results and to avoid delay and frequent repetition. The decreased bulk of present preparations also allows a larger dosage. In severe cases, twice the above amount should be given. McCallum, above all others, has emphasized the importance of full doses, and he speaks, and with reason, of doses of 10,000, 30,000, or even 50,000 units in desperate cases, because results must be produced. In a recent article, H. A. Hare states that antitoxin dosage is not measured by units and that children require as large doses as adults. He cites Sutherlin as giving a total of 498,000 units in one case!

3. *Effects.*—(*a*) The local changes are lessened, the mucosa becomes redder, and the membrane more yellow and it exfoliates in from one-half to two or three days, with decrease in glandular swelling. Extension afterward is rare. (*b*) The fever falls by crisis or by lysis, unless there are complications. A temporary rise in temperature sometimes occurs. Antitoxin is given until the fever falls. (*c*) The pulse is lessened, and hyperleukocytosis is decreased. Light cases rarely become severe, and nonseptic rarely become septic, unless there is coëxisting tuberculosis, syphilis, etc. The kidneys are never injured. The author believes that paralysis and heart failure are less frequent when the administration of the antitoxin is prompt. Both of these complications are thought to occur in the same proportion as in pre-serum days. But even if their percentage is not absolutely reduced, many patients now live who would previously have died before developing paralysis. (*d*) The mortality is reduced from 41 (or 43 to 46 per cent.) to 15 per cent. (Siegert's collection 42,000 cases); 10 per cent. (Wenner, 9.8 per cent., 132,500 cases, American cities); or 6.7 per cent. (Chicago Health Department Report). London statistics give a reduction of one-third in the general morality and one-half in tracheotomy (60 per cent. morality reduced to 32 or 25 per cent.). The fatal cases and severe complications are lessened in direct ratio as the antitoxin is given early; if on first day of disease, 0; second day, 2 per cent.; third day, 8 to 10 per cent.; fourth day, 14 per cent. Failure to use antitoxin is criminal neglect; 50 per cent. of croup cases regress with the use of antitoxin, and heart lesions are reduced 50 per cent. in fatal cases. (5) Antitoxin is a prophylactic, and should be given in special instances; *e. g.*, to weak or very young children (in smaller doses). It protects for two or three weeks in all but 0.7 per cent. of 1,000 cases (Zuppinger). (6) The sequels are: (i) Abscess formation; (ii) exanthemata, which are local (on the fourth or fifth day) or general (in one to two weeks), are due to individual peculiarity or some unknown element in the antitoxin, and appear as urticaria, morbilliform, scarlatiniform, or petechial eruptions; antitoxin rashes occur in 33 per cent. of the cases; and are said to be decreased by the administration of potassium acetate (gr. x to xxx), combined with free catharsis

(Roehr); (iii) nephritis, which is always due to the diphtheria itself; (iv) sudden death which results from vomiting into the larynx, shock, lymphatic diathesis, and from other disease; sudden death is exceedingly rare; (v) tetanus has been inoculated. In a few cases where antitoxin was injected for prophylaxis, the author has seen extremely severe symptoms; pallor alternating with flushing, chills, fever, renal suppression, œdema of the skin and pharynx and very severe cardiac weakness; these cases to a certainty had no diphtheria, for throat cultures were first taken; the statement, therefore, that antitoxin *never* injures does not hold for all prophylactic doses.

Fourth Indication.—General and Symptomatic Treatment.—A rich *fluid diet* of milk, eggs, peptonoids, with tonics, cinchona, wine, etc. is given. In marked dysphagia rectal feeding is indicated. Forced feeding by the stomach tube or nasal catheter is dangerous because injury to the throat is likely.

SEPSIS.—Concentrated diet by mouth and rectum, alcohol *ad libitum*, oxygen, saline infusions or enemata, strychnine and iron, and Marmoreck's antistreptococcic serum should be given; but all measures are usually of no avail if 4,000 units (Baginsky) or tens of thousands of units do not give relief. Credé's collargol is given intravenously in severe cases; Netter, in 586 cases, found that it reduced the death rate by 8 to 20 per cent.

PARALYSES.—General stimulation and alimentation are indicated; electricity is of little or no avail; massage, baths and strychnine are useful in the later stages.

HEART FAILURE.—The ice-bag is indicated for stormy heart action; ether, iron, valerian, oxygen, alcohol, digitalis, strophanthus, coffee, aromatic wines, camphor and strychnine should be given. For severe abdominal pain and vomiting, usually an evidence of cardiac failure, the above remedies, and champagne with menthol and carbolic acid, are indicated. The heart must be watched far into apparently complete convalescence.

NEPHRITIS.—Nephritis cannot be prevented. Alcohol, tincture of ferric chloride, beef tea and eggs are to be avoided; vichy, cardiants, milk diet, baths, laxatives, and care of the skin are advisable.

LOCAL DIPHTHERIA.—Two per cent. nitrate of silver in balsam of Peru affords relief in vaginitis; ice applications, atropia, and silver in ophthalmia; over inflamed glands, a 10 per cent. ichthyol salve may be applied; local diphtheria necessitates antitoxin, independently of its location.

CROUP.—Mercurial inunctions are given, together with steam sprays or tents and ice locally applied; for emergencies, the hot water may be turned on in the bath-room, after closing the door (Jacobi); or the child may be held over a pail of water into which live coals are thrown; emetics may be administered, with which wine (Stricker) is given because emetics often fail in carbon-dioxide narcosis; retraction of the chest walls, suffocation, cyanosis, and cold extremities, necessitate intubation or tracheotomy.

O'DWYER'S INTUBATION.—*Advantages.*—It is bloodless, is shorter, easier, and quicker than tracheotomy; there is less wound infection, and it is less objectionable to the family.

Disadvantages.—Membrane may rise beneath the tube, and tracheotomy may therefore be necessary after intubation. The tube may sometimes produce trauma, false passages, necrosis, ulcers, perichondritis and stricture. The tube is usually replaced two, three, or four times, and rarely need remain more than five or six days when the serum is used. It is removed by the extubator, by pressure, or by a string, which is necessary in private practice. Size of the tube: 5 to 6 millimeters, one to two years; 6 to 7 millimeters, two to four years; 7 to 8 millimeters, four to six years; 8 to 9 millimeters, six to eight years.

TRACHEOTOMY.—Tracheotomy is indicated when the heart is weak or when great asphyxia, much secretion, or gangrenous, diffuse, or septic infiltration or œdema of the lungs exist. In the after-treatment, the patient requires a moist atmosphere, and cleaning of the tracheotomy tube every second day, is important. The tube should not be permanently removed until all stenotic symptoms and all plaques disappear. In cases treated by serotherapy, this may be done on the fourth day as against the sixth or seventh in cases treated before antitoxin was introduced. The prognosis of tracheotomy depends upon possible coincident wound-infection with erysipelas or diphtheria, phlegmon, thrombophlebitis, mediastinal suppuration, ulceration of trachea, inhalation pneumonia, cardiac collapse, and nephritis. Rapid rise of temperature is ominous. Antitoxin has reduced the mortality from 66 per cent. to 35 per cent. (or 25 per cent.).

CEREBROSPINAL FEVER.

Cerebrospinal fever (epidemic meningitis), is an independent infection, but will be considered under diseases of the meninges, with the tuberculous and suppurative forms.

INFLUENZA (LA GRIPPE).

Definition.—A specific infective disease, caused by the Bacillus influenzæ, propagated by direct human intercourse but also by means of fomites; endemic over the entire world and almost yearly epidemic somewhere, characterized by its unparalleled rapidity in extension, the large number of people attacked, its protean symptomatology and its particular affinity for the respiratory tract.

Varieties.—(1) Influenza vera, (*a*) pandemic; (*b*) endemic or epidemic, due to Pfeiffer's bacillus. (2) Influenza notha s. nostras, pseudogrippe, catarrhal fever of unknown bacteriology.

Pandemics originate in Asiatic Russia. No other pandemic has ever been so widely distributed as influenza, which spreads very rapidly but not faster than the fastest trains and steamers. The rapidity of its dissemination is due (1) to the enormous virulence of the virus; (2) to the ease with which the germ is spread; (3) to the universal susceptibility; (4) to the short incubation period (one to three days), and (5) to the fact that a great number of moderately sick individuals frequent cars, conveyances, churches, schools, and places of business. Epidemics develop gradually, usually following pandemics; they occur every few years (pandemics are separated by decades), last one to two months, are less severe because of lessened virulence of the virus and increasing immunity. According to Hirsch, the first epidemic occurred in 1173. The first pandemic seems to have been in 1510.

Etiology.—Though no age confers immunity, most cases occur between the twentieth and fortieth years and especially in robust individuals. Physicians are very often attacked. The immunity usually conferred by one attack is less enduring and absolute than in smallpox, and other infectious diseases.

Bacteriology.—The organism discovered by Pfeiffer (1891–2) is the smallest bacillus which has been cultivated. Its ends are rounded and it resembles a diplococcus when two bacilli lie end to end. It has no capsule, is non-motile, it lies free in the sputum in the early stages and later is found in the leukocytes, but often disappears early from the sputum. The bacilli lie in lines "like schools of fish" (Finckler). Ten minutes are necessary for staining with carbol fuchsin. The bacillus is aërobic, short-lived, and very susceptible to drying. Culturally it grows characteristically on blood-serum as closely compressed yet discrete colonies, clear as water, and almost microscopic in size; it grows at body temperature and is easily overgrown by saprophytes. The germ has also been found in rare instances in the blood, in pneumonic and encephalitic foci, in the peritoneum, spinal fluid, gall-bladder, heart, spleen and kidney, and possesses rather marked pyogenic characters; it is often associated with the pneumococcus and streptococcus. Its *atrium* is probably through the upper respiratory tract although there are instances of primary pneumonia. The nervous and alimentary types are probably likewise explained.

Symptoms.—It is sometimes said that there is no uniform clinical picture of influenza and certainly there is scarcely an acute infection with such varied grouping of symptoms and such varied sequels and complications.

After a *short incubation*, which never exceeds one to three days, the grippe begins suddenly with chill and fever, headache, usually frontal, great anorexia and depression, pains in the back and legs, respiratory catarrh, which is frequently followed by capillary bronchitis or pneumonia in children or the aged, and intense nervous and sometimes digestive symptoms. Lichtenstern *classifies* influenza as follows:

(*A*). **Purely Toxic Type.**—(1) Simple influenzal fever; fever with its symptoms, headache, etc. (2) Nervous form; fever, pains in the back, legs and head, neuralgias, and prostration.

(*B*). **Toxic Inflammatory Type.**—(1) Respiratory catarrh of the nose, throat, larynx, trachea and bronchi. Respiratory symptoms are absent only in 25 per cent. of cases, a fact not recognized before 1889–1890. (2) Gastro-intestinal catarrh, coated tongue, anorexia, vomiting and diarrhœa.

The *duration* is short (one to three days) and the diagnosis is usually easy without the detection of the bacillus. Recurrences are frequent. Nervous symptoms in the primary attack may be followed by respiratory complications in the recurrence, or, more frequently, an initial respiratory catarrh is seen with nervous manifestations in the recrudescence. In rare instances influenza may cause chronic symptoms, chiefly respiratory. Convalescence is marked by great nervous depression, neuralgias, and tardy return of vigor.

Special Symptoms.—1. **Fever.**—The rise is usually sudden to 103° or 104°; but on exceptional occasions it rises gradually. It is entirely atypical. Remittent fever is more common than continuous fever, which may suggest typhoid. It is occasionally intermittent, resembling malaria. Hyperpyrexia is observed particularly in encephalitis grippalis, but again some of these cases and certain nervous forms are afebrile (Eichhorst). The typus inversus has been noted (Pribrâm). The fever lasts from one to three or four days, it is often shorter than the other symptoms and rarely lasts a week. Sometimes the fever returns after two or three days, constituting a recurrence or relapse. Fever may last longer as a result of complications. Often there is a remarkable loss in weight.

2. **Nervous System.**—The nervous system is almost always involved and is second in importance only to the respiratory tract. The most frequent disturbances are functional and psychical, but gross anatomical lesions are sometimes found. Headache is observed in nearly 100 per cent. of the cases. It is especially frontal and often very severe. *Influenza comatosa* results from very acute intoxication, which may cause epileptiform seizures, tremors, or severe delirium. The post-influenzal *psychoses*, acute and chronic, are due to cerebral toxæmia or exhaustion and are observed five to eight times as frequently as in any other acute disease. An hereditary tendency is observed in 62 per cent., and most cases are seen in the young. They may occur from the initial intoxication but usually follow the attack. The majority are of the hypochondriacal type (exhaustion psychoses), but mania is sometimes seen. Suicides were increased 25 per cent. in Paris during the last pandemic. In *encephalitis grippalis*, of which twelve cases are recorded, there are acute hemorrhagic foci of inflammation (Virchow, Senator, Furbringer, Konigsdorf, Schmidt, Oppenheim), which are usually discrete (sometimes confluent) and multiple; they almost always occur in the gray substance of the cortex or ganglia and vary in size from the "flea-bite" spots of inflammatory hemorrhage with surrounding softening to the size of a cherry or pigeon's egg. The inflammation is due to embolism by the Bacillus influenzæ (Pfuhl and Nauwerk) or to encephalitis suppurativa, which in turn is

either primary or secondary to foci in the nose, ear, and lung. Sometimes concomitant venous thrombosis, hemorrhage, pachy- or leptomeningitis occur. Cortical focal symptoms (mono- and hemiplegia) or a sudden apoplectiform onset may mark the process, or there may be diffuse cerebral symptoms at the beginning or acme of the disease, without definite focal localization. Encephalitis is usually fatal but recovery is recorded. *Meningitis grippalis* generally develops early. The virus reaches the brain by the blood stream or by contiguity through the *basis cranii.* In localization it is basal or cortical. Lumbar puncture may show the grippal bacillus, which may distinguish this from other forms of meningitis and from toxæmia (pseudomeningitis or meningismus). Mya (1904) reported three cases of suppurative meningitis in which recovery occurred; the bacillus was found by lumbar puncture. *Spinal complications,* such as transverse myelitis, hemorrhagic disseminated myelitis, Landry's paralysis, and acute poliomyelitis, have been observed clinically though investigated but little pathologically. *Neuritis,* local or general, is usually post-influenzal; nuclear (spinal) changes are sometimes suggested. *Pains* are frequent in the back, legs and joints; *neuralgia* may affect any nerve, but most often the fifth, sciatic, and intercostals; neurasthenia, which is often hypochondriacal, and hysteria may be observed as sequels.

3. The Respiratory Tract.—This is the entrance for the bacillus and is involved in 75 per cent. of cases, either entirely or in one part, as is the case in such inflammations as coryza and bronchitis.

Epistaxis is quite uncommon, though coryza, with reddening of the eyes, is seen in 70 per cent. of cases, and may possibly be accompanied by involvement of the frontal or other sinuses, or even empyema or neuralgia of the same. The bacillus may be found in the conjunctiva.

Angina is seen in 33 per cent. and *tonsillitis* in 60 per cent. of cases.

Laryngitis (6 to 16 per cent.) occurs with hoarseness. Fibrinous deposit or submucous abscess has occurred.

Tracheitis (50 to 80 per cent.) causes pain, tenderness and severe paroxysmal cough, induced by irritation at the bifurcation.

Bronchitis is either diffuse, developing anteriorly as well as posteriorly (Kundrat), or more characteristically, occurs over one lung or one lobe. At autopsy are found great congestion (which is the prime lesion according to Klebs), a tendency to extension, hemorrhage, round-cell infiltration into the mucosa, and exudation and thrombosis in the smaller vessels, which causes necrosis of the mucosa. The sputum, according to Pfeiffer, is characteristically nummular and greenish-yellow. It is often blood-stained, contains the bacillus, is viscid and is often abundant. Rales are most often found above the liver and near the lingual lobe. We also may observe dyspnœa, which is nervous (Graves) or congestive (Biermer) in origin, without appropriate physical findings. Cyanosis may develop, and is particularly dangerous in the young, aged and decrepit.

Acute *bronchiectasis* is not uncommon, but usually escapes clinical recognition.

Influenzal *pneumonia* occurs in from 5 to 8 per cent. of the cases and is the most important complication. Pfeiffer's bacillus is its most frequent cause, according to Leichenstern and Wassermann. The pneumococcus, streptococcus, and staphylococcus, alone or combined with the Bacillus influenzæ, are the cause of some cases.

Genuine *lobar pneumonia* undoubtedly occurs, and a grippe epidemic may double the number of lobar pneumonia cases. When it complicates grippe it is said to be less completely lobar and less clearly granular.

Influenzal pneumonia, strictly speaking, is exclusively *catarrhal* (Pfeiffer, Back, Wassermann), and shows no fibrinous exudation or granulations, but merely infiltration of cells, which commences about the bronchi as multiple distinct foci with normal lung between them; (the *lobular* foci sometimes fuse into secondary lobar pneumonia). It often causes minute abscesses. There is another type like the "acute lobar desquamative pneumonia" of Buhl, and still another, the "acute primary streptococcus pneumonia" of Finkler, which is lobular and catarrhal. Uncertainty as to the exact type may exist, but as Ziegler, Sahli, Kundrat and Weichselbaum have shown, mixed infection is of great importance. Clinically, the pneumonia is usually *atypical* and begins insidiously without a chill, but with increase of the prior cough and dyspnœa. It develops during or after the influenzal attack, although pneumonia may be the first sign of influenzal infection. Its symptoms are more pronounced than its physical signs, and the disproportionate cyanosis and dyspnœa sometimes suggest miliary tuberculosis. Foci of dulness may be found but distinct bronchial breathing is rare; bronchophony is a much more reliable symptom. The heart is often weak and a peculiar redness of the face and head, profuse sweats at the beginning, and characteristic paroxysmal coughing are symptoms not seen in ordinary pneumonia. The foci may be bilateral or multiple (60 per cent. of the cases) and the apices are involved very frequently. The symptoms and signs may recur or intermit as the consolidation wanders from place to place. The sputum is purulent or bloody, but the type of pneumonia is not determined either from the sputum or from its bacteriology. Influenzal pneumonia may result: (1) In solution by lysis; (2) in death in the first stage of congestion from œdema pulmonum; the patient may die later, gray hepatization having set in within three days; the mortality is 20 to 30 per cent.; (3) in chronic influenza pneumonia, with hectic fever and suppuration; *e. g.*, in an upper lobe (Graves, 1837); it may simulate tuberculosis; during a grippal epidemic, an increased mortality from tuberculosis is noticed; (4) in abscess or gangrene of lung .

Pleurisy may follow pulmonary lesions or even the bronchitis (Kundrat). Primary pleurisy occurs in 27 per cent. (12 to 33 per cent.) of the fatal cases of influenza. It may begin early and its symptoms are frequently severe and protracted.

4. Circulation.—The heart-muscle is directly injured, especially in its nervous apparatus. The lesser circulation is most involved. Weakness of contraction, syncope, even death, may occur during convalescence, especially when there are preëxisting fatty, valvular, or arteriosclerotic changes. The pulse is labile, often arrhythmic or dicrotic (Pribrâm); it may be rapid, and out of proportion to the fever; cyanosis, dyspnœa and bronchiolitis are frequent in such cases. A slow pulse is very frequent (45 to 50 per cent.) in severer types and is caused by toxæmic stimulation of the vagus. Acute endocarditis resulting from mixed infection sometimes follows suppurating foci in the lungs; three cases are recorded in which the influenza bacillus was found. Pericarditis is a sequence of other lung or heart disease. Acute phlebitis is seen mostly in severe cases, when the heart is weak (cardiac type), and occurs most frequently in the upper extremities. Arteritis is rare, is embolic or primary, occurs mostly in the popliteal vessels, and may end in gangrene. The existence of leukocytosis is denied by some writers; it occurs in not more than a third of the cases. Blood disintegration and hemorrhages into the skin, serous and mucous membranes, usually indicate severe intoxication.

5. Alimentary Tract.—Digestive symptoms are not especially important. The tongue is coated but rarely dry or typhoidal. *Fetor ex ore* and vomiting (34 per cent.) which is sometimes bilious and persistent, and grippal hyperæsthesia of the stomach may be observed. Constipation is more frequent than diarrhœa. Typhoidal symptoms may occur (see DIFFERENTIATION). There are the usual febrile degenerative changes in the liver. Icterus is rare (2 per cent.). The spleen is enlarged clinically in but 15 per cent. of cases.

6. Genito-Urinary Tract.—Albuminuria (under 10 per cent.) may result from toxæmia or stasis. Altered red blood-cells in the urine are rather frequent. Acute glomerulonephritis occurs in 1 per cent. of cases. Diabetes mellitus is said to have followed influenza; if influenza complicates an old diabetes, the prognosis is doubtful or unfavorable. Hayem found urobilinuria almost constantly. Senator noted the frequent occurrence of the diazo reaction and of indicanuria. Cystitis, hæmaturia, menorrhagia, abortion and orchitis are but occasional complications.

7. Skin, Muscles, Joints.—Urticaria, herpes (5 to 25 per cent. of cases), redness and hyperidrosis (vasomotor paresis), miliaria, erythema nodosum or multiforme, scarlatiniform or morbilliform eruptions on the face or chest, purpura, erysipelas, and pemphigus may be noted. Skin eruptions occur in 16 per cent. of the cases. Polysynovitis (rheumatoid form 7 per cent.), pyarthrosis or myositis are not common; bone troubles are not important and are infrequent.

Eye involvement (7 per cent.), conjunctivitis, keratitis, herpes, pain in the eye-ball, swelling of the lids, and otitis media (30 to 40 per cent.), which is usually purulent, and often hemorrhagic, are due to the pyogenic cocci, pneumococci and the influenza bacillus.

Diagnosis.—At the time of an epidemic the diagnosis is usually easy. Respiratory catarrh, headache, pains in the joints and back, nervous depression and tardy convalescence are significant. The diagnosis of the toxic, nervous and alimentary forms is more difficult and many errors may be made. The importance of bacteriological diagnosis has been over-rated, but positive results are decisive.

Differentiation.—**Typhoid Fever.**—The diseases may be confused when atypical severe influenza begins with gradual, step-like rise of the fever and typhoid symptoms, such as tympany, enteritis, colic, peritoneal manifestations, intestinal hemorrhage, swelling of the spleen, and roseolæ. At autopsy, swelling and even ulceration of the stomach mucosa and Peyer's patches occur (Jürgens). However, coryza, conjunctivitis, the existence of an epidemic and early catarrhal symptoms, indicate influenza, and the serum test and other cardinal typhoid findings (*q.v.*) decide the diagnosis.

Simple Coryza, Bronchitis, etc.—These are distinguished from true influenza by the sudden rise of fever in influenza, its nervous manifestations, pains in the head, bacteriology, spleen and exanthems. Lord found the bacillus of Pfeiffer in 30 per cent. of bronchitis cases in Boston, in which there was no suspicion of influenza.

Measles.—The early diffuse respiratory catarrh in each disease may cause confusion, and doubts may be increased by measles-like eruptions in influenza, but the typical measles fever curve (the fever rising again, after the prodromes, with the exanthem), and Koplik's spots, determine the diagnosis on the third or fourth day.

Miliary Tuberculosis.—This is suggested only in severe respiratory types; the history is usually distinctive.

Meningitis.—Influenza may masquerade as a pseudomeningitis, but focal signs are usually absent and doubts are settled by continued watching and lumbar puncture.

Apoplexy, the acute psychoses and acute poisoning are rarely simulated by influenza.

Prognosis.—(1) The morbidity is enormous, reaching 50 or even 75 per cent. of the population (pandemic of 1889-1890). (2) The mortality is $\frac{1}{10}$ to 1 per cent., but statistics are unreliable since the diagnosis of influenza is often carelessly made. It is certain that influenza increases the total number of deaths from pneumonia and tuberculosis, especially in the weak and aged. In some cases the course is chronic, the bacillus being found in the sputum for weeks or months, especially in tuberculous subjects, who are remarkably susceptible.

Treatment.—There is scarcely any *prophylaxis* except that persons should avoid crowds. Isolation should be practised in asylums, hospitals and prisons. Quinine is thought valuable, but is very uncertain. Gargles and nasal douches may possibly prevent infection and are distinctly indicated in grippal patients. There is *no specific treatment* and symptoms must be met as they arise. In every case, influenza should be treated as a serious disease and complications should be avoided by means of rest in bed and restricted diet. The treatment is much like that of a severe cold.

1. Pain.—At the onset, Dover's powder and antipyrin āā grains x, at once, are given for pain. Antipyrin has been used to excess, but is superior to acetanilid and phenacetin. If these measures do not relieve the pain, the salicylates should be used as they are in rheumatism. Cold baths for high fever are poorly tolerated by the patient.

2. Catarrhal Symptoms.—In conjunctivitis, boric acid solution is excellent; in coryza, douches with Dobelle's solution, tincture of belladonna and of aconite in two- and one-drop doses, respectively, should be used every half hour for about three hours, and the alkaloid cocaine gr. j to albolene ʒj should be used locally; in pharyngitis, silver nitrate (20 per cent.) should be applied once or twice; in tonsillitis, nitrate of silver should be introduced into the follicles which are first opened and touched with Dobelle's solution; for bronchitis (*q. v.*), codeine or morphine is indicated; for enteritis, bismuth and paregoric āā ʒj should be given after each bowel movement.

3. Severe Nervous Symptoms.—These are somewhat relieved by warm baths. Neuralgia necessitates the use of antipyrin, gelsemium, quinine, camphor, iron and arsenic. Headache is treated as it is in typhoid; it is increased by alcohol.

4. Heart.—The heart may require strychnine.

5. Convalescence.—Care in convalescence is important; cases of chronic tuberculosis or bronchitis require high altitudes.

PERTUSSIS (WHOOPING-COUGH).

Definition.—A specific, frequently epidemic, infection of the upp air-passages, occurring chiefly in children, and characterized by cyclic course and a severe convulsive cough, which ends in a lon drawn, spasmodic inspiration or whoop.

History.—Pertussis was described by Ballonius (1578) and Wil (1674).

Etiology.—(*a*) **Age.**—It usually occurs in children and is m frequent at the fourth year (seven months to seven years). It is e tremely infrequent after the twentieth year, though cases do occur middle and advanced life.

(*b*) **Sex.**—Sixty-six per cent. of cases occur in girls, females bein more inclined to spasmodic diseases. (*c*) *Anæmia* and respirato catarrh are predisposing factors. (*d*) Most cases occur in March an April.

Pertussis is clearly an infection, since (1) the disease is directly infective in all stages; (2) Koplik, Affanassiew and Hensel have described a facultatively aërobic bacillus which has small rounded ends, is but little larger than the influenza bacillus, and which occurs in clumps; (3) the disease may occur in epidemics, which develop from sporadic cases; (4) one attack confers almost absolute immunity. *Incubation* lasts from two to five to eight days.

Symptoms.—1. Stage of Prodromes.—The *stadium prodromorum s. catarrhale* is characterized by headache, backache, photophobia, conjunctivitis, coryza, sneezing, angina, and by a cough which becomes dryer and harder toward the end of this stage. In a few cases there is moderate fever. The digestive tract is usually intact. This stage averages one week (one-half to two weeks), but is often shorter when marked epidemics prevail.

2. Convulsive Stage.—The *stadium convulsivum s. spasmodicum* dates from the first "whoop." The fever (aside from complications) ceases with the onset of this stage. The *seizures* are paroxysmal, convulsive, and are accompanied by dyspnœa and vomiting. As a rule the child is well except for the paroxysm, which has for an aura, tickling in the larynx or back of the sternum; thoracic constriction, vertigo, or a creeping sensation, when the child braces himself, and terrified, runs for support. Then the explosion comes in the form of three to ten or more short expiratory coughs following in rapid succession until the breath is lost, when the "whoop" is heard as a long, rapid, deep, sharp, singing or whistling inspiration. Then a thin, tenacious and sometimes blood-tinged mucus is expectorated and vomiting constitutes the crisis of the paroxysm. There may be apnœa, great cyanosis, and usually diminution or suppression of the vesicular murmur. One to two minutes elapse between expiration and inspiration, and inspiratory and expiratory spasms or generalized convulsions may develop. The eyes are injected, the nose runs, the face is like that of an epileptic, the jugular veins are large and the skin is clammy. Involuntary evacuations occur; the pulse is small; ecchymoses sometimes occur in the conjunctivæ, scleræ, neck or face. After the attack, consciousness returns, respiration is fast, and there is fatigue, stupor, sweating, and pain in the abdominal or other muscles from the strain of coughing or occasionally from actual muscle rupture. The attacks occur largely at night, but especially toward morning. In the free interval there may be euphoria or great depression, stomach disturbance, anorexia, inanition, or diarrhœa. The urine has a gravity of 1,022 to 1,032, and may be pale and contain much uric acid. The laryngoscope sometimes reveals laryngeal or tracheal catarrh. Leukocytosis is observed early in the disease, is unusually marked, and the increase is chiefly in the lymphocytes (Meunier); Frank S. Churchill states that lymphocytosis occurs in 85 per cent. of cases, and in 90 per cent. during the catarrhal stage; it also occurs in those diseases which require differentiation from pertussis. The number of the attacks averages about twenty; this may be increased by excessive or rapid eating and drinking, by nasal or psychical irritation, and by respiratory efforts, such as weeping. The average is lower in a warm climate.

3. Stage of Decline.—The *stadium decrementi* usually comes after three or four weeks, but may require months. The attacks become less frequent, and the appetite returns. This stage is longer in proportion as the case is mild. Its complications make it the most dangerous stage.

Course.—The course lasts one week for the incubation, one to two weeks for the catarrhal stage, three to six weeks for the spasmodic, and three weeks for the decline. It is modified by the following complications:

Complications.—I. Increase or Extension of the Usual Symptoms.—*Catarrhal inflammation* is common in the initial stage,—stomatitis, laryngitis, catarrhal diarrhœa (10 per cent.), and otitis media (10 per cent.). *Bronchitis* (bronchiolitis) is not common in the spasmodic stage. It may occur with fever, a pulse of 120 to 150, respirations of 30 to 50, irregularity of breathing, many rales, cyanosis and atelectasis. *Bronchopneumonia* is the most frequent and severe complication, occurring particularly in rhachitic children between the third and fifth years and is most ominous in younger subjects, especially those under the third year. It occurs most often in the convulsive period. The mortality is great, sometimes 20 to 25 per cent. of cases. The onset is gradual with fever of 100° to 102°, a pulse of 130 to 160, respirations of 30 to 80, with cyanosis and dyspnœa. An almost absolutely bad prognosis must be given in the very young. Sometimes the pneumonia greatly lessens or entirely prevents the spasms (Trousseau). *Lobar pneumonia* is much rarer. Exudative pleurisy is a rare but very severe complication, and is seen almost exclusively in older children or adults. Endocarditis, pericarditis, meningitis and nephritis are very infrequent. *Spasm of the glottis* rarely occurs in children before the fourth year and is mostly seen in those who are nervous or scrofulous. Its incidence is largely nocturnal, and it may cause death from asphyxia even in the lightest cases. *Convulsions*, especially in nervous and hydrocephalic children, may occur at any time, but are most frequent at the height of the apnœa, at the time of first teething, and in the second to fifth week of the disease. They are of the carpopedal type. The eyes are rolled upward and outward, the pupils are dilated, and coma follows the dyspnœa.

II. **Mechanical Complications.**—*Hemorrhages* may occur into the skin, conjunctivæ, nose (relieving the cerebral congestion following the cough), ears and throat. Hemorrhage may occur in the brain, resulting fatally, or causing spastic paraplegia. Detachment of the retina, because of hemorrhage, and extravasation of blood into the lungs, stomach, and intestine, are rare. There may be *œdema* of the eye-lids, anasarca, rupture of the tympanum, or rupture of the frenulum of the tongue, the ulceration of which is almost constant. *Vomiting* occurs at the end of the paroxysm as a mechanical crisis to the attack and is so constant that the disease was once thought to originate in the stomach. The development of chronic gastro-intestinal catarrh is infrequently promoted by stasis. Other mechanical complications are cardiac dilatation, pulmonary emphysema (less often interstitial than alveolar), bronchiectasis, pneumothorax, involuntary evacuations, prolapse of the rectum or uterus, aneurysm, muscular ruptures, hernias, rib fracture, and spinal deformity.

III. **Toxæmia.**—Marked toxæmia is rare. Parenchymatous degenerations are found in the fatal cases. Nephritis is not common.

Sequels.—The hysterical coughing and spasm which sometimes follow pertussis may very closely resemble it; rhachitis is sometimes a sequel; tuberculosis may involve the lungs or peritoneal or mediastinal lymph glands, and this sometimes produces retrosternal dulness; it occurs especially after pertussis with measles, both of which infections predispose to tuberculosis. Preëxisting tuberculosis renders the prognosis of whooping-cough very unfavorable. It develops in the third stadium and in most cases between the sixth and tenth years of age.. Landry's ascending paralysis has been observed. The heart-muscle may suffer permanent weakness.

Diagnosis.—Diagnosis is easily made (1) by the definite stages of the disease; (2) by the presence of an epidemic; (3) by the absolutely characteristic convulsive seizures; (4) by stigmata, such as sublingual ulceration, swollen upper eye-lids, œdema of the face, petechiæ, and tender areas in the larynx; (5) pressing the tongue down and back with a spoon often elicits a characteristic attack of coughing. The stridor may be absent in nurslings and adults. The diagnosis may be difficult in intervals between paroxysms in the early or late stages. Intercurrent—especially febrile—diseases, like pneumonia or measles, may render pertussis atypical.

Differentiation.—1. Laryngeal diseases with spasm. 2. Acute glottis œdema. 3. Croup. In the latter there is fever, aphonia, metallic inspiration, lividity and constancy of the symptoms; *i. e.*, no interval. 4. False croup, (laryngismus stridulus) beginning with acute catarrhal laryngotracheitis, occurs at night in healthy children. 5. An epidemic cough in babies (*tusssi fernia*) may occur epidemically, but has no "whoop." 6. Spasmodic cough from disease in the bronchial glands. 7. "Ictus laryngeus" (vertigo or epilepsia laryngea), occurs in asthma, gout, tabes or laryngeal disease, and is recognized by burning in the larynx, vertigo and falling, as in apoplexy (ictus). 8. *Spasmus glottitis* occurs in nervous children at dentition, in cases of rhachitis and tetany; it is a sudden spasmodic closure of the glottis without prodromes, but with dyspnœa, cyanosis and convulsions. 9. *Asthma periodicum* (Millar's asthma) may develop in healthy children from the second to the sixth year, with hoarseness, moderate fever, dyspnœa on the second day, with convulsions, cyanosis, and repeated seizures. Recovery occurs, or death, within the first eight days of the disease. 10. *Hysterical "after"-pertussis* is characterized by the absence of catarrhal symptoms, fever, vomiting and the stigmata of pertussis; *i. e.*, sublingual ulceration, darkly colored upper eye-lids, œdema of the face, petechiæ and the tender point in the larynx, irritation of which induces an attack. Hysterical imitation is associated with the stigmata of hysteria (*q. v.*), the influence of suggestion and a longer course than that of pertussis proper.

Prognosis.—Whooping-cough, with its complications, is a very fatal disease, and ranks third in the English vital statistics for children. Hirsch states that, in Prussia, 85,000 deaths occurred from pertussis from 1875 to 1880, and in England, from 1857 to 1867, 120,000 died from the disease. The mortality averages 3 per cent., but varies with the virulence of the epidemic, and may even reach 15 per cent. The longer the time of development of the convulsive stage, the better is the prognosis, and the clearer are the intervals between paroxysms. The outlook is more unfavorable in girls, in the poor, in nurslings and in children under five years, especially in colored children. The mortality of cases in the first year is about 27 per cent., in the second year 14 per cent.; from the second to the fifth year, 3 per cent.; from the fifth to fifteenth year, 1.8 per cent. (Hagenbach); Toplitz found 41.7 per cent. of pertussis deaths in children under one year, and 48 per cent. in children between one and two years of age. According to Trousseau, when more than forty attacks occur in one day, a grave prognosis is indicated and when over 60 attacks occur a fatal outcome is certain. The writer, however, has seen recovery in cases with over 100 paroxysms daily. Coincident measles, pneumonia or bronchitis, especially if of early development, weak heart, purpura, continued emesis, glottis spasm, eclampsia, sinus thrombosis, skin emphysema, encephalitis, brain hemorrhage, asphyxia, and the sequels of scrofula and tuberculosis, render the prognosis very uncertain.

Treatment.—1. **Prophylaxis.**—Isolation is difficult, but is indicated, particularly in tuberculous children. It may be thought best to expose a child to a mild epidemic. The greatest danger exists during the catarrhal stage, although the coughing period is not devoid of it. The sputum, and the washing of clothes, etc., are cared for on antiseptic principles. It was thought that vaccination mitigated or aborted pertussis (Archer), but in eleven personal cases, which had never been vaccinated, vaccinia had no prophylactic effect.

2. **Medication.**—There is no specific, as proved by the much-vaunted resolvents, emetics, demulcents, expectorants, antispasmodics, narcotics, tonics, and antiseptics. All drugs should be employed cautiously. The following are of value: *Quinine*, the foremost remedy was first introduced by Binz, who gave 1½ times as many grains *per diem* as the child was years old; to children under two years, he gave ⅙ grain for each month. Children object to the taste of quinine less than do adults.

Widerhofer's formula runs as follows:

℞

Extr. belladonnæ	gr. iss.
Quininæ sulph.	gr. viij.
Sacchari albi	gr. xxx.

M. et divide in pulv. no. X.
S.—One powder three times daily.

Camphor, recommended by Jacobi, is a stimulant and may be indicated in asthenia and in increasing bronchitis, ¾ grain at a *dose*.

Antipyrin sometimes decreases the number and severity of the attacks; ⅙ grain for each month or a decigram (1.5 grains) for each year is given, which may be gradually doubled. *Bromine* should be administered as bromide or *bromoform* (Stepp). It is not curative, but moderates the course of the disease. Bromoform should be kept bottled in the dark and should be administered fresh from the stock solution each time: one to five minims in simple syrup.

The *catarrh* may be treated with expectorants and alkaline drinks. A steaming kettle will somewhat alleviate bronchitis, the child being kept in bed and a tent constructed over the bed. Intralaryngeal insufflations of resorcin (1 per cent. with starch) are recommended. Blenorrhœa should be treated with benzoic and tannic acids (āā gr. ij, t. i. d.).

For the *paroxysm* there is no specific. *Belladonna* is given by Jacobi in the form of the extract, one-sixth grain to a child of six to eight months, until flushing is observed. Symptomatically it is also useful for the clammy, cool skin and for collapse symptoms. Though children tolerate belladonna well, great care must be exercised with regard to the very young. The same caution is necessary with ipecac, chloral and Henoch's combination of belladonna and opium.

Chloroform inhalations are dangerous. *Codeine* is less effective than *morphia*. *Chloroform* (Schilling) may be given with advantage in dry coughs, and twice as many drops may be administered as the child is years old. It should be given in one dram of warm water. It is said that the paroxysm may be averted by pulling the lower jaw downward and forward.

Yeo recommends the following formulæ:

℞

Morphinæ hydrochloridi	gr. ¼.
Sodii bromidi	ʒss.
Aquæ laurocerasi	ʒij.
Aquæ chloroformi	ad ℥jss.

Misce, fiat mistura.

S.—One to three teaspoonfuls for a dose, according to the age of the child.

℞

Chloral hydratis	gr. xvj.
Sodii bromidi	gr. xxxij.
Aquæ chloroformi	ad ℥ij.

Misce. fiat mistura.

S.—One to four teaspoonfuls for a dose, according to age.

℞

Sodii benzoatis	gr. lxxij.
Sodii bicarbonatis	gr. xlviij.
Ammonii chloridi	gr. xxiv.
Aquæ chloroformi	℥j.
Aquæ anisi	ad ℥iij.

Misce, fiat mistura.

S.—One to four teaspoonfuls, according to the age of the child, in a little hot milk, every four hours.

Digitalis is used by Koplik and Knight for right-heart dilatation. O'Dwyer recommends *intubation* for the spasm of the glottis. This is open to the criticism that ulceration might occur from the long-continued pressure in the larynx. *Bronchopneumonia* (*q. v.*).

3. **Hygiene.**—In warm climates pertussis is rare and runs a mild course. A suitable climate is likewise prophylactic against tuberculosis. The attacks are often lessened, or the disease quickly ended, by removal to the country. Moderate exercise is indicated. Such irritants as beef tea or stimulants, dry bread, cookies (Vogel), and overfeeding or drinking, should be avoided, because they provoke coughing and vomiting in the convulsive stage. Food should be given at frequent intervals in concentrated form—gruels, milk with lime-water, zwieback in milk, eggs, malt, meat-juice, etc. Older patients can tolerate more solid foods. Rectal feeding and the use of the nasal catheter are sometimes of advantage.

EPIDEMIC PAROTITIS (MUMPS).

Definition.—An acute specific infection, contagious, febrile, often epidemic, and characterized by a primary inflammation of the parotid glands.

History.—Parotitis epidemica was known to Hippocrates, who described every essential point, but later it was confused with other swellings of the parotids and other diseases. Its distribution is world-wide.

Etiology.—(*a*) Its precise cause is unknown, although various micrococci and diplococci have been described. (*b*) Most cases occur between six and fifteen years of age, although isolated cases are seen in adults and the aged. (*c*) The sexes are about equally involved. (*d*) More cases occur in the cold, moist, spring months than in the summer. (*e*) Contagion is more frequently direct from person to person than indirect by a third party or by fomites. (*f*) It prevails largely in epidemics, although sporadic cases are occasionally seen. (*g*) Immunity is, as a rule, conferred by one attack. It rarely prevails with scarlatina, varicella, measles or influenza.

Symptoms.—After an *incubation* of two and a half to three weeks, the disease usually begins with fever, which is followed in a few days by *parotid intumescence and pain*. In some cases prodromes (slight fever, chilliness, and redness of the throat) may exist for thirty-six to forty-eight hours before actual fever and parotitis develop. In other instances the severity of the epidemic determines such severe initial symptoms as vomiting, convulsions, or facial spasm in children; or delirium, prostration, diarrhœa, or a severe typhoidal condition in adults. As a rule, the fever rises at the outset to 101° or even 104°, the pulse averages 100, and in two days the *characteristic parotitis* develops. Its signs are clearly inflammatory (swelling, pain, temperature, and often interference in function). The parotitis is usually bilateral, for the inflammation of one gland is rapidly followed by parotitis in the other. Indeed, Bouchoud said: "Mumps have no singular"; but in some epidemics the parotitis is unilateral (Laveran). Its degree varies and sometimes there is partial swelling. Again it may be diffuse over the entire gland, or even beyond it. We know little of its pathology beyond what Virchow described—hyperæmia of the parenchyma, interstitial swelling, catarrhal secretion in the ducts and inflammation of the contiguous lymph-glands. The swelling about the gland may reach the eyelids, forehead, mastoid region, or even the lower neck and clavicle, making the features unrecognizable. It seems that the swelling is due less to periparotitis than to pressure on the facial and other veins. It is soft and does not pit. In rare instances stasis and œdema may be noted in the pharynx or even in the larynx and with obvious danger. This may explain the occasional epistaxis. Compression by the swollen glands of the masticatory muscles makes chewing or opening the mouth difficult; chewing also excites pain because it excites salivary secretion which often cannot pass the turgid ducts. Compression of the Eustachian tube or external meatus may occasion tinnitus. In the early unilateral involvement, the face inclines to the affected side in order to relax the swollen tissues; in the later bilateral parotitis, the head is often bent forward for the same purpose. The salivary secretion may be normal, increased or decreased. Suppuration is rare and is probably due to mixed infection. Leukocytosis is usually absent.

In severe cases the *submaxillary and sublingual* glands participate in the inflammation or swelling (even in 50 per cent. of some epidemics recorded); in atypical cases they may be the sole seat of the disease, and the parotids may be free from infection. The parotitic swelling reaches its acme in three or four days and an equal period is necessary for its subsidence. The *course* then is *one week*, or perhaps two. The fever throughout is remittent or intermittent, and falls by lysis in from four to seven days. One to two weeks are necessary for convalescence.

Complications and Sequels.—1. The most important are *orchitis* and epididymitis, which were described by Hippocrates. On the sixth to the ninth day fever returns (102° to 105°), sometimes with chills and local redness. Swelling and pain in one testicle develop, and are often accompanied by general symptoms. Orchitis usually follows the parotitis, whence it was formerly considered metastatic. This view is untenable, because, in some epidemics, orchitis precedes or even occurs without parotitis. We must then explain mumps as a general infection, the blood carrying the virus first and most commonly to the parotid glands and later (and even before or alone) to the testicle. A urethritis is sometimes observed, analogous to the catarrh of the parotid duct. It is sometimes held that this inflammation is the primary and *atrial* one. Testicular localization occurs almost wholly in the sexually mature and active testis and is therefore most rare in children or the aged. Orchitis or epididymitis is usually unilateral. It occurs in a variable percentage and depends on the character of the epidemic. Comby observed it in 30 per cent. of cases of mumps among French soldiers. Acute hydrocele is due to the epididymitis. The fever occasionally resolves by lysis, and suppuration from mixed infection is very exceptional. Testicular atrophy occurs in 44 per cent. (Gramers) to 63 per cent. of the cases (Comby) and if bilateral may occasion sterility. In women, ovaritis and vulvovaginitis are infrequent. Mastitis is rather more frequent, and may be observed in men and boys.

2. *Other glandular organs* are less frequently inflamed. Of these the lachrymal glands are most often affected, then the thyroid, with symptoms of acute Graves's disease, pancreas (1.5 per cent., Simonin), and the thymus. The spleen is seldom swollen.

3. *Other complications* are very rare. These are severe delirium, meningitis, encephalitis, paralysis of the facial or other cranial nerves; polyneuritis, or the neuroses; labyrinthine or middle-ear disease, eye inflammations, optic neuritis; endocarditis; synovitis; albuminuria or nephritis; bronchitis or bronchopneumonia; necrosis, suppuration, or gangrene of the parotid or testis.

Diagnosis.—The diagnosis is usually easily made. Osteomyelitis of the jaw, periostitis, parulis, lymphadenitis, thrombo-phlebitis of the external jugular, and furuncle of the external ear, should be considered and excluded. Epidemic parotitis must not be confused with *secondary parotitis*, which results from trauma, or pus burrowing from the middle ear, or with mercurial, iodide or lead parotitis, and purulent metastasis in typhoid, pneumonia, and erysipelas, in which the pyogenic virus enters by the ducts or settles from the blood-current. In *post-operative parotitis*, after laparotomy, the saliva is decreased; anæsthesia first increases, then decreases the salivary flow; trauma to the gland, from holding up the jaw, may promote infection.

Prognosis.—The prognosis is usually good, for but 7 deaths occurred in 58,331 cases. The course is lighter in children than in adults. Atrophy and sterility may result from orchitis.

Treatment.—The patient should be isolated. The fever is controlled as in other fevers, and diet and hygiene are the same. Complications are treated *symptomatically*. The patient should be kept in *bed* for one and a half weeks. *Pain* often necessitates the use of opiates. The local parotid or testicular inflammation is self-limited, and resists all attempts at abortion or mitigation. *Local heat* is soothing and better tolerated than the ice-bag.

ACUTE ARTICULAR RHEUMATISM (RHEUMATIC FEVER).

Definition.—An acute, febrile, non-contagious disease, with multiple articular involvement and with a tendency to heart complications.

Frequency.—McCrae's statistics, from Johns Hopkins Hospital, show that rheumatism constitutes 2 per cent. of admissions, as against 3.5 to 7 per cent. in the London hospitals.

Etiology.—Though strict proof is lacking that rheumatism is an acute infection, the evidence is strongly presumptive. This is shown by its occasional yet undoubted epidemiology; its clinical picture; its tendency to recur like pneumonia and erysipelas; the autopsy findings of exudation, hemorrhage, cloudy swelling, and acute splenic tumor; the frequency with which a suitable atrium may be observed, such as an angina, which furthers the entrance of microörganisms into the system; and its indubitable close affiliations with such diseases as endocarditis, pericarditis, and meningitis, whose mycotic origin is clearly established. Guttmann and Sahli found staphylococci in the joints, kidneys, and pericardium, and Leyden discovered a diplococcus. Sahli early questioned the unity of the disease and is now inclined to regard it as a modified septicopyæmia. Birsch-Hirschfeld, Triboulet, Bouchard, Charrin, Singer, Alchame, and many others, have found bacteria not only in the joints but in the blood and viscera. If all observers found the same bacteria, conclusions would be more easily drawn, but the finding of streptococci, staphylococci, diplococci and bacilli creates confusion, and it must be stated that the *rheumatism virus is unknown.*

Of late the Micrococcus rheumaticus, described by Triboulet (1898) and Wassermann (1899), has attracted especial attention. It is claimed that it differs from the ordinary streptococcus in changing the color of agar serum media and in growing on filtered media in which the ordinary streptococcus has already grown (Marmorek's test); in growing best on very alkaline media and quickly secreting much acid. It is said to produce in the urine formic acid (which we find in rheumatism) and produces hemolysis (like the rheumatic anæmia). Singer likens rheumatism to septicopyæmia in that common symptoms occur in each—temperature, anæmia, articular metastases, endocarditis, leukocytosis, and peptonuria. He places particular stress upon the detection of pyogenic organisms in the rheumatic joints, in the initial angina, in endocarditis, in pneumonia, in erythema multiforme, in the various serous membranes involved, and in the urine. Chvostek and Egger explain the presence of these organisms by bacterial invasion of the tissues during the death agony, "an agonal invasion," since bacteria could not be obtained from aspiration of the joints during life. The rheumatic arthritis is sometimes explained by toxæmia rather than by bacteriæmia, analogous to the toxic pericarditis of uræmia, or the swelling of the articulations after antitoxin injections. Personally, the author can readily conceive of such a theory, inasmuch as he has repeatedly seen negative cultural results from aspirations of the synovitis of chronic nephritis, explained in some instances, at least, by retained toxins. Streptococcus or staphylococcus infections may produce a clinical picture resembling rheumatism or may indeed complicate it, but they are not found in the majority of cases of genuine rheumatism.

Some writers question the "unity" of acute articular rheumatism, because the clinical picture is not always the same. Age modifies the disease, as is shown by the predominance of chorea, pericarditis, and endocarditis in childhood, with striking synovial subordination. The "genius epidemicus" changes the symptomatology, the disease duration, the medicinal results, and the frequency of nervous, and endocardial complications, and of those of the serous membranes. The fact that the onset is accompanied by angina, enteritis, icterus, gastritis, bronchitis, and typhoid symptoms, is an argument against the theory of clinical independence of inflammatory rheumatism. However, no greater variations are noted than in other diseases,—*e. g.*, in typhoid.

Predisposing Factors.—(*a*) *Cold;* 75 per cent. of cases occur in the first half of the year (McCrae); (*b*) heredity (25 per cent.); (*c*) male sex (70 per cent.); (*d*) age, the greatest susceptibility exists between ten and thirty years (60 per cent., McCrae; 81 per cent., Damash); M. Miller collected nineteen cases in nurslings; (*e*) trauma, overexertion, exhaustion, exposure, and occupation, occurring frequently in sailors, divers, bakers, and day laborers.

General Clinical Picture and Course.—The onset is usually sudden, with moderate fever, redness of the skin, considerable prostration, drenching sweats and polyarthritis. The joints are involved in succession, the inflammation lasting a few days in one joint and then passing to many others, with frequent heart localization and the fever ending in solution by lysis. There is marked anæmia and loss of weight. If endocarditis occurs, valvular disease is the usual sequence. The usual duration without therapy is three to four weeks, though abortive forms occur. Recovery is usual, but death may result from hyperpyrexia and from brain toxæmia. A tendency to recur and a family tendency in rheumatism and valvular lesions are noted. The articular disease is directly influenced by the salicylates, but cardiac localizations are much less directly or not at all affected. In adults and adolescents there is febrile urine, constipation, conjunctival injection, coated, moist tongue, some irritability, restlessness, and fever which is renewed as fresh joints are involved.

Symptoms in Detail.—**1. Prodromal Symptoms.**—These are angina or tonsillitis (70 to 80 per cent., Garrod; 21 per cent., Gerhart; 5 per cent., Groedel and Reiss; 1.7 per cent., Pribrâm), regarding which there are two conceptions; (*a*) that the angina is primary and rheumatic, and (*b*) that the rheumatism following angina is pseudorheumatism (polyarthritis anginosa), characterized by exanthems, occurring chiefly in the arms and reacting poorly to salicylates. Mild gastric symptoms are considered by Damash as the most frequent prodrome. Other prodromes are laryngitis, otitis media, chorea, chlorosis, and erythema nodosum. Whether the prodromes are indefinite or wholly absent, or whether they are angina, albuminuria and fever, as Widal has often seen them, or enteric, as often observed in Vienna, any diagnostic doubts are settled by the appearance of the polyarthritis.

2. Polyarthritis.—(*a*) *Localization.*—The joints most frequently involved are those of the lower extremity, particularly the knee (53 to

84 per cent.); and usually one side is affected after the other. The ankles, elbows and wrists are the seats of the next most frequent localizations. Though usually found in the large joints, it may involve the smaller articulations, such as the fingers. Early arthritis of the small bones of the carpus was thought by Trousseau to promote endocarditis. Rheumatism may invade the spine, jaw, sternoclavicular joints, the synchondroses of the ribs, the sacroiliac or pubic articulations, and the larynx. Occupation has a bearing on the disease; for instance, rheumatism may occur in the arms of washerwomen. If a joint of an upper extremity is first involved, the opposite arm is usually next inflamed: (*b*) *In general* the arthritis is *ascending*, fleeting and almost invariably polyarticular (Senator). The characteristic erratic character of the arthritis is mentioned by the oldest writers, as Van Swieten, who spoke of it as "migratory inflammation" or "fleeting gout." Though usually fleeting in its nature, the virus may settle in one joint for a considerable period, but persistency in one joint always suggests pseudorheumatism (symptomatic or secondary forms). (*c*) *Signs and Symptoms.*—Polyarthritis is a better term than polysynovitis because the inflammation pervades all structures of the joint, the periarticular bursæ, cellular tissue, muscular insertions and tendons, and the external œdema is usually greater than the synovial effusion. Rattling over the joints is usually due to tendosynovitis. The joints are swollen, red, tender and painful. Pain is the most prominent and frequent joint finding. Lasègue attributes the pain not to the joint but to inflammation in the associated muscles and tendons, affirming that if these be relaxed voluntarily by the patient or suitably supported by the physician, the joint can be moved freely in all directions. Lasègue's phenomenon is said to be absent in gonorrhœal and surgical arthritis. The contiguous tendon reflexes are lessened or abolished. (*d*) *Duration.*—The joint inflammation remains in one joint from one to eight days, seldom longer, and its involution is shorter than its evolution. (*e*) *Sequels.*—The joints become quite normal; ankylosis is so infrequent that its occurrence usually reflects doubt upon a previous diagnosis of rheumatism. Suppuration is extremely exceptional, but may result from mixed infection and more frequently in children than in adults. Spontaneous dislocations are rarities.

3. **Temperature.**—The temperature is directly related to the intensity of infection, the number of articulations involved, and to the development and progression of visceral complications. As Wunderlich noted in his celebrated communication on fever, the temperature is usually highest when the patient is first examined. The fever rises with each new localization, be it arthritic, endocarditic, or pericarditic. Friedländer spoke of a short (monoleptic) and a long cycle (polyleptic), yet the fever curve is in no wise cyclic, beyond undulation with each new manifestation, and is rarely higher than 102° or 104°. The temperature is usually seen to drop when the sweats occur. Prodromal fever was early noted by Todd, Graves and Fuller. The

fever averages ten days, defervescence occurring spontaneously or with the salicylate therapy. The fever is longer when treatment is commenced late. Kahler held that fever could occur independently of joint inflammation (febris rheumatica). Pleuritis, renewed arthritis, angina, erythemata, embolic and other complications, prolong the pyrexia. Simple fixation of the joints reduces the fever, tachycardia and tachypnœa (Pribrâm). Chills suggest septicopyæmia rather than rheumatism. Hyperpyrexia is exceptional. The fever may, however, even reach 111° with severe nervous symptoms ("cerebral rheumatism"), coma, convulsions, rapid pulse, transitory paralysis, and stertorous breathing. 50 to 60 per cent. of cases of hyperpyrexia occur in the first attack of polyarthritis with lesser frequency in later seizures. It is less frequent since the introduction of the salicylates.

The pulse is accelerated and is usually said to exceed 100, although McCrae found that its rate was not over 100 in 59 per cent. of his cases. Dicrotism and arrhythmia may sometimes be noted before involvement of the joints. The respiration follows the fever; Pribrâm noted one case with 80 to 100 per minute.

4. Heart Complications.—These are almost an integral part of rheumatism and constitute *visceral rheumatism* as distinguished from articular rheumatism. Heredity seems to be a factor in certain cases. Heart lesions occur as frequently in light as in severe forms. They vary somewhat with the epidemic, are most frequent at the time of puberty when they are most dangerous, and increase in frequency with repeated attacks of rheumatism. Lasègue says: "Acute rheumatism licks the joints, the pleura, and even the meninges, but it bites the heart."

(A). ENDOCARDITIS.—This, the most vital complication, was first described by Pitcairn (1788), and Pidoux, Besnier and Homolle followed Bouillaud in developing the subject. Bouillaud (1832) formulated the statement that "in every severe, generalized case of acute, febrile rheumatism, endocarditis was the rule." At the present time many French writers hold the same view. Besnier and Widal maintain that in severe cases the "heart is diseased to some degree." The frequency of endocarditis is about 20 per cent. (Other figures are 50 per cent. Damash, Fuller, Latham; 40 per cent. Pribrâm; 20 to 28 per cent. Bamberger, Jaccoud, Oettinger.) Mackenzie finds endocarditis in 58 per cent. of first attacks, 63 per cent. of second attacks, and 71 per cent. of third attacks. In McCrae's series we might divide the cases into thirds; in one third, the heart is normal; in another third, it is doubtful; and in the last third, it is diseased. Hospital statistics are somewhat misleading, because they represent the severest cases. Clinically, endocarditis often escapes recognition, or is latent;—*i. e.*, it is found more frequent at the autopsy table than at the bedside. It is usually verrucose, and rarely ulcerative, even in the fatal cases. Accidental or "functional" murmurs must not be called endocarditis. To be certain that an endocarditis exists, two points are absolutely necessary: (1) Continued observation of the patient after convalescence; (2) attention to signs of valvular disease (*q. v.*), other than the mere murmur, such as dilatation, hypertrophy, and secondary pulmonic accentuation. The distinguished Trousseau called attention to the possibility of the functional murmur. Potain, almost alone among the present French writers, properly estimates its importance. The diagnosis of acute endocarditis must long be uncertain. According to Leube, functional murmurs are rare in rheumatism, though Senator considers them frequent; yet therapeutically we must keep on the side of extreme caution by keeping the doubtful case sufficiently long in bed. We may interpret the disappearance of a murmur in two ways,—first, as the disappearance of a functional murmur when the fever falls, the toxæmia disappears, the heart slows, and dilatation subsides, or the anæmia mends; or, second, as the healing of a slight endocarditis, though this is always the less probable issue.

Valves Affected.—McCrae finds the mitral valve involved in 95 per cent. of the cases, the aortic in 23 per cent., and both valves in 18 per cent. (Aortic lesions are usually estimated at 12 per cent.) The fever may be continuous with that of the arthritis but is often higher. Less often the fever is remittent with severe rigors and may require differentiation from malaria, pyæmia, or gonorrhœal endocarditis. A low remittent fever may suggest pulmonary or serous membrane tuberculosis. The endocarditis exceptionally antedates the polyarthritis (Jaccoud, Trousseau).

Masked or atypical rheumatism was known to Immermann, who spoke of rheumatism masquerading under the guise of an endocarditis or trifacial neuralgia and yielding to salicylate treatment. Kahler's opinion of rheumatic fever without arthritis has been mentioned. The rheumatic virus is claimed to sometimes produce pericarditis or endocarditis without articular inflammation—"polyarthritis rheumatica *sine* arthritide." We may call this the "rheumatic equivalent," borrowing from the nomenclature of epilepsy.

In old heart lesions with renewed rheumatism, lingering fever suggests the possibility of an exacerbation of the old endocarditis which may have been aroused by the last rheumatic attack. Various embolisms may occur. The bacteriological findings are most varied, and not knowing the actual cause of rheumatism, we cannot say exactly what is rheumatic and what is mixed infection. Many cultures are negative.

(B). PERICARDITIS.—Osler states that rheumatism explains more cases of pericarditis than all other causes combined. It is the most frequent lesion in children. Its frequency is variously given as 20 per cent. (Hache, Wunderlich, Leudet, Sibson); 54 to 70 per cent. (Williams, Omerod, Taylor); 13 per cent. (Chambers); 10 per cent. (Oettinger); 6 per cent. (McCrae), 5 per cent. (Pribrâm). Pericarditis is very frequently latent. In children, especially, a subacute or latent course ending in synechia is most common. Baginsky found that adhesive pericarditis was the most common autopsy finding in rheumatic children.

(C). MYOCARDITIS.—Disturbance of the heart in the acute stage is almost certainly myocardial. It may be toxic or mycotic. Acute interstitial myocarditis (in 28 per cent. Poynton), changes in the heart ganglia, coronary embolism or thrombosis, direct myocardial invasion by contiguity from endocarditis in the later stages, and sometimes the use of salicylates or antipyrin, are possible causes. Recovery is usual, but chronic fibrous myocarditis is a not uncommon sequel. Acute myocarditis may produce a systolic murmur, and confusion with early endocarditis is possible. Myocarditis may cause sudden heart pain with weakness, in which case the subject may die. This is the "heart rheumatism" of the laity. Transient palpitation, fugitive cardiac oppression, or tachycardia, due to pain or excitement, may occur. Bradycardia with vertigo and syncope is probably myocardial.

As a practical point, it must be remembered that the right heart may be dilated as in other fevers and as in various abdominal affections; this dilatation disappears with convalescence. When accompanied by a murmur it may lead to an incautious and wholly unjustified diagnosis of endocarditis. The bloodvessels are rarely inflamed, but coronary or peripheral endarteritis occasionally develops. French writers maintain that acute arteritis is common; severe pain, well localized over the artery, is followed by paræsthesia and pain over its smaller branches and in the extremities; pathologically only part of the vessel-wall is affected and healing usually follows, but gangrene may develop after complete arterial occlusion. Endophlebitis is somewhat more common.

5. Skin.—Sweating is most profuse, constant and characteristic; in odor it is sour or mouse-like (Schoenlein); in reaction it is first acid and later neutral or alkaline. This may result from decomposition or from large doses of alkalies. The skin is frequently macerated and sudamina are common, usually *sudamina alba* (in 4 per cent., *sudamina rubra*). *Rheumatic nodes* (first described by Hilliers, Meynert, Hirschsprung, Barlow and Warner) are subcutaneous nodes, as large as buckshot, and occur mostly on the fingers, hands and wrists, but also on the elbows and spine. They may appear during or after an attack, or independently of acute seizures. They occur especially in children, but are also observed in adults, and particularly in women. They are abundant, sometimes numbering even sixty, are frequently symmetrical, hard, and are usually tender. Sometimes osseous or arterial endothelial proliferation is observed. Nodes occur most often in chronic cases or in cases with severe endocarditis or pericarditis. If very numerous or large they indicate an unfavorable prognosis. In contradistinction to these more fixed nodosities, Féréol has described more fugitive and less indurated nodes, which are rather analogous to the fugitive polyarthritis (*nodositiés cutanées éphémères*). Œdema of the skin (Comby, Ferret, Guyon) may be septic, rheumatic, or angioneurotic. Erythema polymorphum is generally regarded as an independent affection but in rheumatism it does occur in the nodose or vesiculated forms. Some cases are possibly septic or anginose eruptions. The salicylates relieve some cases and fail to benefit others. Morbilliform or scarlatiniform rashes are infrequent, and may be attributed in part to errors in diagnosis, or to drug eruptions. Purpuric eruptions are not uncommon. The intestinal hemorrhage, colic, and vomiting, with articular intumescence, described by Henoch, are entirely distinct from genuine rheumatism and will be considered under Purpura (*q. v.*).

6. Muscles and Bones.—*Myositis* is infrequent, though *myalgia* is common. Muscular atrophy may follow inflammatory extension from the joints to the muscles. "Muscle scars" are not frequent, and are much more often due to trauma, to other infections, to septicopyæmia, polymyositis and dermatomyositis than to rheumatism, which rarely seems causal, although undoubtedly sometimes coincident. Cases of torticollis in children have recently been considered instances of cervical rheumatism. *Periostitis* may occur, and is thought by some authorities to be latent syphilis aroused by rheumatism. Some cases of albuminous periostitis are clearly rheumatic. Isolated instances of osteomyelitis (deforming rheumatism) are possibly rheumatic.

7. Respiratory Tract.—*Laryngitis* may be prodromal. It may be a possible atrium for a systemic invasion, or may occur later as diffuse catarrh or as local nodes. The rare acute œdema of the larynx is most dangerous. *Pleurisy* may result from the rheumatic virus even when it shows wholly negative bacteriological examinations or it may result from secondary infections. Its frequency is 2 to 3 per cent. (to 4 to 6 or 10 per cent.). Seé described two types (*a*) a violent and (*b*) a mild (latent) form, both of which usually run a favorable, rapid course, perhaps with considerable fever. A primary prearthritic pleurisy (Fiedler, Lasègue) is very rarely observed. Pleurisy and pneumonia are usually associated with heart disease. Pleurisy is found in 54 per cent. of cases of endopericarditis (Pribrâm). While the writer has occasionally seen cases of serous pleurisy in association with rheumatism, he believes that the so-called rheumatic pleurisy is almost always tuberculous (see ETIOLOGY OF PLEURISY). *Pneumonia* occurs in but one-tenth of 1 per cent. of cases of rheumatism not involving the heart, and in 66 per cent. of cases with endopericarditis. Howard (quoted by Osler) found lung and pleural complications in only 10 per cent. of cases with rheumatic endocarditis, in 58 per cent. of those with pericarditis, and in 71 per cent. of those with endopericarditis. These figures seem remarkably high, and without questioning their accuracy, it is certain that many cases are called pneumonia which are pulmonary infarction, stasis, or compression from pleural or pericardial exudates. French writers describe two forms of pulmonary complications: (1) The *forme pneumonique* (Besnier, Herzog, Hirsch), which is fugitive or migrating (pneumonia migrans). It may even appear before arthritis. It is said to be less typical in onset and solution; in the sputum; in absence of cyanosis and dyspnœa; in its asthenic prolonged course; and in its lobular development and incomplete hepatization; (2) the *forme œdemateuse* (Houdet, Ball, Bernheim), in which œdema may be widespread, may resemble the acute pulmonary œdema of Bright's disease, or may be localized in one lobe or in the bases.

8. Digestive Tract.—Angina or tonsillitis often occurs as a prodrome (*v. s.*) or as a later development. The tongue is coated and moist except in the severest cases where it is dry. The salivary secretion is sometimes acid, and increased sulphocyanides have been noted. Gastric symptoms are rare, except from such rare complications as meningitis and uræmia, though Damash considers that gastric disturbance is the most frequent prodrome. Constipation is the rule. Diarrhœa occurs as prodromal enteritis or as a rare complication, and is rarely bloody. Peritonitis, icterus and liver enlargement are most exceptional.

9. Genito-Urinary Tract.—Cystitis resulting from the use of cantharides blisters has been noted by Lebert, Senator, and by Homolle. Pribrâm considers it suggestive of gonorrhœa. Fibrinuria is very infrequent. Acute nephritis is seldom seen ($\frac{3}{10}$ to 1 per cent.); it occurs early, and chiefly in endopericarditis, and is usually benign but may become chronic. Renal infarction usually occurs late in the course of cardiac cases, and kidney suppuration indicates the pyogenic pseudorheumatism. But few of the author's cases in private practice show nephritis, though it is present in 75 per cent. of hospital patients. In these it is probably latent until awakened by the fresh toxæmia; it is probably due to their social status, vagabondism, exposure and alcoholism. *Albuminuria* is mostly transitory. It occurs late in the course or after several attacks, and mostly in individuals over thirty years of age. The percentage ranges up to 34 (McCrae). Albumosuria is frequent, especially during resorption, whence it has a slight diagnostic value. Cylindruria may occur without albuminuria; hæmatoporphyrin has been found, also urobilin, formic acid, sugar, the characteristics of fever urine, reduction of the chlorides and bacteria (sepsis). The sexual organs are involved chiefly in gonorrhœal pseudorheumatism. Amenorrhœa is usual. Uterine atrophy, hemorrhagic endometritis and genital hemorrhage are infrequent. Rheumatic arthritis as well as gonorrhœal "rheumatism" may occur in pregnancy. In the puerperium, gonorrhœal, septic or endocarditic joint inflammation may occur (see DIAGNOSIS).

10. Blood.—The blood shows increased fibrin and, in low dilutions, is said to sometimes respond to the Widal reaction. The red disks are reduced 25 to 30 per cent. and chlorotic characteristics are noted. The fever, the anæmia and the intensity of articular involvement run more or less parallel (Hayem and Garrod). The white cells, in moderate cases, range between 10,000 and 15,000, and higher figures (20,000) are only reached in extensive endocarditis, pericarditis, pleurisy, pneumonia, or in septicæmic pseudorheumatism or suppurative arthritis. The increase is in the polymorphonuclear neutrophiles while other cells are normal or decreased. The eosinophiles are practically absent, but increase with betterment of the articular findings. If they are increased in a florid case, the prognosis is good, since the course will probably be mild. Bacteriological findings are (1) negative, or (2) positive from mixed infection.

11. **Nervous System.**—The relation of chorea to rheumatism is an unsettled question. *Chorea,* rheumatism and endocarditis are associated (in 50 per cent. of cases, Pribrâm), or coördinated, and possibly a common, though unknown, infection is the essential correlating cause. A constant anatomical cause of chorea, as arterial change, encephalitis, or embolism, is not known. Cases with rheumatic or endocarditic association usually run a slow and often a relapsing course; cases which react to the salicylates are probably rheumatic. *Cerebral rheumatism* is very infrequent; it may develop acutely at the acme of the disease, with psychical alteration, often with hyperpyrexia, sometimes with warnings, or again precipitately. Delirium, unrest, convulsions, and coma, frequently result in death. High fever may be solely a cerebral symptom, but cerebral symptoms may occur without high temperature. The course is *foudroyant* or lasts several days, and even a week. Drug poisoning, alcoholism, inanition, previous exhaustion, hysteria, and the *tempérament cérébrale* are possible factors. The author has seen one case of recovery after mania, convulsions, meningeal symptoms, and coma. Chronic *psychoses,* hallucinations, sopor, mania, coma, convulsions, or melancholia, may occur in convalescence. They are usually due to inanition and the patient generally recovers. Brain *embolism* (from endocarditis), hemorrhage or rheumatic meningitis, are infrequent, and are not classed with the above varieties. *Spinal rheumatism* may be confused with myelitis, septic or gonorrhœal meningitis, multiple neuritis, local myopathies adjacent to the joints involved, or cerebral embolism. Exaggerated tendon reflexes, decubitus, and vesical paralysis, occur in some rheumatisms. In Kraus' clinic the author saw one fatal case characterized pathologically by meningomyelitis. *Multiple neuritis* is often rheumatic; it may precede the polyarthritis, but usually occurs in the first or second week of rheumatism and presents the usual characteristics, such as pain and tenderness over the muscles and nerve trunks, paralysis, muscle atrophy, skin dystrophy, reaction of degeneration, anæsthesia and lost reflexes. The prognosis is good and the neuritis reacts well to electrotherapy. Mononeuritis occurs in the oculomotor, sciatic, trigeminal, and other nerves, and usually regresses entirely.

THE EYE.—Rheumatism may result in benign episcleritis, iritis, iridocyclitis, and possibly conjunctivitis. Optic neuritis, with or without atrophy, and retinal embolism or thrombosis occur exceptionally; their prognosis is doubtful.

(Acute *thyroïditis* is most uncommon. Lymphadenitis is not uncommon, but is rarely marked or extreme. It occurs, with arthritis, erythemata, endocarditis or pericarditis.)

Prognosis.—The prognosis as to life is usually excellent and is generally proportionate to the severity and duration of the infection and to the number of joints involved. The percentage of deaths is 1.5 per cent. (Rubin); 3 per cent. (Lebert, Eichhorst, Raymond) 3.6 per cent. (Senator), or 3.7 per cent. (Roth), but these are hospital figures and therefore higher than in general practice. The death-rate varies with the geography of the disease; *e.g.*, in Ireland 2 in 1,000 die, in New South Wales, 3; in New York, 3; in Paris, 4+; and in Scotland the rate, including deaths from heart lesions, is nearly 5. In statistics other arthritides are not carefully excluded. There are waves of increased mortality every three or four years. In late years the death-rate has somewhat decreased. The *causes of death* are (1) hyperpyrexia, the most important; (2) cerebral rheumatism, chorea; (3) peri-, endo- and myocarditis; (4) pneumonia, lung infarcts. *Recurrences* occur in 19 per cent. of cases (Church) or in a larger percentage in general practice. They are more dangerous in pregnancy or with complications such as influenza, etc. The prognosis is *less favorable in children* than in adults. The salicylate *therapy* influences the disease and also its relapses; but the heart complications are only slightly or indirectly relieved. There is some danger of rheumatism becoming chronic. At the onset it is impossible to declare the case light or severe. According to Trousseau, the involvement of the small joints of the hands and feet justify the prediction of a severe course.

Diagnosis.—**1. Rheumatism in Childhood.**—In the very young the disease is most rare (3 cases in 85,000 nurslings) but becomes more common after the fifth year. The course is more rapid and the urine is normal or less febrile. Generally speaking, the joints are less susceptible and the arthritis less intense, while the skin, nervous system, and heart, are more often involved. Heredity is most potent in the young, and the sexes are equally involved. The various phases of rheumatism occur separately,—first arthritis, then endocarditis, later chorea, subcutaneous nodules, pericarditis, and so on. The child rarely lies quietly, but tosses about; the tendons and fasciæ are more often involved than the joints, and in this the rheumatism may simulate talipes. The pain is less and the arthritis may entirely escape recognition. The lower temperature is particularly remarkable at this epoch of life, when high fever is often excited by trivial lesions. There is much less sweating. The anæmia in childhood is especially marked, and is as rapid in development as that of diphtheria or that of malaria in adults. In children the differential possibilities are *acute osteomyelitis*, acute pyæmia, monarticular arthritis, especially of the knee and hip in nurslings, gonorrhœal arthritis, Barlow's disease, syphilis, hæmophilic joints, and infantile palsy; if the rheumatism occurs in the spine, meningitis or torticollis are of differential importance. Endocarditis is usually not so clearly nor so immediately connected with the polyarthritis as in adults, but it develops more slowly, insidiously, later, and twice as frequently as in adults (80 per cent. Vernay). Thomas Watson knew but three instances of acute rheumatism, before puberty, which escaped severe heart disease.

This is due to the greater functional demands on the child's heart and to the aorta being narrow at the isthmus. Endocarditis occurs relatively early in what Cheadle terms the "rheumatic progression," but pericarditis occurs later after the valvular lesion has inaugurated hypertrophy and dilatation. Pericarditis is almost always found (Sturges) in necropsies on children with valvular disease. Rheumatic heart disease in general at this epoch, leads to greater hypertrophy because of greater nutrition, to more marked dilatation because of greater tissue elasticity, and to more frequent recurrence. The prognosis is therefore much more unfavorable than in adults. In the diagnosis, functional murmurs are not frequent in children, although they do occur. The pulse is often irregular; the precordium more readily bulges; left-sided pleurisy is more frequent than in adults, and delirium and rheumatic nodes are more common. Oppression over the heart is infrequent, as are pain, cyanosis, dropsy, hepatic congestion, pulmonary infarction, dyspnœa and fever. Rheumatic children are more nervous and more inclined to night terrors, and to anæmia. More marginate, papular and urticarious erythemata and more purpura develop in children. Large subcutaneous nodes (rheumatismus nodosus) are serious prognostically and always suggest danger from severe intercurrent valvulitis. Chorea is prone to develop with arthritis, heart disease, nodes, or with the erythemata.

2. **Anomalous Rheumatism.**—By anomalous rheumatism we mean mixed infections,—*e. g.*, rheumatism with gonorrhœa. Typical rheumatism is easily recognized, while diagnosis in the masked or abortive variety is always problematical.

3. **Rheumatoides.**—Rheumatoides (Gerhardt) or *pseudorheumatism* (Bouchard) are symptomatic arthritides resembling rheumatism in that fever, arthritis, or endocarditis, may occasionally develop in their course. The nomenclature of Quincke, who speaks of *polyarthritis gonorrhoica, scarlatinosa, etc.*, is the least objectionable. (*a*) Polyarthritis *gonorrhoica* resembles rheumatism most closely and most frequently. Koenig holds that the most common of all joint diseases is the gonorrhœal (*v.* GONORRHŒAL INFECTION). (*b*) Polyarthritis *scarlatinosa* often most closely resembles rheumatism, occurs early or more frequently in the third to the fourth weeks of scarlatina, and varies with the epidemic (0 to 6.3 per cent.). In 66 per cent. of patients it occurs in the hands, and lasts four days, frequently with endocarditis or pericarditis (32 per cent. Hodges), and sometimes with recurrence and serous or purulent exudation. With it have been observed nodes, erythema and chorea. (*c*) Polyarthritis may also complicate many other diseases including most of the infections; measles, smallpox, varicella, erysipelas, dengue, malaria, typhoid, typhus, Malta fever, angina, appendicitis, diphtheria, syphilis, dysentery, pneumonia, influenza, recurrent fever, epidemic meningitis, neuritis, mumps, bronchiectasis, glanders, osteomyelitis, pyæmia, puerperal fever, septicæmia, tuberculosis, purpura, peliosis, scurvy, hæmophilia, anæmia, erythema-nodosum, Addison's disease, psoriasis and some neuroses and intoxications.

4. **Severe Types.**—In grave types of genuine rheumatism the visceral findings may be confusing but are least so when secondary, both in development and degree, to the arthritis. Jaccoud speaks of the primary visceral type, wherein the findings (*e. g.*, pulmonary or cardiac) antedate the arthritis, but here the diagnosis remains uncertain, until involvement of the joints develops.

5. **Gout** (*q. v.*).

6. **Arthritis Deformans** (*q. v.*).

7. **Tuberculous Polyarthritis** (*q. v.*).

The *number of joints* involved is always highly suggestive, but not finally diagnostic. True rheumatism and gonorrhœal rheumatism are often polyarticular. Diagnosis of gonorrhœal rheumatism in women is often difficult for notable reasons. In women, puerperal infections, and an acute monarticular osteoarthritis, beginning acutely from exhaustion in pregnancy or lactation, may cause difficulty. In growing children, *acute osteomyelitis* may cause articular swelling, which, however, is monarticular.

Treatment.—**I. Diet.**—The diet should be limited to fluids, arrowroot, milk, gruels, etc. Biot's results, with a strict milk diet, were extremely good.

II. Clothing.—The patient should wear a flannel gown and lie between blankets, which are more comfortable than sheets on account of the sweating. Care must be taken lest the drenching sweats macerate the skin. This can be avoided by dusting talcum powder in the axillæ, between the toes, etc., and by sponging the body with warm water and alcohol.

III. Local Treatment—Fixation lessens pain, fever, pulse and respiration-rate. Cold applications, boric fomentations, and salicylic salve are useful [Acidi salicylici ʒj, Adipis lanæ hydrosi ℥j]. Davies' cantharides blister and the actual cautery are indicated for tardy involution.

IV. Rest.—The patient should be in the position in which the greatest comfort can be obtained and in which there is the least danger of ankylosis. Splints afford relief to the muscles and joints, but muscular atrophy may result from the use of plaster-of-Paris casts. The absolute dorsal decubitus, long into convalescence, or at least for two weeks, must be enforced because of the weak condition of the heart.

V. Salicylate Treatment (Kolbe, 1874, Buss, 1875, and Stricker, 1876).—1. Effect on the Arthritis.—The pain and swelling are usually relieved after three to five full doses have been taken, or after twenty-four hours from the taking of the first dose. Salicylates are the best analgesic in rheumatism. Hypodermics of morphine are rarely necessary, except, perhaps, in the first twelve hours.

2. Effect on the Temperature.—The fever falls coincidently with relief of the articular symptoms, and reaches normal in two days. In the great majority of cases the acute stage is over within three days. In hyperpyrexia less influence is noted, especially if the salicylates are administered late.

3. Effect on the Endocarditis.—Though some consider the salicylates specific, the general evidence is that endocarditis occurs as frequently with their use as without it,—*i. e.*, salicylates are analgesic and antipyretic but not strictly antirheumatic. Many, however, claim that the salicylates are in every sense antirheumatic. The personal belief of the writer is that the salicylates are practically specific and that heart complications are lessened by shortening of the acute stage.

4. Relapses.—Relapses are claimed to be more frequent with the administration of the salicylates than with the alkaline treatment, and especially so when treatment is discontinued early. This claim seems unfounded. Relapses are apparently less frequent with the continued use of salicylates for two weeks after the initial symptoms are relieved.

Physiological Action of the Salicyl-preparations.—They are absorbed from local applications. The smallest doses produce such symptoms as cinchonism, fulness of the head or roaring in the head and ears. After larger doses dulness, headache, giddiness, a flushed dusky and moist skin, disturbed sight and hearing, and tremor, develop. Toxic doses produce the above symptoms, plus eye-muscle paralysis, pupil dilatation, deafness, increased reflexes, restlessness, delirium, dyspnœa, disturbed circulation, skin eruptions, sweating, and also green urine. Postmortem breaking down of the blood, visceral congestion, and serous ecchymoses are seen.

Administration.—Sodium salicylate is the best preparation. Its action is most prompt and certain; it is soluble and least irritating. Where it fails, other preparations are usually unsuccessful. All salicyl-preparations are absorbed as sodium salicylate produced by the alkaline intestinal juices. Grains xv of the natural acid, which is better than the synthetic, or grains xx of the sodium salt, are given every two or three hours until cinchonism appears; *i. e.*, until slight dulness, dyspnœa and deafness develop. This action is maintained for twelve hours, when the interval between doses is lengthened to four or six hours. The effects are better in proportion as the disease is more acute, the symptoms more pronounced, the inflammation greater, the number of involved joints larger, and the fever higher. It is well tolerated by children and usually can be given by mouth in fluid, if the taste be disguised by wintergreen oil, syrup of ginger, and syrup of orange. Even then, however, it is frequently nauseating.

℞

Sodii salicylatis	ʒvj.
Sodii bicarb.	ʒiv.
Syrupi zingiberis	℥j.
Syrupi aurantii	℥j.
Aquæ	q.s. ad ℥iv.

M. et S.—One teaspoonful every three hours.

Theoretically it should not be given in capsules for in this form it may irritate the stomach, but it may be given in powders which are taken in starch wafers. Because the action of sodium salicylate is analogous to that of quinine, bromide of potassium may be given to lessen the ringing in the ears. In case the stomach is intolerant, salicylates may be administered in pepsin, or given by rectum in peptonized milk. When given as an inunction, it is soon found in the urine, but absorption by the skin is uncertain. Aspirin is less irritating to the stomach than salicylate of soda. It is given in doses of five to ten grains at the same intervals. Salol (Phenylis salicylas) is given in the same dose at the same interval, but the kidneys must be carefully watched.

The *alkaline treatment* is said to decrease the heart complications and shorten the disease. Though these claims are not established, the alkaline treatment may be used in conjunction with the salicylates, using vichy salts with enough citrate of potassium or bicarbonate of soda to make and keep the urine alkaline. Long ago Fuller advised the following:—

℞

Sodii bicarbonatis	ʒss.
Potassii acetatis	ʒss.
Liquoris ammonii acetatis	ʒiij.
Aquæ	ad ℥ij.

M. f. haust.
S.—To be taken in effervescence with—

℞

Acidi citrici	ʒss.
Aquæ	℥ij.

Baginsky thinks that the administration of gr. xv to xl of potassium iodide daily produces better results in children than do the salicylates.

UNUSUAL RESULTS.—Unusual results of salicylate treatment:

1. *Nervous.*—Salicylate delirium has been observed in anæmic girls, neurotics, and in alcoholics. It may be violent and protracted. Convulsions are quite infrequent; they are centric.

2. *Cardiac.*—Small doses stimulate the heart, and cardiac depression is observed only when the largest dosage is given, when the preparations are impure, or when the heart is already diseased. If the heart becomes weak or irregular, acute myocarditis is probably developing and salicylate treatment must be suspended, the dose must be decreased, or the heart muscle must be stimulated by coffee, strychnine or alcohol. Slight irregularity without weakness is not a contraindication. Cardiac depression may be noted in nervous subjects. Thorne has observed collapse from aspirin.

3. *Respiratory.*—With antecedent or concomitant bronchitis, the lungs should be explored for possible congestion which may result from the remedy. Cardiac depression, sudden respiratory failure and violent expiratory dyspnœa are observed in a few cases. Tuberculous patients should be carefully watched.

4. *Renal.*—Caution is necessary in old nephritics, because the salicylates are excreted by the kidneys as salicylic, oxalic, or salicyluric acids and as salicin; however infective albuminuria, directly due to the rheumatic virus, indicates, rather than contraindicates, the use of the salicylates. The green color of the urine is due to indican

and pyrocatechin. Binz believes that the salicylates may induce abortion.

5. *Alimentary.*—Vomiting (*v. s.*) and diarrhœa may result from salicylate treatment.

Diagnostic Therapy.—Senator claimed that sodium salicylate is specific in true rheumatism and inert in pseudorheumatism. Though the general truth of this statement is acknowledged, its application is not absolute. Salicylates are of some value in the arthritic type of grippe. They usually are useless in the gonorrhœal, syphilitic, tuberculous, septic, scarlatinal, and other arthritides.

Convalescence.—Absolute rest must be enforced for two to four weeks to save the heart.

DENGUE.

Definition.—An acute specific infection, occurring chiefly in tropical and subtropical countries, extremely contagious, and characterized by fever, an initial prodromal eruption, violent articular and muscular pains, and a secondary polymorphous eruption.

Etiology and Epidemiology.—The actual cause is uncertain. A small diplococcus or bacillus resembling the Bacillus influenzæ has been described. Graham found, in the blood-cells, an organism resembling the Plasmodium malariæ, but small and unpigmented. For this reason infection has been connected with mosquito-bites. Eberle describes a "plasmeba." The disease is very contagious and seems disseminated less by direct than indirect infection, *e. g.*, by fomites, bites, etc. It is carried by pilgrims, sailors, and emigrants. The disease spreads with remarkable rapidity and infects large numbers of people. Its home is in the tropical and subtropical zones and it prevails chiefly in the hot months. It has invaded Europe, Asiatic Turkey, South America and the United States. It is remarkable that the coast ports are alone invaded. It very seldom reaches inland, and even in regions where the disease prevails endemically, closely contiguous inland towns and higher places are exempt. Unhygienic conditions promote its development. In the first Philadelphia epidemic it was called "Front Street Fever," on account of the squalid condition of the houses along the river front. The first epidemic, in 1779, in Java, was described by Brylon, and Benjamin Rush wrote of the Philadelphia epidemic in 1780. The chief later epidemics or pandemics occurred in 1818, 1824–1828 (reaching Savannah and New Orleans), 1830–1870 (again reaching America, and seen even as far north as Ohio), 1876 (Virginia), 1889, 1890, 1895–1897. The morbidity is enormous but the mortality is strikingly inconspicuous. Epidemics last two to five months. No immunity is conferred by one attack and two, three or even four, infections have been observed.

Symptoms.—After an incubation of one to two days, or sometimes a little longer, the disease *begins suddenly.*

1. Febrile Stage.—The fever rises quickly to 102°, 104° or even 107°, usually with a chill, severe frontal or orbital headache, sudden and intense pains in the back and limbs, which incapacitate the patient while walking and which involve especially the knees. The names "breakbone fever," first mentioned in Rush's description, and "dandy fever," given because of the mincing gait which results, bring out this salient feature. Dengue is thought by some to originate from the Spanish for "dandy." The *joints* are usually *swollen* (pseudorheumatism), and many of them, both large and small, are involved one after another. The *muscles* are often *painful* and the skin is sometimes hyperæsthetic. The pulse averages 100. The *face is suffused* and swollen, the conjunctivæ are injected, the flow of tears is increased, and in some cases there is coryza. Total anorexia, foul breath, coated tongue, and great thirst, develop; vomiting is occasional and constipation the rule. The *premonitory rash*, usually hyperæmic, is of short duration. It may last but one to five hours, and is frequently absent. The urine is febrile. Prostration may be marked, and, in some cases, severe delirium, convulsions, and coma may develop. This stage lasts three days ("three-day fever") and ends with profuse sweating. Fever is absent in the lightest cases.

2. Exanthematous Stage.—This stage begins on the fourth day. The eruption occurs chiefly on the face, hands, forearms and chest, and is decidedly polymorphous. It has been described as scarlatiniform, morbilliform, urticarial, erysipelatoid, lichen-like, vesicular, or even pustular. It is present in 60 to 100 per cent. of the cases. It is evanescent, lasts two to three days, and is followed by fine desquamation and by intense itching, sometimes by falling of the hair, and by cutaneous hyperæsthesia. The throat is sometimes red or even inflamed, and occasionally lymph-gland enlargement, parotitis and orchitis occur. The fever in this stage may be elevated, may be absent, or subnormal temperature be noted. The pain often recurs. Herpes, splenic tumor, otitis, albuminuria and leukocytosis are absent. Leukopenia is persistent.

3. Convalescence.—Convalescence begins in five to seven days, after a crisis or lysis, and is very tardy. There is great prostration, gastric uneasiness, insomnia, emaciation, and anæmia, all of which are remarkable after such a short disease. In no other disease of equal duration is such marked constitutional disturbance noted.

Complications and Sequels.—These are great rarities. Cutaneous or gastric hemorrhage, neuralgia, endocarditis, pericarditis, nephritis, pneumonia, pleurisy, œdema or ulcers, rarely develop.

Prognosis.—Death is rare, though the morbidity is enormous. The highest recorded death-rate was ½ of 1 per cent. One death occurred in 7,435 cases in the Anglo-Indian army (1872), and 10 deaths in 100,000 cases occurred in Smyrna. Relapse may occur in one to two weeks.

Diagnosis.—The geography and the presence of an epidemic are suggestive. The exanthemata, rheumatism, insolation, yellow fever (*q. v.*) and epidemic *erythema exudativum multiforme* may be suggested. *Influenza* is differentiated by its epidemic-pandemic incidence, its occurrence in all climates, seasons and locations (inland as well as on seaboard), its more frequent direct infection, the more general immunity conferred, the higher coincident mortality (pneumonia and tuberculosis), its more frequent respiratory catarrh and lung complications, its more severe nervous, intestinal and cardiac symptoms, and the greater frequency of herpes, splenic tumor, otitis media, and albuminuria. In the rare cases of *acrodynia*, observed in France, Belgium, Turkey, Persia, and Mexico, Scheube gives the following differential points: (1) digestive disturbance; (2) facial swelling; (3) pains in the extremities, sometimes with anæsthesia of the soles; (4) spastic symptoms; (5) afebrile course; (6) erythema multiforme.

Treatment.—*Isolation* and quarantine are necessary. *Pain* is the prominent indication, for which antipyrin and salicylates serve fairly well, though they are much inferior to opium.

CHOLERA ASIATICA.

Definition.—A specific infection caused by Koch's comma bacillus. It is chiefly disseminated by drinking-water, is characterized clinically by vomiting, severe purging, and by such symptoms as cardiac failure and suppression of urine which are due to loss of fluids and to toxæmia.

History.—The name has been used since the time of Hippocrates, Galen and Celsus, but apparently not for Asiatic cholera. Asiatic cholera is said to have existed before Christ, and travelers in India in the sixteenth century spoke of it. Its first European invasion was in 1817, from which time our knowledge of cholera dates. Other epidemics have occurred in 1826, 1832, 1847–1855, 1865–1867, 1873, 1881–1884, and 1892–1894. Cholera visited America in 1832–1835, 1836, 1848, 1849, 1854, 1865, 1867, and 1873. This was the last epidemic though isolated cases have occurred since.

Bacteriology.—Koch, while studying cholera in Egypt and India, in 1883–1884, discovered the specific organism in the fæces during life and in the bowel at autopsy. The *comma bacillus* is 1 to 1.5 μ long and half as wide; is smaller than the tubercle bacillus, but more plump and curved; is motile and flagellated (Loeffler). Gaffky (1887) first obtained pure growths on various media. In cultures its form varies, being sometimes S-shaped, and thread- or spirillum-like. The comma bacillus is found in the stools and in the intestines, usually in pure cultures. After several days it may be found in the bowel wall, though rarely in the stomach or vomitus. It usually disappears from the movements after six to eleven days and very rarely reaches the blood or viscera.

Etiology.—In its dissemination and contagion, cholera resembles typhoid rather than the exanthemata, *i. e.*, infection is less direct than indirect. Attendance on cholera patients is not dangerous if the hands are kept clean. The germ is very susceptible to boiling, to acids, to ordinary antiseptics, to drying, and to the presence of saprophytes in sewers and water. There is little risk of aërial dissemination, though flies may carry infection. Koch early maintained that cholera was conveyed by *water contamination*. He found the cholera vibrios in a water-tank in Calcutta from which infection was clearly traced. Fraenkel, von Ermingen, Biernacki, Lubarsch, Loeffler, and others, found it in flowing and stagnant water. Pettenkofer vigorously objected to Koch's teaching and insisted that individual and local disposition was more essential. He even swallowed pure cultures, and luckily escaped infection. It is well known that cholera vibrios may be found in the movements without the existence of cholera symptoms, just as diphtheria bacilli may be found in healthy throats. The normal acid of the stomach protects the individual and animals are infected experimentally only when the gastric acidity is neutralized and peristalsis is arrested by opium. Nevertheless, the terrible Hamburg epidemic (1892–1894) amounted, as Koch said, to a demonstration of almost laboratory exactness. The disease developed "explosively." On August 16 there was one case; by August 27 there were 1,000 deaths, and September 3, there were 10,000 cases and 4,300 deaths. The total mortality was over 8,300. The Hamburg drinking water came unfiltered from the Elbe River, which was then at a low level, the weather being hot. The germ was found in this supply. Altona and Wandsbeck, which are parts of Hamburg, had a supply of good water, and practically escaped. That part of Hamburg which used Altona water was exempt. Instances might be multiplied. The cholera germ is difficult to find in water because it is so easily overgrown by saprophytes.

Infection by ice, by milk, by green lettuce, by flies, or in the laboratory, is less common. The washing of infected linen is particularly dangerous to washerwomen. In India, hundreds of thousands of pilgrims drink the "holy water" of the Ganges, in which they also bathe and defecate. Similar infections occur in the pilgrimages to Mecca. In former years the caravans carried infection from India through Persia and Afghanistan to Russia, which was also the route of dissemination in 1892. Ships from the Orient are extremely dangerous.

Individual Predisposition.—Decrease of physiological resistance increases the risk of infection. Acute and chronic alcoholism, the use of strong purgatives, dyspepsia, overexertion, poor blood, lack of hygienic conditions, and debilitating diseases are therefore predisposing factors. By counting the mortality in a cholera-stricken community for a few years, it can be seen that the total death-rate is not increased, which means that weakly persons succumb to cholera and that later epidemics of other diseases have a diminished prey.

Epidemics involve a smaller percentage of the people in large than in small cities, but they last for months in large cities and usually only for weeks in small towns. Epidemics end with a lessening mortality, and both their ending and the lessened mortality are probably best explained by the decreasing virulence of the germ. Warm weather tends to increase epidemics, and they are usually checked by frost. Nevertheless, Russia's severest epidemic occurred in the winter time.

Symptoms.—After an *incubation of two or three days* (one to eight, possibly fourteen days), the disease develops with varying virulence. The bacilli are found in the stools of some healthy persons. Several stages are usually described. These may be presented, as they also correspond to clinical types of different severity.

1. **Prodromal Diarrhœa.**—The stools contain the comma bacillus, are thin and normal in color, are voided without pain or tenesmus, and sometimes produce only moderate depression or malaise. The trouble may cease here, when we speak of cholera diarrhœa, or it may pass into the choleraic stage.

2. **Choleraic Stage.**—This may usher in the disease or follow the first stage. The stools, at first yellow and voided frequently, become colorless because the bile pigment is diluted and decreased, and become thin,—the ominous "rice-water" stools. Diarrhœa usually comes on for the first time at night. The stools may number twenty or thirty and a pint to five quarts (500 to 5,000 c.c.) are voided daily. They have a fetid, not a fecal odor, and contain 98 to 99 per cent. water, some salts, and a trace of albumin. In the evacuations are seen flocculi which are made up of comma bacilli, leukocytes, epithelial cells or shreds, detritus, and sometimes a few red cells. There is often gurgling, although little gas is voided. Much fluid is thus lost to the circulating blood, and this causes some of the following symptoms: the abdomen is flat, usually neither tender nor painful, and fluid may be percussed in the bowels. *Vomiting* is frequent, but usually follows the diarrhœa. It is painless, although exhausting, and is thin, and profuse, even to three quarts daily. Sometimes Koch's bacillus is found, or again either urea or its decomposed product (carbonate of ammonia) may be found when uræmia exists. The *skin* early shows the lack of fluid; it becomes *shrunken, lax,* wrinkled, and when pinched, its folds disappear but slowly. The nose becomes small and pointed, the eyes hollowed, and the malar bones jut out prominently (*facies cholerica*); the extremities, nose, and ears become cold and cyanotic, and grayish-blue rings develop beneath the sunken eyes. If pleurisy or dropsy are present they are rapidly absorbed. The secretions such as the saliva and urine become lessened and disappear, though nursing mothers may sometimes continue to secrete milk. The loss of fluid occasions distressing *thirst* and *muscle cramps,* especially in the calves of the legs, but also in other muscles, which are found dry at autopsy. The immense transudation into the bowel has other effects; it *concentrates the blood,* whose cells and hæmoglobin rise; the red cells may total six to eight millions and the serum has a specific gravity of 1,040 or even 1,070 (1,028 being the normal). The *heart tones* become *weak* and the pulse rises to 100, because the body cannot lose 10 per cent. of its fluid without injury. The heart has not enough blood upon which to contract and the blood is viscid;

the heart also suffers reflexly, as it does in intestinal incarceration, and degenerates from absorption of toxins (soluble substances from the bacilli). The difficulty in oxygenation increases the breathing to thirty or forty, and occasions precordial and epigastric anxiety. The *vox cholerica* is due to the dry larynx and weak muscles. The reflexes are decreased or absent, the pupils tardy; the sensorium is normal, although exhaustion, apathy, and vertigo are usual. The urine contains albumin and casts which are largely hyaline. While the external parts are colder by 5° to 10° or more, the patient feels hot, and the internal temperature may be 102° or higher. The loss of weight is great (even 0.5 to 1 per cent. hourly!). In some few cases the patient dies before diarrhœa develops (*cholera sicca*). Sometimes these are cases incorrectly diagnosticated. Recovery is possible, even with these ominous symptoms; reaction may set in, the diarrhœa and algidity may lessen, the heart may improve, and erythematous, roseolous, urticarial and other rashes may develop. The urine is often albuminous, and indican, which was previously absent, may reappear; the urea is increased two- or threefold and the ethereal sulphates are increased. The symptoms are then chiefly toxic, and probably result from absorption of the cholera and other toxins from the intestine (the so-called *cholera typhoid*,—*i. e.*, cholera with a pronounced typhoid state).

3. Stage of Collapse.—This stage is characterized by asphyxia (literally meaning without pulse), paralysis, or algidity. The heart tones are inaudible, the radial pulse is imperceptible, the arteries are empty, and the veins are full. In days of venesection the blood would not drain from the incised veins, and at autopsy little or no blood escapes from the vessels. The picture is that of collapse. The skin is lead-colored, cyanotic, even violet or purple, and will not vesicate after blistering. There is dyspnœa, the expired air is cool, the voice gone, the mouth dry, the conjunctivæ dry or opaque, and the lids are parted, yet the mind is clear. The evacuations cease. Pericardial or pleural rubs may be heard, due to the great viscidity found in all the serosæ at necropsy. Very few cases with the symptoms of cholera-typhoid react and recover, but most of them die within a few hours, or within a day or two. After death the muscles frequently contract, to the great terror of observers. The face is distorted, the fingers flexed, and the legs drawn up. Post-agonal rise in temperature may occur. At autopsy the small intestines are injected, filled with rice-water contents, their epithelium or villi are desquamated or necrosed, the lymphatic structures are sometimes swollen, reddened and reticulated. Comma bacilli are found in the stools and intestinal wall, and occasionally there is an agonal invagination of the gut. The muscles are dry, the spleen small, and the kidneys show a pale cortex with great cortical degeneration, especially in the convoluted tubules; the renal changes, once attributed to loss of water and disturbed circulation, are probably toxæmic.

Complications.—The so-called croupous or diphtheritic enteritis is due to the pyogenic cocci. Diphtheroid changes may also be found in the uterus, vagina and throat. Hemorrhages from mucous or serous surfaces, though usually small, may occur. Pneumonia (20 per cent. of fatal cases), pleurisy or lung infarct; skin, parotid or other suppurations; icterus, bronchitis, venous thrombosis or post-febrile psychoses, are uncommon. Griesinger's "cholera-typhoid" is the most frequent sequel (25 per cent. of cases); nervous complications and uræmia are far less common causes than the toxins absorbed late from the intestine and operating intensely on the already exhausted, slightly degenerated, and anæmic brain.

Diagnosis.—Diagnosis is usually easy in the presence of an epidemic. The first cases occasion the most difficulty, especially if they are light. As already stated, robust individuals may not react to the germs found in their stools; simple diarrhœa may result in other cases, and only a mild depression may mark some cases known as *cholerine*. On the other hand, aged, weakly, or tuberculous persons may succumb to cholera before its characteristic stages develop. In clinically suspicious cases, *laboratory* measures are necessary for diagnosis. The pseudocholera vibrios in the Elbe, Seine, or Danube Canal (Vienna) are differentiated with difficulty. The comma bacillus is obtained (*a*) in nearly *pure cultures* from the stools, especially from their shreds; (*b*) in peptone cultures (1 per cent., with 1 per cent. salt) in which the germs are found on the surface in six hours, because they rise to obtain oxygen; (*c*) in 10 per cent. gelatin plates, in which they develop in fifteen to twenty hours; (*d*) the nitrosoindol (cholera-red) reaction results on adding sulphuric or hydrochloric acid. It was thought that no other bacterium produced both nitrates and indol, but the pseudocomma organisms may produce the same reaction; (*e*) in animals, in which the cholera germ produces a sudden fall of temperature, but this is uncertain; (*f*) *Pfeiffer's test* is the sole absolute criterion, according to which the blood of animals immunized to cholera produces agglutination of the true cholera bacillus.

Cholera nostras produces a similar picture. It is usually less severe, though sometimes fatal. In some of these cases the Finckler-Prior comma bacillus is found, but in the majority of cases it is absent and other microbes are present, such as the colon bacillus, and less often the streptococcus, staphylococcus, and a bacillus like the Bacillus subtilis; vibrios, proteus and pyocyaneus forms; an encapsulated bacillus; Gärtner's Bacillus enterides; in meat, anaërobic forms; and in mollusks, Zieber's Bacillus piscicidus agilis. It is possible that many of our domestic intestinal organisms may become virulent. Liebermeister said cholera nostras bears the same relation to cholera Asiatica as varicella does to variola, German measles to measles, or epidemic icterus to yellow fever. It occurs at the same season. Its symptoms differ chiefly in degree, and occasionally, a large percentage (even 40) of deaths occur, chiefly in the extremes of life.

Arsenical poisoning, peritonitis, and intussusception may without bacteriological confirmation closely resemble cholera.

Prognosis.—The prognosis is always grave. Cholera ranks next after the plague in death-rate, and averages over 50 per cent., probably nearer 60 per cent., and sometimes 80 per cent.; 100,000 to 700,000 die yearly in India. In 1883, 28,000 died in Egypt, 8,605 died in Hamburg, and 800,000 in Russia (1892–1894). About 45 per cent. of the deaths in Hamburg occurred in the first twenty-four hours and 17 per cent. in forty-eight hours, whence the prognosis is more favorable if patients last longer than two days; 85 per cent. die when the stage of collapse is reached. Light cases may become severe or the worst cases may recover. Unfavorable factors are youth and old age; pregnancy; colorless stools, early frequent movements, early vomiting, anuria and low peripheral temperature.

Treatment.—**1. Prophylaxis.**—Quarantine of ships, isolation of early cases, thorough disinfection of the stools, soiled linen, etc., and special attention to light cases and to any suspicious diarrhœa, are most important. In convalescence the stools may remain infective for seven weeks (Kolle). Despite the sudden outbreaks in Germany in 1892–1894, sanitary measures kept the total of deaths at 10,000, while in Russia 800,000 died in the same period. Individual prophylaxis concerns the maintenance of the physiological resistance; moderation in eating and drinking; the boiling of water for drinking and washing; the boiling of milk; care in regard to ice; avoidance of green fruits or vegetables, and of contamination of food by flies. The prophylactic serum of Ferrán and Haffkine is valuable. In one series of cases the use of the serum reduced the morbidity two-thirds and the mortality one-half.

2. Diarrhœa.—This should be treated by absolute rest in bed, and by initial cleansing of the bowels wth calomel and castor oil; thin gruels should be given as a diet; red wine with aromatics, tea, and full doses of bismuth and opium should be given. Intestinal antiseptics are inert. The diarrhœa, except the early movements, is not easily stopped, because absorption is arrested. Cantani advised that 1 or 2 quarts of 1 per cent. tannic acid solution be given slowly by rectum in the hope that it would reach the small intestine, and Griesinger advised that larger quantities be given, and that expulsion by the rectum be forcibly prevented. The fluid was given until it was vomited, apparently with some results.

3. Vomiting.—Vomiting is intractable. Fluids should not be withheld. The stomach may be washed with normal salt solution. Hot wine, camphor, coffee, champagne, and hypodermics of morphia are also indicated.

4. Heart.—Failing heart action and renal secretion are sometimes helped by a warm bath. Hypodermatoclysis was recommended by Cantani (1865) and intravenous transfusions were employed by Hermann and Latta (1830). Their use was studied during the Hamburg epidemic and their employment often increased the heart's force and benefited the general condition greatly, at least for a time. The results are somewhat better than the mortality (65 to 77 per cent.)

would indicate, for they were used in the stage of profound collapse (where the death-rate is as high as 85 per cent.) If used earlier the results might have been better. The kidney secretion is apparently little influenced by this treatment.

5. **Muscles.**—Muscular cramps are helped by warm baths, sinapisms, friction, local heat, saline infusions, and morphine, which should be given hypodermatically.

6. **Convalescence.**—In convalescence, great care is necessary lest the diarrhœa return.

Other symptoms, complications and sequels are treated expectantly.

YELLOW FEVER.

Definition.—A specific infection of unknown etiology, which is transmitted by the mosquito, is endemic in the American tropics and subtropics, and is characterized pathologically by moderate degeneration, especially of the liver and kidneys, and clinically by a rapidly developing fever, by icterus, albuminuria, adynamia, hæmatemesis, and by other hemorrhages.

Etiology.—The cause is unknown, although two microörganisms have been especially obtained: (1) Sanarelli's Bacillus icteroides, which is said to be present in the blood, spleen, liver and kidneys, but not in the intestine; it is a facultative anaërobe, with rounded ends, 2 to 4 μ long and half as wide; positive agglutination tests are said to have been made; its action is said to be fourfold: (*a*) steatogenic (producing fatty degeneration); (*b*) congestive and hemorrhagic; (*c*) emetic, and (*d*) icterogenic. Sanarelli's organism is generally considered as a contamination. (2) Sternberg's bacillus, resembling the influenza germ. While the cause is not known, it is certain that the disease is transmitted by the mosquito (*Culex* or *Stegomyia fasciata*). This was first suspected by Carlos Findlay, in 1881, and the mosquito was demonstrated to be the intermediate host by the investigating board composed of Walter Reed, James Carroll, A. Argamonte and J. W. Lazear. Dr. Carroll recovered from a severe attack, but Dr. Lazear died from inoculation. Dr. Myers, of the English Commission, also succumbed. The Culex fasciata breeds not only in stagnant street pools, but also in cesspools and sewers, whence its special danger in unhygienic subtropical and tropical towns. Voluntary inoculations by mosquitoes, which twelve days previously had bitten yellow-fever patients, in the first three days produced yellow fever in non-immunes. Findlay failed to prove his correct suspicion because he only allowed the mosquitoes three to six days in which to develop the germs which they had absorbed, while twelve days are necessary. As in malaria, the tropical foci, the hot summer and autumn seasons, the invasion of low altitudes, river courses and coast towns, humidity, and exposure to night air, are conditions which promote yellow fever because they are suitable to the mosquito. Frost stops infection because it kills the mosquito. Direct contagion from person to person is unlikely. Nurses and physicians are seldom infected from contact with yellow-fever patients. Persons going from the City of Mexico to Vera Cruz may acquire the disease, but on coming home with it they do not cause its dissemination, because the mosquito is not present. Postmortem wounds and direct voluntary inoculations, however, have directly communicated the disease. The yellow-fever virus is the only virus, infecting man, which passes through fine filters. It was thought, until recently, that fomites, clothing, etc., were infective, but Reed's committee has experimentally exploded this theory. Volunteers remained in rooms occupied by yellow-fever patients and slept in their soiled linen without contracting the disease.

Predisposition.—The lighter races are more prone to infection. The value of acclimatization has been overestimated, and it is thought that immunes are merely those who have had light and unrecognized infections. Most cases occur in males between twenty and forty years of age. Yellow fever is kept alive in endemic foci by mild cases, especially among the Creole children.

Foci and Epidemiology.—The two prominent homes of yellow fever are (1) Mexico, the Antilles, Mexican Gulf and Caribbean Sea; and (2) Africa, from Senegal, through Sierra Leone, to the Gold Coast. The first clear description of yellow fever, according to Sodré and Couto, was in 1686, but certainly yellow fever was known before the discovery of America. It reached this country possibly in the sixteenth century and has extended as far north on the coast as Boston and on the Mississippi River as far as St. Louis. It has reached Quebec and Chicago. In Philadelphia (1793, 1797–1798), 4,041 died in a year; in New York, 2,080; and in New Orleans, in 1853, 8,000 died. Epidemics occurred also in 1802, 1805, 1853, 1867, 1873, 1878, and to a slight degree in 1897–1899; New Orleans was visited severely in 1905; there were about 9,000 cases and 1,000 deaths. Europe is seldom, but South America is frequently, invaded.

General Clinical Description.—**Incubation.**—The incubation averages three or four days. Prodromal symptoms are not infrequent and last less than a day. They may be malaise, fatigue, headache, dyspepsia, colic, or other indistinctive symptoms. The disease is often divided into stages, not all of which are always present:

Stage I.—The *stage of congestion* begins (*a*) with some headache, frontal, supraorbital or retroöcular, the earliest and most prominent symptom; (*b*) sometimes with a chill; (*c*) with flushed face; the conjunctivæ are injected and sometimes icteric even at this stage; the eyes are sensitive to light, the lids somewhat puffed up and the expression is a mixture of pain, restlessness and confusion, comparable to the alcoholic facies; (*d*) with backache, and pains radiating into the calves of the legs; (*e*) with fever rising to 104° or more; and (*f*) with a full, tense pulse of 100 to 110.

The tongue is usually clean or slightly coated dorsally, and its tip and edges are red. The urine is febrile and very early albuminous.

The skin is intensely red and dry. The epigastrium is tender early in the disease and there is marked gastric irritability and intolerance of food, water and medicine. This stage lasts two days or somewhat longer.

Stage II.—The *remission stage,* stage of calm, or deceptive stage, occurs in some cases and is so-called because of later severe symptoms following this remission. The fever may fall nearly to normal or to subnormal; the pulse falls to 80 or 90, and pain and hyperæsthesia remit. Slight jaundice may now be first apparent.

Stage III.—*The Reaction Stage.*—Slight temperature, the "black vomit" (*vomito negro*) of blood, hemorrhage into the nose, intestines, uterus or skin, hiccough, epigastric pain and tenderness, liver enlargement, and icterus of the skin and accessible mucosæ usually mark this period. The urine becomes scant and is laden with bile pigment, or its secretion is suppressed; the pulse becomes weaker, and such nervous symptoms as insomnia, prostration and restlessness may appear.

Various *issues* are henceforth possible: (*a*) Gradual amelioration; (*b*) fatal anuria; (*c*) profuse hemorrhages; especially from the alimentary mucosa, which usually end in fatal collapse, and rarely in recovery; (*d*) profound icterus, which results in adynamic phenomena and usually in death, though recovery is possible, and (*e*) death, with pronounced cardiac insufficiency.

Special forms of yellow fever are described by Soudé and Couto which vary with the intensity of infection and of special visceral involvement. (*A*). *Variations in intensity* include (1) light, abortive, ambulatory, atypical forms, especially in children; (2) the usual severe forms, and (3) the foudroyant type, which is rapidly lethal in twenty-four to forty-eight hours. (*B*). *Visceral forms:* (1) The hepatic type, which is slow, with two weeks of the status typhosus, often with normal temperature and sometimes with hemorrhages; (2) the uræmic type, often with early death; (3) the hemorrhagic; (4) the cardiac; and (5) nervous or atactic type.

Special Symptomatology.—**1. Fever.**—The fever is neither cyclic nor diagnostic, though Sternberg considers that there is a single febrile paroxysm of two to seven or eight days. It is usually highest in the first stage, and reaches an average of 104° (though sometimes even reaching 108°). It may fall on the second or third day to nearly normal in benign cases or in the ominous uræmic type. High fever on or after the second day (second stage) is a poor prognostic, and is sometimes due to secondary infection with the pyogenic cocci, or the typhoid or colon bacillus. In the second stage the fever is usually between 99° and 100°, and may rise in the third stage. A high post-mortem rise is frequent. In convalescence, the temperature, aside from complications, is always subnormal.

2. Nervous Symptoms.—Nervous symptoms are often absent in both benign and malignant cases. Insomnia and headache are common in the congestive period. Delirium is infrequent, and in 80 per cent. of the cases, is uræmic rather than purely toxæmic. Atactic symptoms are far more common in children than in adults although they may develop in alcoholics and in the debilitated. In children, the status typhosus or meningo-encephalitic symptoms (convulsions, even unequal pupils, Cheyne-Stokes breathing, and coma) are possible. Pathologically, few changes are found except the slight degenerations common to the febrile stage, punctate hemorrhage (mostly agonal), or bile-tinging.

3. **Circulatory Symptoms.**—(*a*) The pulse in the congestive stage is usually 100 to 130 and is tense from increased blood pressure; on the third day, 90 to 100 (even 70 to 80); in the second stage it is still lower in benign and more rapid in severe cases (110 to 140), is small, and the manometer shows lowered arterial tension; in convalescence it is usually slow (50 to 60, or even 30). Faget, of New Orleans, emphasized the slowing of the pulse with maintenance of the fever, which he and others consider almost pathognomonic. (*b*) In severe forms the first tone over the apex of the heart becomes weak and is finally lost. The same is true of the second tone. The heart action is rarely irregular or intermittent. Embryocardia is found in severe cardiac types. In the second or third stage, cardiac dilatation is common (in 70 per cent. of autopsies). Cardiac insufficiency may develop early or late. These cardiac signs are due to cloudy and fatty myocardial degeneration; pericardial and endocardial ecchymoses are frequent. Cardiac action is exaggerated when uræmia is present. Endocarditis, pericarditis and acute aortitis are infrequent complications. (*c*) There may be some anæmia, but often there is none. The white cells are decreased in proportion to the severity of infection. The polynuclears are relatively increased.

4. **Respiratory Symptoms.**—Respiratory symptoms, such as dyspnœa, may be due to intercurrent inflammation or more often to cardiac or renal complications.

5. **Digestive Tract.**—Severe hiccough is attributed to irritation of the phrenic nerve by distention of the stomach or by enlargement of the liver. Epigastric pain, tenderness and anxiety may be symptomatic of cardiac distress, premonitory of the black vomiting, or indicative of disturbed circulation in the medulla, which is expressed in the endings of the vagus.

6. **Icterus (Yellow Fever, Typhus Icteroides).**—Jaundice may appear early, but usually develops on the third to the fifth day. It is frequently absent in children, or in cases of early death from uræmia or from nervous symptoms. According to Féraud, it is absent in 50 per cent. of cases which recover. The explanation for this is an open question. Féraud distinguishes two forms (*a*) the early, lighter jaundice, due to blood pigment, and (*b*) the later and more severe type due to bile pigments. Sodré declares that the yellow-fever liver cannot produce bile pigments.

7. **Liver.**—The liver is slightly enlarged and is tender. Pathologically, its color is light, and it has a cooked appearance. The left

lobe may reach the spleen. On section, it is oily, fatty, anæmic, and under the lens, shows fatty degeneration, disorder, and deformation of the liver cells.

8. **Urine.**—The urine is early febrile, is sometimes scanty, and shows decreased urea, uric acid, chlorides and phosphates. *Albuminuria* is an important finding and occurs in 88 per cent. of cases in the first three days. Leucin and the bile pigments are common. Indicanuria is very infrequent. Casts are common. The kidneys, anatomically, are normal in size, rather pale or yellowish, and, microscopically, show great fatty degeneration of the tubules, which in severe cases are actually choked by desquamated epithelium and casts. The glomeruli show remarkably few changes. *Anuria* is due to these changes and to lowered blood pressure; it is fatal in 97 per cent. of cases. *Uræmia* causes 33 per cent. of the fatalities; it is marked by delirium, nervousness, restlessness, twitching or convulsions, by decreased or suppressed urine, by rapid, slow, forced, irregular, labored, or Cheyne-Stokes breathing, and by tense pulse. Death in coma may occur as early as the third day. Uræmia and anuria are rare in children.

9. **Hemorrhages.**—These may occur anywhere. They are most frequent in (*a*) the alimentary tract, (*b*) the nose, and (*c*) the uterus. They result from fatty degeneration, undue permeability, or actual rupture of the bloodvessels. (*a*) *Alimentary tract.* Hemorrhages may occur from the mouth, tongue, lips, stomach, or intestine. The *black vomit* is gradually transuded blood altered by the gastric acid; it most often occurs on the fourth to the sixth day; it is the ordinary "coffee grounds" vomit or is ink-like; the stomach, at autopsy, shows the black contents, congestion, or even dark discoloration, epithelial desquamation and fatty change in the muscularis. Hemorrhage from the small intestine is almost equally frequent. (*b*) *Epistaxis* may occur early or late, is often profuse, and sometimes fatal. (*c*) *Uterine hemorrhages* are severe, especially at the menstrual periods, and in pregnant women cause abortion or premature delivery. Less frequent are (*d*) *skin petechiæ*, which are found sometimes in large plaques or hypostatic ecchymoses, clinically and at postmortem; (*e*) *muscular hemorrhages* which are painful, often large, and which sometimes suppurate. (*f*) Hemorrhage in other mucosæ (larynx, urethra, and eye), the serosæ (pleuræ, endocardium, or meninges), or in the viscera (brain, medulla, and lungs).

Complications and Sequelæ.—These include (1) secondary infections with the pneumococcus, pyogenic cocci, or the colon bacillus, such as endocarditis, pericarditis, pneumonia, erysipelas, parotitis, arthritis, suppuration, and gangrene. (2) Coincident typhoid, malaria, influenza, and insolation. (3) Post-febrile psychoses.

Diagnosis.—The diagnosis is usually easy from (1) the flushed, semi-intoxicated facies; (2) icterus; (3) the almost invariable and early albuminuria—the most valuable finding in doubtful cases; (4) black vomit, gastric intolerance, and epigastric uneasiness; (5) other hemorrhages, and (6) the slow pulse, which is absent in some severe, but therefore clear, cases. *Dengue* lacks these features. Pernicious *malaria*, with icterus, is distinguished by the blood findings, the enlargement of the spleen (always absent in yellow fever), the infrequency of early icterus, albuminuria and hemorrhages (except hæmaturia). This form of malaria is rare in the cities, whence the constant suggestion of yellow fever, when more than sporadic cases are reported. *Weil's disease* (infectious jaundice), and acute yellow atrophy of the liver are usually readily distinguished.

Prognosis.—The mortality of epidemics varies from 10 per cent. to 85 per cent. The average death-rate is about 30 per cent. The mean death-rate, according to *age*, is 33 per cent. (one to three years), 14 per cent. (four to eighteen years), and 37 per cent. (nineteen to forty years). Unfavorable events are the early development of the third stage, hepatic, renal, or cardiac insufficiency, the typhoid state, diffuse hemorrhages, combination of special types (cardiac plus renal, or hepatic plus renal), high fever with hepatic symptoms, and hemorrhages.

Treatment.—**I. Prophylaxis.**—Disinfection of the fomites is now less important than the question of the mosquito. Probably no greater triumph of hygiene exists than the stamping out of yellow fever since the American occupation of Havana. It illustrates not only the efficiency of the investigating scientific commission, but also the possibilities of medicine in administrative capacity with unlimited discretionary power. In Havana the average death-rate was 500 yearly, until Gorgas cut it down to *nil.* The patient should be isolated, not to prevent direct personal contagion but to keep the Culex fasciata from the infected person. Healthy individuals should be kept from infection by the mosquito by the same measures that are employed in malaria, *e. g.*, screens, and extermination of breeding foci. The Culex fasciata is found outside the habitual yellow-fever foci, and after biting patients from abroad, may inoculate other persons. Infected insects, too, may be carried by ships.

II. Symptomatic Treatment.—(*a*) In the *first stage*, fluidextractum aconiti, ♏. iij, sodium salicylat., gr. xx, and antipyrin, gr. x, may be given every two to four hours for a few doses, until the congestion and pains are relieved. Coal-tar products are not to be employed when depression exists. Fractional doses of calomel, gr. ¼ every hour for five doses, should be given and followed by two or three ounces of castor oil. Overmedication is particularly to be avoided because of the frequent gastric irritability, and feeding by rectum is often indicated. Early or violent cerebral, uræmic, or pulmonary symptoms are sometimes treated by phlebotomy. (*b*) In the *last stage*, the black vomit contraindicates any fluid, food, or medication by mouth; warm peptonized milk and normal salt solution should be given by rectum; morphine, when the patient is in this condition, is dangerous, as is the perchloride of iron, when given by mouth. (*c*) In *cardiac insufficiency*, digitalis is especially good, and it is apparently beneficial to the patient even when cardiac failure is absent; saline solution, by rectum or subcutaneously, produces better results than intravenous transfusions. The patient must be kept absolutely quiet. (*d*) *Uræmic* symptoms are treated in much the same way. Much water should be given, chiefly by the bowel. (*e*) *Nervous* manifestations necessitate cool spongings, bromides, morphine, and hyoscine, as in Typhoid. (*f*) Vomiting (see TYPHOID) is relieved most efficaciously by chopped ice.

THE PLAGUE.

Definition.—The pest, or bubonic plague, is a specific acute infection caused by the Bacillus pestis and characterized by a pronounced typhoid state, fever, buboes and carbuncles, very frequently by septicæmia (pesticæmia), and by extremely high mortality.

History.—The first clear accounts date from the times of Trajan, in the second century, and of Justinian, in the year 542. The plague was important among the pestilences of the middle ages. In the fourteenth century the "black death" swept over Europe with a mortality of 25,000,000 (one-quarter of the world's population). It appeared from time to time in the sixteenth and seventeenth centuries, but in the eighteenth century it withdrew southeast, and disappeared from Europe until the small Russian epidemic in 1878–1879. Egypt was the early focus for African epidemics, and European epidemics started from Turkey. Syria was the leading Asiatic focus, thirteen epidemics occurring between 1773 and 1843. The present Oriental focus seems to be Southern Thibet, whence India and China have been invaded thirty times between 1823 and 1897. Uganda is considered a permanent plague home by Koch; plague is endemic in Asia near Mecca, in Mesopotamia and in Siberia. In 1899-1900, New York, San Francisco, Glasgow, Cape Town, South America and Australia were visited. Epidemics appear, reach their maximum, and decline at nearly the same time each year. The theory that the virus remains permanently in the soil has many supporters.

Etiology.—The *Bacillus pestis* was found by Yersin, in 1894, and at the same time Kitasato found a similar, possibly an identical, bacillus. Its ends are rounded and stain deeply; its sides are somewhat convex; it measures 1½ to 1¾ by ½ to ¾ μ and is encapsulated. It is polymorphous, and appears with equal frequency as rods or as diplobacilli. Atypical forms are oval, ovoid, annular, chain-, thread-, or branch-like. Other degenerative or involution forms are the pyriform, biscuit, comma-shaped, or spermatozoid. Though it is most often outside of the cells, it may appear in them. It contains no spores nor flagella. It is found in all organs, in the spleen, the lungs and tonsils; in the sputum, the urine and fæces; and in the bubo and carbuncles. Its cultures are typical, and superficial, and have bulging, wall-like edges. It stains with ordinary stains,—*e. g.*, alkaline methylene blue, etc.—but not by Gram's method. It is readily killed by heat, sunlight, desiccation, antiseptics, and is often overgrown by secondary infection. It lives, at the most, for three to four weeks in buried bodies, but may persist much longer (*v. i.*) in the saliva, sputum, urine or fæces of convalescents.

Atrium.—It may enter (1) by the *skin*, through invisible wounds, as does erysipelas; through scratches, or as shown by Albrecht and Ghon, through the intact integument. Postmortem infections are frequent, even though there is no visible abrasion. Laboratory inoculation is possible, as was unfortunately demonstrated in the Vienna cases, among which was that of Müller, who was then writing his well known monograph. Flies, fleas, ants, bed-bugs, etc., may inoculate the disease. (2) The accessible *mucosæ*, conjunctivæ (experimentally), and clinically the nose, tonsils, anus and external genitalia, may be primarily infected. The *nose and throat* are especially frequent points of entrance in children, for obvious reasons. (3) The alimentary tract affords a possible atrium. (4) The bacillus may cause primary plague pneumonia, whence metastatic infections occur.

Contagion may be direct or indirect. (*a*) *Direct.*—Dissemination by the sputum of plague pneumonia is most dangerous, for the bacilli are scattered by coughing, as shown in Flügge's experiments in tuberculosis (*q. v.*). The pest spreads rapidly only when it is of the pneumonic type. The bacilli may penetrate from unruptured carbuncles or buboes or scatter from them after their rupture. The urine, the fæces, and the milk of nursing women are also dangerous. Direct contagion may result from autopsies, from operations, or from nasal or urinary hemorrhages. (*b*) *Indirect.*—This may result from fomites, from bites of animals or insects containing the germ, and, possibly, from contaminated water. Epidemics among rats may precede human epidemics. In Canton, 35,000 dead rats were found in one district. The disease may also exist among apes, cats, mice, and other animals. Infection by flies, fleas, etc., is certainly possible, and Simond considered the rat-flea particularly responsible, though the parasites of animals seldom attack man. Flies may convey infection from the urine or fæces of infected rats. The bacillus produces *virulent toxins*.

Symptoms.—There are two factors in the clinical picture: (*a*) The local changes, such as those in the primary localization (bubo or pneumonia), and (*b*) the successive septicæmia (pesticæmia) and toxæmia. The average *incubation is five days* (two, to seven or ten). Occasionally there is transitory prodromal depression, nausea, or glandular pain, but usually the *onset is very acute*, with marked headache, intense vertigo, severe rigor, sudden fever, and, in children, convulsions. Early vomiting is quite frequent. There is *early glandular pain*, the prostration is profound, and an early typhoid state develops. The *fever* reaches 104° on the second day, is either continuous or remittent, in convalescing cases falls by lysis (fifth to seventh day), or may remain high until death. Crisis is seen in light cases only. The *skin* is *hot* and dry or may be covered with sweat as death approaches. Herpes never develops. Skin *hemorrhages* are frequent. They are due to embolism caused by the Bacillus pestis, they occur almost always in the agony or shortly before death, and are seen mostly in the upper trunk, arms, head and neck, and sometimes also in the muscles and periosteum. Abscesses, phlegmons, gangrene, or bedsores, are not common.

1. **Characteristic Local Finding.**—This is the bubo, for the lymph glands are more important than in any other infection. The *several clinical forms* of the plague usually described are (*a*) the *bubonic* type (77.7 per cent.); (*b*) the *septicæmic* type (14 per cent.); (*c*) the *pulmonary* type (4.3 per cent.); (*d*) the *carbuncle* type (2.5 per cent.); (*e*) mixed infection (1 per cent.), and (*f*) the ambulatory type (pestis minor, 0.5 per cent.), which is considered by Scheube as not plague but "climatic bubo." The figures are from Choksey, of Bombay. In the great majority of cases, probably in all except in the pulmonary type, *the bubo is found* very early (90 to 98 per cent.) and local pain, tenderness and swelling are present. The primary bubo occurs in the external lymph glands in the region of the atrium; it is large, very painful and tender, and is attended by great periadenitis, œdema, hemorrhage and vesicle formation in the skin. Its size and rapid growth are due in part to medullary swelling of the lymph glands, but chiefly to hemorrhage, which may spread along the nerves, arteries or veins, into which it may break, thus disseminating the bacilli. The glands may fuse, and a brawny mass, as large as an egg, or a man's fist, results. The glands and surrounding tissues contain enormous numbers of bacilli at this stage. The location is inguinal (70 per cent.), axillary (20 per cent.), and cervical (10 per cent.). The inguinal and axillary adenitis is more common in males; cervical adenitis results in children from infection of nose and throat by dirty fingers; the popliteal and epitrochlear glands remain uninfected with curious frequency, being involved in but 1 or 2 per cent. Strangely, too, the lymph-vessels are usually unaffected, although they are sometimes inflamed near the buboes or carbuncles, or around the atrium of infection. From the primary bubo (especially when the blood is not early infected) the contiguous glands are involved; *e. g.*, after the infection of the inguinal glands, the iliac, aortic, mediastinal, subpectoral, infra- and supraclavicular or cervical glands may be involved; *i. e.*, infection usually occurs in the direction of the lymph-current, but retrograde involvement is possible. On the fourth to the sixth day necrosis occurs, and on the eighth or ninth day suppuration develops, resulting from mixed infection (33 per cent.), but more often (66 per cent.) from the Bacillus pestis alone. Some glands may resolve without suppuration. The primary bubo is often inconspicuous and its size has no bearing on the intensity of the disease, although cases with marked periadenitis rarely recover. Besides the primary bubo and its contiguous lymphatic extension, Albrecht and Ghon described the *secondary bubo* which is infected by the bacilli-laden blood current. The bacilli have a remarkable affinity for lymphatic structures and settle in them largely. These *hæmatogenous buboes* are generally smaller and less tender, painful, œdematous, and hemor-

rhagic, than the primary bubo, and rarely exhibit periadenitis unless the clinical course is protracted. They contain the bacilli in numbers, as do other lymphatic tissues, such as the mesenteric glands, the pharyngeal follicles, the stomach, intestine, and lung. *Carbuncles* result chiefly from pesticæmia, although apparently in some cases they are the atrium. They occur in 10 to 15 per cent. of the cases, often near the primary bubo. The crowning serous vesicle is ruptured, discharges bacilli, and leaves an ulcer, whose floor is bluish-red from hemorrhage, or yellow from swarms of bacilli. A dark crust then forms. There is great peripheral infiltration, and the whole limb or part of it may become œdematous. These lesions indicate that the pesticæmic type is less a distinct type than an almost regular part of the disease. Cases rarely recover which show bacillæmia. The blood shows some anæmia and moderate polymorphonuclear leukocytosis. The spleen is increased three to four times, is reddish, and is somewhat less soft than it is in typhoid, anthrax, or streptococcic infections. Its cut surface is finely granular and is slightly glistening. It is infiltrated with white cells and bacilli, which are found especially in the innumerable foci of minute necrosis. It is usually palpable on the first or second day.

2. **Nervous System.**—The nervous system suffers few anatomical changes except meningeal hemorrhages and brain œdema. Clinically the toxæmic nervous "typhoid" symptoms are as prominent as the buboes. There is early headache and marked prostration. Vertigo is especially notable, causing a drunken, cerebellar gait, and vomiting. The delirium is more often dull, dreamy and docile. than active or maniacal, though the patient may fight the attendants or run away. Motor twitching, tonic or clonic spasms, subsultus, rigidity of the neck or spine, and even bulbar symptoms, such as increased pulse and respiration, polyphagia or polydipsia, may be suggestive of meningitis, which sometimes actually occurs by pesticæmia. Acute ataxia, myelitis, paraplegia, hemiplegia, or isolated neuritis (monoplegia, vagus weakness, aphonia, palate paralysis) and other nervous sequels are uncommon. Ear symptoms are unusual, but are easily masked by the profound typhoid state. Conjunctival hyperæmia, sometimes with ecchymoses, is so frequent that it is, together with the fever and buboes, made one of the cardinal symptoms by Yamagiwa. Iridocyclitis is not uncommon.

3. **Circulation.**—The circulatory and the nervous changes measure the intensity of infection and forecast the prognosis. Possibly there is no other cardiac poison as intense as is the plague toxin which operates on the medulla and its vagus nucleus. Early in the attack the pulse is full and dicrotic, with a rate of 80 to 90. Later, the pulse increases to 120, 150, or even 200; the heart tones become muffled; tachypnœa, great dyspnœa, and all the signs of acute cardiac insufficiency develop. In some virulent infections the pulse may remain slow. At autopsy the heart is lax, the right ventricle dilated, the myo-

cardium cloudy, and the serous investment ecchymotic. Endocarditis or pericarditis almost never occurs.

4. **Respiratory Tract.**—The lymph structures in the larynx, trachea, or bronchi are infected by aspiration from tonsillar foci or through the blood. Fatal laryngeal œdema may occur. Most important is the *plague pneumonia*, of which there are two varieties, (*a*) the *primary*, in which the lung is the atrium (or analogue of the primary bubo). It was clearly the "black death" of the middle ages (Forbes). It was seen in India, in 1838, and became well known in the Indo-Chinese epidemic of 1896–1897, when Childe first found the Bacillus pestis. The bacillus, if inhaled, causes typical confluent lobar pneumonia and salient lymphatic involvement. The bacilli exist in hordes in the lung and sputum, and may cause hæmatogenous buboes and carbuncles. The diagnosis is positive only when the sputum appears; this is followed by cyanosis, dyspnœa, profuse hæmoptysis, splenic tumor, pleuritic pains, signs of consolidation, adynamia and collapse. Death occurs in 97 per cent. of the cases. (*b*) *Secondary* pneumonia is (i) embolic (Yamagiwa), and occurs with abscess in the liver and kidney; it develops as multiple small foci, is difficult to diagnosticate surely, though bacilli in the sputum, dyspnœa, and pleuritic pain are suggestive. (ii) Aspiration pneumonia usually occurs in the lower lobes, its diagnosis is difficult, it follows bubonic disease of the tonsil, pharynx or bronchi, and is almost invariably fatal.

5. **Digestive Tract.**—A moist, then a dry, tongue, hemorrhage from the gums or throat, vomiting and involvement of the tonsils or pharynx, are common. There may be ecchymoses in the stomach and intestines, hemorrhagic erosions, bacilli in the bloodvessels, and bubonic changes in the lymph structures. The latter, generally considered rare, were found by Wilm in 20 per cent. of his cases, and mesenteric adenitis in 60 per cent. Meteorism and diarrhœa sometimes develop. The liver is slightly swollen from cloudy and fatty degeneration or from stasis; bacilli may be found in the ecchymoses, in the bile, or in the fæces.

6. **The Genito-urinary Tract.**—The urine is decreased, the specific gravity is 1,010 to 1,020, the chlorides are decreased, indicanuria is frequent, and moderate albuminuria is very common. Nephritis is quite frequent. Anatomically, degeneration, minute focal necrosis, and glomerular hemorrhage resulting from capillary embolism of the bacilli, are common. Bacilluria occurs in about 30 per cent. of the cases. The bacilli have been found in the aborted fœtus. Genital complications, except uterine bleeding, are rare.

Diagnosis.—The *cardinal signs* are the fever, buboes, pesticæmia, and the results of examinations by means of cover slips, cultures, agglutination, and inoculation. Though the finding of bacilli in the blood is important in cases without buboes, the cultural results are more reliable. The agglutination reaction is not well marked until the second week of infection, is seen most advantageously in convalescence, and may still be seen for eight weeks or more. A positive test is final, though negative results do not exclude plague.

Prognosis.—A patient may become ill in the morning and die before night. The average duration of the disease is about a week. If the patient should outlive this time, the chances of recovery are good. Recurrences are not frequent but have occurred, and the immunity conferred is only relative. Symptoms which are unfavorable, are especially marked nervous and cardiac toxæmia, digestive disorder, hemorrhages, cervical buboes, tonsillar involvement, and pneumonia. The death-rate varies with the epidemic. It is often 80 per cent. and sometimes 90 or 95 per cent. It is highest between the fiftieth and sixtieth years; next between the thirtieth and fiftieth. In Toulon (1721), 77 per cent. of the population had the plague and 62 per cent. died—higher percentages than in any other epidemic disease. In Canton, 8 per cent. (1894), and in Damaum, 30 per cent. of the population died; 430,500 died in Bombay, from 1896 to 1899.

Treatment.—**1. Prophylaxis.**—The plague patient should be isolated and carefully covered with netting. Infected houses should be isolated, unroofed to admit the sun, fumigated to kill insects and rats, and the walls should be washed with antiseptics. Vigorous attacks should be made on rats. Ships should be held in quarantine until the rats in their holds are killed, the bilge-water disinfected, and all clothing fumigated. The sputum may be infective for three months and the urine for one and a half months. Carbuncles and buboes should be washed with bichloride of mercury and dressed antiseptically. Prophylactic inoculation with dead cultures and with serum from immunized animals and from convalescent patients, is a measure assured of success (Haffkine, Yersin, Roux and Lustig).

2. Symptomatic Treatment.—Buboes and carbuncles are most efficaciously treated with dressings of weak bichloride of mercury. Surgical extirpation may disseminate infection, and injections, into the buboes, of carbolic acid, do not stay the septicæmia. Digitalis, alcohol, and hydrotherapy are employed as in other toxæmias. Bichloride of mercury is well tolerated and has been used freely since it was noted that syphilitics under mercurial treatment seemed to withstand the disease.

DYSENTERY.

Definition.—An acute contagious disease, (*a*) with varying etiology, but caused chiefly by the Amœba dysenterica and Shiga's Bacillus dysenteriæ; (*b*) attended anatomically by bowel inflammation (catarrhal, necrotic, or ulcerative) and (*c*) characterized clinically by colic, pain, tenesmus, and frequent muco-bloody passages.

History.—The name dysentery dates from Hippocrates (480 B. C.), and the affection is clearly mentioned in the oldest medical script, the *Papyrus Ebers* (1550 B. C.), and in the *Veda*. It has been the scourge of armies from the times of the Greco-Persian wars. The most severe and wide-spread epidemic occurred in 1847–1856. In our civil war there were 287,522 cases among the Northern troops. Among the English soldiers in India 30 per cent. of the deaths were due to dysentery. It prevailed in the recent South African and Spanish-American wars, but was practically absent among the Japanese forces (1904–1905). Though essentially a disease of the tropics and subtropics, it is frequently observed in temperate zones, especially in prisons and in asylums. Lack of hygiene, overcrowding, eating of spoiled food or fruits, impure water, and alcoholic or other excesses, are predisposing causes. For several decades the disease has decreased in Europe and America.

Classification.—A classification and description of dysentery is most difficult, because our knowledge of all its causes is as yet imperfect. Two well known forms will be described first, and various indeterminate allied forms afterward.

I. Shiga's Bacillary Form of Dysentery.—In 1897 Shiga, of Japan, described the *Bacillus dysenteriæ* as the sole cause of the Japanese epidemic form. His claims are based on these facts: (*a*) it is always found; (*b*) it occurs in no other affection; (*c*) its numbers, in a given case, correspond to the intensity of the infection; (*d*) it is found in the intestinal wall; (*e*) the bacillus and its toxins, when inoculated, produce intestinal hemorrhage; (*f*) there is a specific agglutination which occurs in no other disease, increasing in intensity under observation and most marked in convalescents; (*g*) the serum of the immunized cases is of prophylactic and therapeutic value; (*h*) actual inoculations in man produce dysentery, as in Strong's inoculation of a criminal under sentence of death, and in Flexner's assistant, as well as in accidental laboratory infections. The *bacillus* resembles the colon and typhoid bacilli in form. It usually occurs singly, but sometimes in pairs. It is less motile than the Bacillus typhosus but is flagellated; it is a facultative anaërobe, contains no spores, stains readily but not by Gram's method, and grows on all media. In gelatin it grows with deep, pin-point cultures, is brownish-yellow without liquefaction, and has well marked borders; it develops best in glucose agar (grayish growth along the puncture, without fermentation), or in litmus milk (some slight acidification, followed by alkalinization and a brownish color). Inoculations by mouth succeed, even without first disordering digestion. Inoculation intraperitoneally or subcutaneously is usually fatal, but sometimes causes colitis. Almost pure cultures are obtained from the movements. They are most abundant when the lower intestine is greatly involved, and are less in the early stages from diarrhœal stools than they are in the later stages from muco-bloody stools. When the dejecta become purulent (latest stages), the bacillus disappears. It is found in the mesenteric glands, but not in the blood, spleen, or liver. Infection is probably carried by contaminated drinking water. Flexner, Barker, Strong, Musgrave and Craig have found this type in the Philippines; Kruse found it in Germany, Flexner in America (1901); Goodliffe and others also report it. It has been found in Porto-Rico. Duval, Bassett, Wollstein and other clinicians have found it in infantile types. There are *three strains* of the bacillus: (1) The Shiga type, which attacks glucose but not other sugars; (2) Flexner-Harris type, which attacks glucose, mannite and dextrine but not lactose, and which forms more acid than the Shiga type; (3) Hiss-Russell type, which attacks glucose and mannite but not lactose or dextrine. The blood of a dysentery subject will agglutinate all types, but particularly the second. The bacillus constituted 54 per cent. of Strong's and Musgrave's series of 1,328 cases in the Philippines. Epidemics have existed for centuries in Japan, chiefly between the months of June and October. In the Philippines, dysentery prevails at the end of the rainy season.

Symptoms.—After an *incubation* of two to eight days, there is a *sudden onset* with fever, reaching 100° to 102° (even 104°), colic, and diarrhœa. Pain becomes marked and the simple diarrhœal stools soon show mucus, and then *bloody mucus* or pure blood, which is voided with great frequency and severe rectal spasm (tenesmus). The passages are small, numbering twenty to sixty or even one hundred a day, and contain Shiga's bacillus. The rectum may prolapse. The urine is scanty. In mild cases the symptoms abate in two to three weeks, and the stools become purulent and contain fewer bacilli.

The symptoms arise from *local inflammation* and from *systemic reaction*, not to toxins, according to Shiga, but to solution of the bacilli themselves in the tissues. In the more severe cases the systemic reaction is profound, especially when lesions occur in the lower small intestine, the absorptive power of which is great. A *typhoidal* condition results, with coated tongue, nervous toxæmia, and weak pulse, and death frequently occurs as early as the fourth or seventh day. Shiga names this entero-dysentery, as distinguished from the ordinary type, colo-dysentery. If the stools become putrid or gangrenous, the outlook is hopeless. Ecchymoses on the chest, epigastrium, and inner surface of the limbs are quite frequent. Some choleriform cases occur. The disease sometimes lasts for weeks or months. Emaciation is marked in acute and chronic forms, which is due to diarrhœa, inanition, pain, and intoxication.

The symptoms are largely explained by the *anatomical findings*. The bacilli lodge in the small folds and large flexures of the colon and sigmoid. Their action is furthered by constipation. The mucosa is intensely hyperæmic and shows several degrees of inflammation, which varies according to the intensity and duration of the infection. It becomes catarrhal, hemorrhagic, diphtheritic, and finally necrotic with ulceration. The rugæ are wide and swollen; the entire mucosa is swollen, œdematous, thickened, and shows superficial coagulation necrosis, fibrinous exudation, and cellular infiltration; the submucosa is infiltrated and vascular; the muscularis is sometimes infiltrated and the serosa is ecchymotic and freshly fibrinous. In marked cases the necrotic areas become ulcers, in or between the follicles; they have a flat base, occupy chiefly the folds, usually reach only the muscularis mucosæ, and in intense cases impart to it a sieve-like aspect. The swelling may be such as to obliterate the lumen of the intestine. Cocci and colon bacilli are in the upper layers but never in the lower ones, where we find the Bacillus dysenteriæ in and around the glands. If the small intestine is implicated, the mesenteric glands swell. In the virulent cases the gut becomes gangrenous. The spleen is never involved. Stenosis may follow cicatrization of the ulcers.

Diagnosis.—*Local rectal disease* (syphilis, cancer, or papilloma) may possibly be confusing. In contradistinction to *amœbic dysentery,* it is generally acute, with a course of two or three weeks; toxæmic symptoms, such as fever, headache and delirium, and hemorrhages, are common, because involvement of the small intestine is usual; anatomically the ulcers are flat and are on the folds of the intestine. Amœbic dysentery is characteristically subacute or chronic. The small intestine less frequently suffers (Shiga); livèr abscess is common (20 to 33 per cent.), and anatomically necrosis begins in the submucosa and produces ulcers with undermined edges. *Agglutination* is similar to that of the Widal reaction in typhoid; it occurs in dilutions of 1 to 20 (even 1 to 50, 100, or 1,000). Shiga observed that it gradually increased as the disease progressed, as Foerster noted in typhoid fever. It may appear on the third day, but is usually seen in the second week and most clearly in convalescence. It is therefore rather late for diagnostic purposes. If it increases while under observation and is marked, it is a good prognostic, and the converse is true.

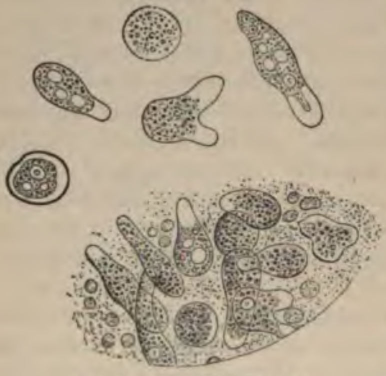
Prognosis.—The prognosis is more favorable in *involvement* of the *rectum* and sigmoid flexure than when the upper colon or cæcum is affected. *Intoxication* is a bad prognostic. The effect of *serotherapy* (v. i.) is another factor. The mortality is high, particularly in Japan, where, in 1893, there were 167,300 cases with 25 per cent. mortality, and in 1900, 462,000, with 22 per cent. mortality. *No immunity* is conferred. A second attack was observed in 2.7 per cent. and a third in 0.3 per cent. in Ogata's collection of 25,279 cases.

COMPLICATIONS.—These are parotitis or muscle suppuration, which are secondary infections occurring in severe cases and in the third to the fifth week; ascites in debilitated subjects and beriberi.

II. Amœbic Dysentery.

Lambl (1859) and Loesch (1876) discovered an amœba in the bowel movements, but Kartulis, in Egypt, studied over 500 cases and found the amœba in them and found the secondary liver abscess. Kartulis established his claims for the Amœba dysenteriæ on the grounds that (*a*) it was constant in certain forms of dysentery, in the ulcers, fæces and liver abscesses; (*b*) it was inoculable in cats; (*c*) negative results were obtained with other organisms and with the amœba which is found in normal fæces. This was confirmed by Koch, Hlava, Fenoglio, Quincke, Roos, Pfeiffer, Kovâcs, Kruse, Pasquale, and others in Europe, and in America first by Osler (1890); the important monographs of Councilman and Lafleur, Dock, Simon, Musser, Stengel, and Harris soon followed. The home of amœbic dysentery is in the tropics, especially in Egypt, India, Formosa, China, Arabia and in Italy, where it is endemic and sometimes epidemic. Sporadic cases occur everywhere in the temperate zone, especially in Germany and in America, where it is the most frequent form. The author has seen six cases which were clearly acquired in Chicago. In the Philippines it constituted 42 per cent. of the Strong and Musgrave series. According to Flexner and Strong, it is more frequent in chronic than in acute cases. The amœba is found in the pus, glairy mucus, or blood-tinged mucus of the evacuations. It is round in the quiescent stage or irregular from protrusion of pseudopodia, particularly when the slide is slightly warmed. It measures 12 to 30 μ and consists of a hyaline ectoplasm and a finely granular endoplasm. The minutely granular nucleus measures 5 to 7 μ. Besides the nucleus one or several vacuoles may be seen in the cell. Its glistening appearance, size, movement, swarming granules, vacuoles, and frequent red blood-cell inclusions, readily differentiate it from swollen epithelia or leukocytes. It stains readily. The amœba probably enters in the food or drink. It has been cultivated in *pure* cultures (Wooley and Musgrave).

Fig. 18.



Amœba coli. (Hallopeau.)

Symptoms.—The symptoms of *acute amœbic dysentery* begin rather abruptly with much the same picture as in Shiga's type, although the fever is generally lower and shorter, rarely lasts more than two or three days, and is sometimes absent. The movements are alkaline, pultaceous, fluid, and yellow, at first, and later, mucous and bloody. Sometimes in one movement fæces are voided first, followed by mucus and blood, or blood-tinged fluid containing amœbæ, slightly yellow, glairy clumps like swollen sago or frog spawn, pus cells, detritus, and sometimes the Charcot-Leyden crystals. The stools may be chocolate-colored. Sometimes large masses of necrotic tissue are voided. The stools number 5 to 20 or more daily; their odor is fetid when fæces are present; they are odorless when only blood or mucus is present; and are stinking when gangrene occurs. The repeated and painful movements cause straining, sweating, and sometimes syncope. *Colic* and borborygmus are nearly always present; pain is referred to the navel; tenderness is usually marked over the sigmoid, colon and frequently over the cæcum, and tenesmus and a burning anal pain mark involvement of the lower bowel. The rectum may prolapse and vesical symptoms, such as strangury, may appear. The abdomen is sometimes distended. *Very light* cases may only show catarrhal movements, but blood, pus, membrane, or shreds, indicate necrosis and ulceration. *Severe* cases may begin with chills, fever of 103° or 104°, vomiting, herpes, and symptoms like those of cholera; *e. g.*, dry tongue, depression, 20 to 100 movements daily, leg cramps, scanty urine, failing pulse, rapid emaciation from loss of rest and of fluid, and from pain. Early death may result. *Peracute* cases, with chill, high fever, great abdominal pain, collapse, and even sudden death, are exceptional, but may result from extensive rapid necrosis or gangrene. In the average acute case, the course of one and a half to three weeks, or to several weeks, varies with several factors, but especially with the individual resistance and the treatment. Perforative peritonitis sometimes occurs, as well as profuse exhausting hemorrhages. Light cases, if neglected, easily become severe or chronic, and spontaneous recovery is quite uncommon.

Chronic amœbic dysentery may result from the acute form or stage, or be subacute (subchronic) from the beginning. The symptoms are essentially the same, but less intense. As a rule there is marked, and sometimes extreme emaciation, particularly in the tropics. On the other hand, some patients maintain their weight and suffer only from recurrent diarrhœa every few weeks or months, or from irregular moderate intestinal derangement. In the average case the tongue is lacquered and red, the abdomen is somewhat distended and tender, the lower colon and sigmoid are palpable as a thick cord, and sometimes mental depression or hypochondriasis is observed. The *outcome* is (1) recovery after two to four months; (2) recurrent attacks for months or years; (3) complications, such as liver abscess (20 to 33 per cent.), especially in the tropics (peritonitis, empyema, or lung abscess). Perforative peritonitis, exhausting bowel hemorrhages, brain or splenic abscess, pseudorheumatism (Sydenham, Trousseau), myelitis (Gubler, Mitchell), neuritis, and less frequently, endocarditis, or chronic nephritis, are other complications; (4) gradually increasing anæmia, emaciation and death from exhaustion.

Pathology.—The early changes of the mucosa are catarrh, inflammation with injection, swelling, and hemorrhage, mostly in the large intestine. The essential change is not superficial in the mucosa, but in the *submucosa*, as John Hunter first noted, though Councilman and Lafleur first brought out this point clearly. The amœbæ enter by the tubular glands and reach the submucosa where, by their action alone, without bacterial coöperation, they cause cellular accumulations which are not leukocytes, but chiefly fibrinous exudation and connective-tissue cells. Sometimes the amœbæ enter the bloodvessels early and pass through them to the submucosa. These foci undergo coagulation necrosis and slightly elevate the mucosa which at first covers the small submucous cavities, but afterward sloughs, and exposes the primary grayish-yellow necrosis in the submucosa. When the necrotic tissue sloughs out, the dysenteric ulcers are more apparent. They lie, according to Kartulis, in the whole of the large intestine (50 per cent.

of cases); in the colon descendens and in the sigmoid flexure (25 per cent.); in the cæcum, with ascending or descending colon, or in the rectum (25 per cent.); in the rectum alone (20 per cent.); in the cæcum alone (5 per cent.), and only rarely in the small bowel or in the appendix; Musgrave found the appendix involved in 7 per cent., the ileum in 3.5 per cent., and the entire bowel in 80 per cent., of his series of 200 cases. The deep ulceration generally exceeds the superficial ulceration, and the ulcers in the early stage may communicate with the intestinal lumen only by fistulous tracts which undermine the apparently healthy mucous membrane. Even when the ulcer's opening is large, its edges are generally undermined. Ulceration of the solitary follicles is less frequent than submucous necrosis. The ulcers may be round, serpiginous and are usually discrete. They may penetrate to the muscularis or to the serosa, which is sometimes perforated, thus causing diffuse or localized peritonitis. The peritoneum is often pigmented and shows fresh fibrin deposits or local adhesions. Secondary changes may include membranous diphtheroid deposits (usually streptococcic), diffuse sloughing of entire blocks of the intestinal wall, gangrene, and great hyperplasia of the mucosa. In chronic cases the muscularis frequently thickens, which may greatly narrow the lumen of the intestine. The small intestine may atrophy or hypertrophy. Healing ulcers produce pigmented stellate scars which sometimes cause stenosis. Amœbæ have been seen in the mesenteric bloodvessels, by which they reach the portal circulation (pylephlebitis, liver abscess). The liver shows focal necroses which are probably toxæmic, and, in a large percentage, it shows solitary or multiple abscesses. The single, large abscess generally develops in the right lobe near the diaphragm or colon. The multiple small abscesses are also superficial. In both types there is central necrosis and softening, the contents are yellow, green or chocolate-colored, contain fat droplets, liver detritus, and the amœbæ lie on the abscess walls. In the older cases there is a firm connective tissue capsule but round cell infiltration is as infrequent as it is in the intestine. Amœbæ may be found in the sputum when the liver abscess ruptures into the lung, or in the pus of secondary empyema. The pathology of complicating myelitis is disputed, but the pseudorheumatism and neuritis are probably toxæmic. The diagnosis is easy on careful microscopic examination of the stools. The mortality ranges from 1 to 20 per cent.; 7 per cent. of the deaths in Cairo are dysenteric.

III. Indeterminate Dysenteries.

The classification and cause of these forms are not yet defined; but some are apparently primary and others are secondary infections. Various microörganisms have been found, such as the colon bacillus, proteus, pyocyaneus, pneumococcus, streptococcus, and staphylococcus, but their action is still undetermined. Varieties: (*a*) *Catarrhal* dysentery, in which the mucous membrane is injected, cloudy, and infiltrated; the solitary follicles are swollen and often ulcerated,—the "acute follicular colitis" seen especially in children; the ulcers are round, deep, and small. In some instances there is also inflammation in the small intestine. The symptoms are usually mild pain, and muco-bloody evacuations. (*b*) The *croupous* or diphtheroid dysentery represents a severe stage or degree. The epithelium necroses, the submucosa is considerably infiltrated, and there are membranous deposits on the rugæ over slight superficial ulcerations, or there may be deeper, wider ulcers with great thickening of all the intestinal layers. This may occur (1) as a primary infection, with dysenteric symptoms, marked typhoidal symptoms, fever and chill, and frequently has a fatal outcome; or (ii) as a secondary incident in various infections; *e. g.*, in pneumonia particularly, in typhoid, scarlatina, diphtheria, cholera, syphilis, cardiac affections, cachectic conditions, mercurial or other poisonings and in uræmia. This secondary form may produce marked symptoms or may be clinically latent. (*c*) The *gangrenous* dysentery is attended by the same changes, but results in enormous swelling, softening, and sloughing of dark, gangrenous shreds, measuring one-quarter to three-quarters inch in thickness, and sometimes resulting in tubular sloughs of the larger part of the intestinal circumference. Perforation sometimes occurs. In the Cochin China endemic form, enlarged glands, splenic tumor and intumescence of the liver are found. *Coprostasis, foreign bodies and parasites* are promoting factors in all these types. In some cases the Cercomonas, Trichocephalus dispar, or Anguillula stercoralis may be found. The Distoma hæmatobium (Bilharz) may cause symptoms closely resembling those of dysentery; bloody urine, and strangury result from invasion of the vesical venous plexus by the Distoma; anatomically, hypertrophy of the mucosa, even to the extent of small or large polypoids, may result, as well as superficial ulceration and infiltration of the submucosa.

Treatment of all Forms.—**1. Specific Treatment.**—This is limited to the bacillary type. Shiga's bactericidal serum seems to have reduced the mortality at least one-half and to have shortened the course; in mild cases one and a half to two and a half drams [6 to 10 c.c.] are injected once; in medium cases two and a half drams [10 c.c.] twice, and in severe cases one to two ounces [30 to 60 c.c.] daily. The best results are obtained by early injection.

2. Prophylaxis.—Drinking water should be boiled, and fruits and vegetables should be cooked. In the tropics, sudden variations of temperature are to be carefully avoided.

3. Diet.—Acute and chronic cases must be carefully dieted and the patient must be kept in bed. Boiled milk, raw or slightly cooked eggs, or the whites of eggs beaten with water should be given to relieve colic and tenesmus. The nearest approach to solid food which is allowable is well-cooked rice or sago. Broths and cold drinks irritate the stomach and excite peristalsis. Some lemonade is permissible.

In convalescent acute or in chronic cases, solids should be given late, commencing with well-cooked tender meat and starches, and avoiding vegetables and fruit until convalescence is absolutely established.

4. **Eliminative Treatment.**—Sodium or magnesium sulphate are given in early cases to cleanse the bowel and to minimize the danger of ascent from the lower intestine (ʒj every two hours until the bowel is thoroughly emptied). Aromatic sulphuric acid mxx p. c. is sometimes given after the saline treatment. Castor oil is a mild laxative but is of little use except in the early stages. Calomel is considered beneficial by some writers, by whom it is given in frequent doses and in small amounts until slight mercurialization is noted.

5. **Antisepsis.**—Beta-naphthol and salol (phenylis salicylas) āā gr. x, t. i. d. or q. i. d., are sometimes helpful, although probably but small parts of the drugs reach the larger intestine. Bichloride of mercury, gr. $\frac{1}{30}$ to $\frac{1}{20}$, is efficient in some cases.

6. **Ipecac.**—This was introduced from Brazil, by Piso, in 1648. It is considered almost specific in India and Brazil, but, like the drugs above named, has little effect in the amœbic type. Frayer held that it reduced the mortality from 11 per cent. to 5 per cent. in his series. In India it is given as follows: food is withheld for four or five hours, and then a hypodermic of morphine, or opium by mouth, is administered to quiet the stomach and lessen nausea; 15 grains of powdered ipecac are administered every hour for two, three, or possibly four, doses. If the first dose is vomited, the opium and ipecac are given again. A mustard plaster should be applied to the epigastrium or a turpentine stupe to the abdomen. In Brazil, a decoction is preferred. The *Pulv. ipecac. et opii* and salol may be given āā gr. x for a few doses.

7. **Pain.**—Pain necessitates the use of opium, which also decreases tenesmus and peristalsis. It is given most efficaciously by rectum in a suppository, or, if poorly retained, by enema, mixing Tr. opii deodorat. ♏ xx with an ounce of starch water.

8. **Local Measures.**—Under this caption the use of *bismuth* by mouth may be mentioned, because its action is for the most part local; doses of ʒj to ij are necessary, and even two ounces daily are given. Musgrave dislikes the use of bismuth because it cakes in the ulcers. *Lavage* is difficult because of tenesmus and bowel intolerance, and is not without danger, for perforation has resulted from copious irrigations. The rectal tube must be introduced carefully as the rectum is very sensitive. Tenesmus may be avoided by injection of a cocaine or laudanum solution, or a cocaine, opium, or iodoform (five-grain) suppository.

℞

Ac. tannici	gr. xv.
Extr. opii	gr. iv.
Extr. belladonnae	gr. j.
Olei theobromatis	q. s.

M. et ft.— Suppos. No. iv.

Carbolic acid and bichloride of mercury solutions are seldom employed; Bellei employs as an antiseptic and anæsthetic douche, carbolic acid ♏xx, tannin ʒj, marshmallow root ℥ij to a quart of warm water. Among the other solutions of value are quinine (1 to 1,000 or 5,000, Osler), or a weak peroxide solution for the amœbic variety, and Thiersch's solution of boric and salicylic acids; all enemata should be warm. Strong silver solutions (1 to 500 or less) cause great pain, and weak dilutions are inert; silver, however, is most suitable for chronic cases. In the author's experience, Kartulis's method is the most valuable;—*e.g.*, ½ per cent. solution of tannic acid, injecting as much as the bowel will tolerate and retain. It kills the amœbæ and two or three injections are usually sufficient. Kartulis has "never been disappointed in hundreds of cases." It may be given in the genupectoral position as an enteroclysis, as advocated by Cantani in cholera. An irrigator at an elevation of two feet should be used and the ordinary rectal syringe should be avoided.

9. **Turpentine Emulsion.**—This may be used for tympanites, and the ice-bag, or Leiter's coil with opium, may be used for hemorrhage or peritonitis.

10. **Colostomy.**—In chronic cases, colostomy has been advocated.

MALTA FEVER.

Definition.—A specific infection, which is endemic on the coast and in the islands of the Mediterranean Sea, is caused by the *Micrococcus melitensis*, somewhat resembles typhoid, but is characterized by a much longer course, by recurrent, irregular, "undulant" fever, severe sweats, splenic tumor, and rheumatoid or neuralgic pains.

History.—Observed about a century ago, it was first described by Marston (1858) as a separate disease. Marston, Bruce and Hughes, English army surgeons, have been the largest contributors to our knowledge of Malta fever. Its distribution is fairly well covered by its names,—Mediterranean, Rock or Gibraltar fever, Malta fever, Neopolitan or Levant fever. It has also been seen on the Danube, Red Sea, Bosphorus; in China, India, the Philippines; in the Canaries, Azores, and Antilles; in Venezuela (Caracas fever), Brazil, and South Africa. Less than a dozen cases have come to this country. Musser and Sailer published an account of the first one.

Etiology.—The *Micrococcus melitensis*, which was discovered by Bruce (1887), is round or oval, measures 0.3 μ, often occurs in chains, is non-motile, and stains with methylene blue, or gentian violet, but not by Gram's method. It grows best on 1.5 per cent. beef-water, with peptone and agar, in which state cultures grow in the form of small pearl-like spots. These after a few weeks become yellowish-brown. The bacillus is not found in the blood, but in the urine, even after recovery, and in the liver and spleen. The apes inoculated by Bruce and Hughes showed a typical clinical course and postmortem findings. The disease prevails chiefly between the sixth and thirtieth years, in the hot, dry weather from May to mid-October, and in old and unhygienic buildings. It is not directly contagious. The *atrium* is the lungs (by dust, Hughes), or the alimentary tract (by food or drinking-water, Bruce). In Strong's laboratory infection, the conjunctiva was the point of entrance. It has been recently suggested that mosquitoes and goats convey infection.

Symptoms.—Symptoms appear after an *incubation of six to ten days.* Some of them so resemble those of typhoid and malaria that the three diseases were formerly confounded. The *fever* rises gradually, reaching 104° to 105° after a few days. The face is red, there are chilly sensations, frontal headache, insomnia, depression, and pains in the back and legs. Epistaxis is occasional. The tongue is coated, is at first moist, then dry, and fissured, and sometimes bleeds. The tonsils are frequently swollen, and the pharynx is red or at times ulcerated. Anorexia, nausea, epigastric pain and tenderness, and constipation, are the rule, and vomiting, icterus, meteorism and diarrhœa, with dark, malodorous, mucous, and even bloody stools, are sometimes seen. One of the chief anatomical findings is intestinal hyperæmia, and, in rare cases, swelling or ulceration of Peyer's patches occurs (4 per cent.). There is considerable bronchitis and the sputum is often streaked with blood. Dyspnœa is quite frequent. The pulse-rate is at first between 80 and 90; later it becomes faster, and is frequently attended by palpitation. Severe sweats occur, usually after midnight, and are accompanied by sudamina (*febris sudoralis*). Roseolæ are never observed. An acute splenic tumor and hepatic intumescence develop, which are due to degeneration and infiltration by round-cells.

After one to three weeks, the *fever*, which at first is continuous, remits, other symptoms abate, and convalescence is apparently established; but the symptoms again return, and frequently recur severa times. The fever curve shows waves accompanying each relapse (literally "undulant fever"). The patient becomes weak, emaciated and pallid. There is no leukocytosis, the urine does not show the diazo reaction, and albuminuria is very uncommon.

Complications.—Complications, which are uncommon in typhoid, are frequent. *Pseudorheumatism* in one or more joints occurs in 50 per cent. of the cases (Bruce) and is accompanied by redness, swelling, and pain in the shoulders, knees, hips, and sacroiliac joints, and sometimes also in the bursæ, periosteum and fibrous tissues. *Neuralgias* are common. *Orchitis* and epididymitis occasionally develop, and are usually unilateral and ephemeral. Mastitis is very rare. Occasionally *polyneuritis* develops and is evidenced by partial anæsthesia, hyperæsthesia, especially of the soles of the feet, paræsthesia and rarely paralysis. These result from intoxication and exhaustion. Psychoses and disturbance of the special senses may result from Malta fever.

Skin eruptions occur late in the relapses and are chiefly erythematous or hemorrhagic, and are sometimes accompanied by hemorrhages from the nose, tongue, gums or lungs; desquamation in scales, in large shreds, particularly from the soles of the feet, and falling of the hair, are common. Glomerular nephritis, pneumonia, and endocarditis are very unusual. In convalescence, tuberculosis not infrequently develops.

Course and Prognosis.—In the majority of cases relapses occur after two or three weeks. The fever sometimes remains near 104° continuously for months. The average duration of the disease is ninety days, but Bruce records a case which lasted over two years. Hughes describes three types: (a) the *undulant*, as above described, which is the most common type; (b) the *malignant*, which ends in death in from one to three weeks from weak heart, from hyperpyrexia (110° or 111°), or possibly from lobular pneumonia; (c) the *intermittent*, which lasts for months. The severity of the infection varies; Melland found that 50 per cent. of cases in the Canary Islands lasted from two to three weeks, and in only 10 per cent. did the protracted undulating type occur. The death-rate is constantly decreasing. In the seventh decade of the last century it was over 3 per cent., but it is now under 1 per cent. *Immunity* after one attack is usual, but is not absolute.

Diagnosis.—The diagnosis of Malta fever is often difficult, especially the differentiation from typhoid. In Malta fever the roseolæ, and the bacillæmia are absent; the blood does not respond to the Widal test; nor does the urine show the diazo reaction. The course of Malta fever is longer than that of typhoid, the sweats are more severe, and the joints are inflamed; orchitis, and neuralgias develop. A positive diagnosis can be made by the use of Wright's *serum reaction.* The blood responds to this test after the fifth day, and continues to do so, in some cases, for three years. Agglutination may be obtained equally well with the living or dead cultures; the dilution of the blood is 1 to 50, and the time for the test is limited to one hour. The severe bronchitis, fever, and sweats may cause confusion with tuberculosis. Indeed, the older writers spoke of Malta fever as "Mediterranean phthisis." The bronchi in Malta fever are intensely hyperæmic and the sputum is frequently blood-tinged. Malaria, ulcerative endocarditis, febrile pseudoleukæmia, and liver abscess, are differentiated by the agglutination test.

Treatment.—There is no specific treatment, though serum therapy is likely to prove valuable (Aldridge, Fitzgerald, Ewart). Quinine and arsenic are ineffectual. Headache and backache are relieved by antipyrin and morphine; insomnia by bromides, chloral and hyoscine; diarrhœa by tr. ferri chlor.; pseudorheumatism by heat, by wool investment, by tr. iodi, locally, and by salicylates, internally; orchitis, by local heat and by suspensory elevation. The general therapy is the same as it is in typhoid. Milk diet lessens the chance of intestinal complications, though Dalton advocates a solid diet. In convalescence a change of climate is necessary.

BERIBERI.

Definition.—Beriberi, or *polyneuritis endemica* (Baelz, Scheube), is an endemic and epidemic affection of the tropics and subtropics, of unknown etiology, characterized anatomically by degenerative inflammation of the peripheral nerves, and clinically by motor and sensory disturbances, anasarca, cardiac disturbance and by a subacute or chronic course. *Beri* is said to signify a sheep's gait, and *kakke* also means disturbance of the gait.

History.—Beriberi is mentioned in the oldest Chinese writings and was observed in the Roman legions, 24 B. C., by Strabo and Cassius. In the seventeenth century, beriberi was noted in Brazil and in the Malay archipelago; in recent times (1835) it has been described by Malcolmson in India; by Baelz, Scheube, Grimm, and Miura in Japan; by the physicians in the Dutch Oriental colonies; and by Birge, Putnam, and Bonduret in this country.

Distribution.—The main foci of beriberi are (1) the Malay archipelago, Sumatra, Borneo, and Java; also China, India (where it is decreasing), the Philippines, Japan, and even Australia. The mortality has been very high among the Chinese coolies in the Dutch possessions, where epidemics developed from endemics. (2) Africa, including the mainland and the islands. (3) America and the West Indies, Brazil, Honduras, and Cuba. Cases have appeared in Dublin (1894–1898, with forty-two deaths), in England, and in Paris. Scheube doubts that the cases which developed in the United States, in the epidemic at Tuscaloosa, Alabama, were beriberi, but from description, they are apparently identical with oriental beriberi.

Etiology.—The etiology is greatly disputed and there are two distinct views: (1) that it is an *acute infection* (Baelz, Scheube), because it occurs in the young (fifteen to thirty-five years) and healthy, it occurs in certain localities as epidemics and endemics, it occurs in the summer months, and in moist, alluvial land near the sea, and near the river mouths,—*i. e.*, the same telluric conditions promote beriberi, which promote malaria. It is considered by some persons to be contagious, though no constant microörganism has been found. The fact that many cases develop in houses, barracks, and ships is an argument in favor of the theory of contagion. In the 80,000 cases which developed among the Japanese troops in the Russo-Japanese War, Kokubo frequently found a coccus. (2) The second view is that beriberi results from *food*. Shelled rice is said to have caused it in the Dutch East Indies, and raw fish is considered the cause of the disease among the Japanese (Miura). The use of fish in the navy was forbidden by Takaki, and the mortality fell from 25 per cent. to almost nothing. Natives are more often affected than Europeans, and males more frequently than females. Improper diet may be a cause of beriberi or at least an indirect factor. In 1879 there were 8,197 cases in Japan and in 1891 this number decreased to one. Poor food, overcrowding and lack of hygiene are important and avoidable factors.

Symptoms.—The incubation is long, probably several months, and the development of the disease is due to repeated infections. The *symptoms* are threefold: (*a*) those of multiple neuritis; (*b*) dropsy, and (*c*) cardiac insufficiency.

(*A*). **Neuritic Symptoms.**—A more detailed description of these symptoms will be given under MULTIPLE NEURITIS. Anatomically there is the same symmetrical degeneration (or inflammation) in the distal parts of the peripheral nerves to which, however, it is not limited; the phrenic and pneumogastric nerves and the cardiac, renal, and solar plexuses, are often involved; the nuclei of the sheath multiply; the axis cylinders degenerate; there is a lumpy degeneration in the medullary sheath; and later, there is connective-tissue hypertrophy. In eight cases there were degenerative changes in the cord, anterior horns, and posterior ganglia. Clinically, the neuritis comes on gradually and runs a chronic course, which is varied by acute exacerbations.

1. MOTOR SYMPTOMS.—The muscles of the calves of the legs, the knee extensors, and the abductors and flexors of the thigh are weakened, flaccid, swollen and are frequently atrophied. The muscles are more frequently tender than the nerve-trunks. In severe cases the trunk, arms and, rather frequently, the face, are involved. The muscles are anatomically much degenerated. Phrenic weakness and vagus symptoms, such as rapid heart, vomiting, epigastric oppression, or digestive disorder are not infrequent; the lungs are sometimes acutely emphysematous, which results from involvement of the pulmonary plexus. Symptoms of involvement of the larynx, of glossopharyngeal, hypoglossal or ocular participation are more rare. Cramps in the calves of the legs and fibrillary contractions are frequent, but ataxia is rather uncommon, although the name beriberi refers specifically to disturbed locomotion. The gait is described as like that of one walking in wet clothes or wading in water. Tremors are rare.

2. SENSORY SYMPTOMS.—Paræsthesia is common. Hypæsthesia is most marked in the distal parts, though the sole of the foot is exempt; it is more frequent than anæsthesia, which is rarely complete. Hyperæsthesia is not very frequent, but may occur over the spine and abdomen. Pains over the joints and intercostal spaces are quite common.

3. TROPHIC SYMPTOMS.—These are uncommon. They include joint relaxation, herpes or skin atrophy.

4. REFLEXES.—The skin reflexes are seldom abolished. Reduction of the tendon reflexes develops equally with the paralysis.

5. DEGENERATION.—Slight reaction of degeneration is common and may be marked in severe cases. It occurs particularly in the leg.

(*B*). **Dropsy.**—Dropsy to some degree occurs in 97 per cent. of cases; it may be marked in the legs, and face, and in all the serous chambers. It is most severe in the pericardium, is quite severe in the pleuræ, and is least severe in the peritoneum. It is caused by cardiac and perhaps also by vasomotor involvement. Dropsy invariably occurs in fatal cases.

(*C*). **Cardiac Insufficiency.**—This occurs particularly in the acute cases; death may result in a day, and acute forms in 65 per cent. of the cases lead to a protracted and distressing death within two weeks. There is palpitation, cardiac or epigastric oppression, or actual pain; tachycardia, dyspnœa, and cyanosis develop; the right ventricle is dilatated, the first apical tone is weak; there is splitting of the heart tones, gallop-rhythm, and systolic or even diastolic functional bruits; the urine is decreased in amount, in phosphates, and in uric acid; the liver is turgid. Anæmia is marked and the leukocytes may be increased. The heart failure may be accompanied by vocal cord paralysis and weakness of the respiratory muscles. Acute pulmonary œdema is usually fatal. The heart muscle is pale, friable, colloid, or is the seat of acute myocarditis.

The *types* of beriberi only accentuate some of the above-named symptoms, any one of which may stand out prominently. (1) The *rudimentary form* may begin with slight fever, and with catarrh of the nose, air-passages, and alimentary tract. There is a slight œdema of the ankles; muscular weakness, which occurs especially in the legs, indicates neuritis; muscular tenderness, paræsthesia, hypæsthesia, and palpitation are also symptoms. (2) The *atrophic form* is marked by atrophy and paralysis. If œdema does not develop, both of these types may be called the "dry forms." (3) The *dropsical form* is characterized by much transudation and by pronounced insufficiency of the heart. (4) The *acute cardiac or pernicious form* (*v. s.*).

Diagnosis.—Diagnosis of beriberi is difficult when it occurs in the temperate countries. The leading points are (*a*) the multiple neuritis, which is most marked in the legs; (*b*) vagus neuritis and cardiac insufficiency; and (*c*) dropsy.

Prognosis.—The death-rate varies, even in the same endemic locality. In Japan and the Dutch Indies it ranges between 3 and 4 per cent.; in Brazil it formerly varied from 50 to 75 per cent.; and in the Malay archipelago it reached 40 to 50 per cent. Death results primarily from heart failure, secondarily from paralysis of respiration, less frequently from uræmia, embolism of the lungs, aspiration pneumonia, intercurrent disease, or marasmus. Even in recovery, permanent residua are common, such as weakness, obstinate contractures of the legs (necessitating tenotomy), absence of knee-jerk, and cardiac instability and hypertrophy. *Recurrences* during ten, twenty, and even thirty years, are common.

Treatment.—In the early stages of the disease purgation and salicylates are beneficial. In the later stages, electrotherapy and other measures which are employed in multiple neuritis (*q. v.*) are indicated, and the heart should receive stimulation. Venesection is advisable in order to relieve the overladen right heart and the dyspnœa, and promote action of the kidneys. Digitalis is strongly recommended by some, whereas others fail to find any benefit from its administration and attribute such a result to degeneration of the vagus. In dropsical forms it is helpful as is caffeine. Blisters and the ice-bag over the heart, bromides and belladonna, may mitigate cardiac unrest and palpitation. (See TREATMENT OF VALVULAR DISEASE OF THE HEART.)

ANTHRAX.

Anthrax is one of the *zoönoses*—(diseases acquired from animals). Glanders, foot-and-mouth disease, vaccinia and hydrophobia constitute the remainder of this group. Actinomycosis is less a zoönosis than a disease acquired from a common cause by man and beast. Anthrax is also called splenic fever, pustula maligna, and carbunculus contagiosus.

Definition.—It is an acute infection caused by the Bacillus anthracis, and is usually communicated to man from the herbivora.

History.—Over a century ago Morand and Fournier distinguished between the simple carbuncle and that of anthrax, which could be inoculated. The bacilli were seen by Pollanders (1855) and Branell (1858), but Davaine (1864–1873) worked out all the essentials of the disease and noted that the organisms were bacteria. It remained for Koch with his epoch-breaking technique to find the spores and to absolutely differentiate the disease, which is now one of the best known maladies.

Bacteriology.—The anthrax bacillus is the largest pathogenic microbe, and measures 3 to 10 by 1 to 1.5 μ. In growths it may occur singly or in chains of 2 to 10 bacilli. The ends are slightly rounded, and the chains somewhat resemble the phalanges of the finger for the organisms lie end to end: They are non-motile, and transparent, and may be stained by Gram's method, and also with various dyes, especially with Bismarck brown. At high temperatures they grow in long, thread-like, winding, segmented filaments. Spores are absent in the cadaver or in the living tissue, possibly because of the presence of carbonic acid; in the living tissues they multiply by fission.

In attenuated cultures, one spore develops for each bacillus; these become bacilli, which, when young, may be slightly motile, and are extremely resistant to drying and to high temperature.

Cultures.—The bacillus is an obligate aërobe which requires oxygen and carbon dioxide, and grows best on neutral or weakly alkaline media and at a temperature between 95° and 102°. Stab cultures of agar are characteristic. A white, milky yet transparent and band-like growth appears in one or two days, which develops horizontally in blood serum, and grows downward at an acute angle, so that in three days it resembles a quill with a feathered upper end. Growths on gelatin appear to have a thick, wooly covering. The gelatin liquefies after a few days, and a white, granular sediment falls to the bottom of the tube. The bacillus loses its virulence in blood which has been rapidly dried, but in blood which has been slowly dried, it develops spores.

Anthrax is *endemic* and *epidemic* in Europe and Asia. There are persistent foci in Germany, especially in Saxony, parts of Bavaria, and in Frankfort; in France, in the provinces of Burgundy, and Auvergne; in Hungary; in Russia, principally in Siberia (the "Siberian plague"); in China, India, and in South America. Anthrax is rare in America, though small epidemics have appeared in Delaware and in Pennsylvania. Among animals, the herbivora are most often affected, especially sheep and cattle, and in Russia, horses. Omnivora are more susceptible than carnivora. Mice, guinea-pigs, rabbits, dogs, and fowls, rarely acquire the disease. Pasteur thought that earth-worms carried the bacilli from buried bodies to the surface. Transmission of the spores by means of snails, flies and fleas is possible. Spores reaching water may be widely disseminated. The germ may have a facultative saprophytic existence on vegetable media.

Infection may be carried to man from animals, and, on rare occasions, from other human beings. Of Koranyi's cases, 65 per cent. were attributed to sheep and 35 per cent. to cattle; Bourgois considers that the greatest percentage comes from cattle, the second largest from sheep, and the third largest from horses.

I. External Anthrax.

External anthrax is by far the most common type. The atrium is through an abrasion of the skin, and possibly through intact hair-follicles. Koch observed that sheep were infected by fly-bites on the exposed skin of the neck. Shepherds, coachmen, and all those who may attend diseased animals; butchers, tanners, wool-sorters and persons who may handle diseased hides or meat; and saddlers, tanners, rag-pickers and gardeners are most likely to acquire the disease. The bacillus may enter the system through insect-bites, and bruises. Jacobi records infection from a hypodermic needle which was first used on a patient suffering from anthrax. The spores are more virulent than the bacilli.

Site of the Pustula Maligna.—In Koch's collection of 1,077 cases, the exact distribution is noted in 966; 51 per cent. developed on the head and face, 38 per cent. on the upper extremities, 2 per cent. on the lower, 5 per cent. on the neck, and 4 per cent. on the trunk. That is, the exposed parts are the usual seat of primary infection. The lesion is usually single, but several pustules or carbuncles may develop, possibly from inoculation by the finger-nails in scratching.

Symptoms.—The incubation lasts two or three days. A small red prominence is first seen, which resembles an insect-bite. It itches, and burns, and in about twelve hours develops a small vesicle. This is filled with yellowish or blood-stained serum, which is purulent if ruptured or scratched, and which dries with scab formation. It develops a dark, dry, central, necrotic, leathery slough, which is caused by streptococcic infection, and is surrounded by dense round-cell, fibrinous, and often hemorrhagic, infiltration. This in turn is surrounded by œdema of the connective tissue, in the deeper lymphvessels of which the Bacillus anthracis abounds. If the carbuncle becomes extended, a circle of new vesicles, or even two or three concentric circles of vesicles appear, which fuse and increase the infiltration and œdema. The eyes may be closed by swelling of the lids, or the lips may be so swollen that the jaws cannot be opened. Demarcation may now occur, followed by sloughing; granulation tissue and cicatrization or *general infection* develop, and the adjacent lymphvessels and glands become infected, soft, and hemorrhagic. This usually occurs on the fourth to the sixth day, or earlier in cases of violent infection or of reduced physiological resistance. It is marked by depression, by headache, backache, pain in the limbs, chilliness, continuous or remittent fever, soft pulse, rapid breathing, vomiting of mucus or blood, vertigo, leukocytosis (20,000 to 70,000), colic, diarrhœa, and collapse. Hemorrhages may occur or even gangrene may develop around the carbuncle. Anthrax bacillæmia is sometimes found. The mind is usually clear, although delirium, convulsions or coma may develop, and death may occur on the seventh to ninth day with an antemortem fall of temperature. Toxæmia and septicæmia cause the symptoms given above. In fatal cases there is only slight rigor mortis but rapid decomposition, dark fluid blood, hemorrhages into various tissues, swelling of the lymph glands, and lymphadenoid tissue of the intestine and sometimes of the spleen. In the liver, kidneys, brain and meninges, deposition of bacilli, and hemorrhages and swelling may be found; the bacilli are seen less in the large vessels than in the capillaries of the brain, kidneys, lungs, spleen and heart. Bourgois describes another cutaneous form of the disease in which there is great œdema (*charbon blanc*, anthrax œdema), but no pustule or carbuncle. He considers that it occurs more often on the trunk of the body than does the carbuncle.

Diagnosis.—In most cases this is not difficult. The *simple carbuncle* resembles that of anthrax, in that it is also hard, has a prominent central necrosis, is surrounded by œdema. and sometimes is vesiculated. It usually causes great pain, has numerous openings which discharge ordinary pus, and is often malodorous. The *anthrax carbuncle*, however, may be differentiated by the presence of bacilli, by depression of the necrotic centre, by the corona of vesicles, by more rapid evolution, by greater œdema, and by lack of odor. *Glanders* may be distinguished by involvement of the mucosæ, by painful nodes without eschars, and by ulcerations which discharge the Bacillus mallei. It is often difficult to recognize Bourgois's *charbon blanc*.

Prognosis.—The prognosis is more favorable in children than in adults, in cases with local than in those with general symptoms, and in those with simple carbuncle than in those with œdema. In Koch's collection of cases, 68 per cent. recovered. Nasarow's figures show 26 per cent. mortality when anthrax occurs in the head and face, 23 per cent. when it occurs in the trunk, 19 per cent. in cases of infection in the neck, 14 per cent. in the upper, and 5 per cent. in lower extremity localization.

Treatment.—**Prophylaxis.**—Infected animals should be cremated entire and their hides and wool should be destroyed. Toussaint (1880) and later, Pasteur, Chauveau and Colbert, attenuated the virulence of anthrax cultures by exposing them to sunlight or to increased atmospheric pressure, and used them to immunize animals. In France (1882–1893), 1,788,677 sheep were thus inoculated, with a resulting mortality of 0.9 per cent. and 200,962 cattle were also inoculated, and a 0.3-per-cent. mortality occurred.

Active Treatment.—Wounds in those who have been exposed to infection should be promptly washed with strong sublimate solution. Cauterization with the live cautery, with carbolic or nitric acids, caustic potash; surgical excision through sound tissue; injections of iodine or corrosive sublimate; these have all been recommended, but it is also maintained that equally good results may be obtained by elevation, by the use of mercurial paste, and by expectant treatment, which at least avoid dissemination of the parasite through sound tissues. Erysipelas serum has apparently been useful in anthrax infections. Sclavo (1903) reported 164 cases which were treated by serotherapy; the mortality was but 6 per cent. In 1,073 cases treated with Mendez's serum, the mortality was but 4 per cent.

II. Internal Anthrax.

Internal anthrax is less frequent than the external type. It occurs in the alimentary and respiratory tracts.

1. **The alimentary form** (mycosis intestinalis), was first described by Wahl and Recklinghausen, and was more fully discussed by Waldeyer and Münch. Bacilli may enter the system through cuts in the mouth and throat, but usually directly invade the intestines and stomach, which are more frequently infected by spores in milk or water than by diseased meat. The gastric juice kills the bacilli but not the spores. Peasants often eat the pickled meat of diseased animals without being infected. In Wurzen, Saxony, there were 206 cases of intestinal anthrax in 1877.

Symptoms.—There may be prodromal chills, fever, epigastric or lumbar pains, or depression. The onset is, however, usually abrupt, with fever, coated tongue, thirst, and vomiting of mucus or of blood. This is followed by sensitiveness or great pain over the bowels, by serous and afterward by bloody movements, meteorism, dyspnœa, weak pulse, and by symptoms of collapse which resemble those seen in the generalization of infection in the cutaneous form. Secondary carbuncles, or more often hemorrhages into the skin of the neck and of the abdomen, may develop. The course is usually severe and, in some instances, is peracute; death may result in two or three days, in well marked cases, although recovery is possible.

Diagnosis.—The history of the case, the finding of bacilli in the stools and in the blood, or the findings at autopsy, determine the diagnosis. In the small intestine there are changes like those of the cutaneous carbuncle. These consist of localized induration with central sloughing which usually occurs opposite the mesenteric attachment and is accompanied by surrounding hemorrhage and œdema in the mucosa, and of swelling of the solitary follicles, of Peyer's patches, of the mesenteric glands, and of the omental and retroperitoneal cellular tissue. The bacillus is found in these lymphadenoid structures in large numbers and sometimes also in the chylevessels and portal bloodvessels. The greatest changes most frequently occur in the upper small intestine, sometimes in the stomach or ileum, seldom in the colon, and most rarely in the rectum. More than thirty or forty foci may be seen in the small gut, but the lesions in the stomach or rectum are fewer in number and more discrete. Secondary embolism of the intestinal arteries may result from anthrax of the skin, in which event hemorrhagic stools are uncommon. Serohemorrhagic fluid may be found in the peritoneal cavity. Treatment is without effect, though ipecac, calomel and bichloride of mercury are often recommended.

2. **The respiratory form** was described by Bell (1840), and by Spear and Carpenter (1881) in England, as the "wool-sorters' disease"; and by Eppinger (1894) in Steiermark, Austria, as the "rag-sorters' disease." Infection may, on rare occasions, occur in the nose, larynx, tonsils (Wiggin), or bronchi. Particles of inhaled chalk are thought to facilitate infection by producing minute traumata, but the bacilli are apparently capable of entering the uninjured bronchioles and alveoli. Wool-sorters' disease results especially from infection by hides imported from Russia and Brazil, and, in 1897, anthrax developed in this country from hides of Chinese origin.

Symptoms.—Prodromes are uncommon. The symptoms begin suddenly with chill and high fever, tachypnœa, and dyspnœa; there are bronchitic and irregular pneumonic findings; and the sputum, which contains in some instances the pathognomonic bacilli is viscid or hemorrhagic. The later symptoms are cyanosis, which is caused by diffuse mediastinal infiltration; serous pleurisy, weak heart, and the terminal collapse and antemortem fall of temperature. Icterus, hæmaturia, glycosuria, and anthrax endocarditis have been observed. The sensorium is clear to the end in most of the fatal cases, though convulsions and coma, which in some cases are due to meningeal hemorrhage, may occur. The patient may die as early as the second or third day. The mortality in rag-sorters' disease is 50 to 87 per cent., and is rather lower in wool-sorters' disease. At the autopsy the special findings are as follows: the bacilli enter the ultimate air-tubes and alveoli, wherein they may produce alveolar desquamation and cellulo-fibrinous exudation—the analogue of the carbuncle—and much serohemorrhagic exudate. The latter is comparable to the skin œdema.

In cases where there is much cellular exudate the bacilli become decreased, and recovery may result from demarcation; where there is much serous infiltration the bacilli are abundant, and extension of the infection is likely along the lymphvessels in the direction of the mediastinal cellular tissue, of the peribronchial lymph glands and of the pleuræ, which may hold as much as four quarts of anthrax-infected serous blood-stained exudate.

Treatment.—The disease may, to a great extent, be prevented by sterilization of the germ-laden hair, of hides, or of hair used in the manufacture of paper. The actual therapy is wholly symptomatic and supportive.

3. **Anthrax septicæmia** is a rare form in which the blood is charged with bacilli, though no point of entrance can be found. It is almost always fatal.

GLANDERS.

Definition.—An acute or more often a chronic contagion, which is caused by the *Bacillus mallei*. It is characterized by the formation of nodules and ulcers, which occur chiefly in the nose and skin, and less often in other structures. It is observed largely in the horse, which is the main source of human infection.

Etiology.—Glanders and farcy were known to Aristotle and Hippocrates. The Bacillus mallei was discovered by Loeffler and Schuetz (1882), and independently by Bouchard, Capitau, and Charrin. By its discovery, glanders and farcy were identified as one disease. Weichselbaum (1885) first found the bacillus in human infection. It closely resembles the tubercle bacillus morphologically, but is shorter and thicker, and measures 2 to 5 by 0.4 to 1.5 μ; it is straight or slightly bent; its ends are somewhat rounded; it is usually single, but may lie in pairs or groups; and it has no movement. It stains with the alkaline anilines, easily in cultures but with more difficulty in sections, and the bacillus tinges unevenly, showing lighter areas, which are thought by some to be spores. It is a facultative aërobe and its most characteristic culture is that grown on the potato, in which a thin light yellow film develops in two days, soon becomes amber-colored, and in eight days assumes a weakly red tinge with somewhat greenish-blue borders. The *atrium* is usually the skin, through which the bacillus enters by some small, perhaps microscopic, lesion. Babes considers that infection may take place through the intact mucosa of the nose and air-passages. Horse-bites may cause inoculation. Infection may also occur through the conjunctiva, the nose, the respiratory tract, or very rarely, the digestive tract (Nocard). Man is usually infected by chronically diseased horses; man-to-man infection is far less frequent. The bacillus is found in the nasal nodes (*glanders*); in the skin nodes (*farcy*); in the lymphvessels and glands (in which its virulence is greatest); in the saliva, urine, sweat, semen and in the blood-current, where it occurs more frequently in man than it does in animals, for in the latter it is found in the most acute cases only.

The vitality of the bacillus is not great. It is soon destroyed in pus, but may endure for three or four weeks in the viscera of cadavers or three or four months in cultures; it is rapidly killed by sunlight, but persists two or more weeks in damp protected places. It is killed in two minutes at 212°, in fifteen minutes by 1 to 1,000 sublimate solution, and in one hour by 5 per cent. carbolic solution. Babes (1890) and Kalming (1891) isolated a toxin, and Kalming, during his researches, succumbed to acute glanders. Hellmann (1891) isolated mallein, and Babes (1892) morvine, both of which are successfully injected for diagnosis and for immunization. The horse, ass, and mule are the most susceptible animals, and from them, goats, sheep, cats, lions, rabbits and guinea-pigs may be inoculated. Babes found that in some localities half the horses have latent glanders. Cattle, mice, rats, and fowls are practically immune. Infection is carried by the nasal or cutaneous discharge, whence glanders and farcy are chiefly observed in stable-boys, coachmen, farmers and veterinary surgeons.

Symptoms, Pathology and Course.—These are considered under two topics: (*A*). Farcy, the cutaneous type; (1) acute or (2) chronic; and (*B*). glanders, the nasal type, (1) acute or (2) chronic. In most cases the types are not wholly distinct, though symptoms of one localization may prevail.

(*A*). **Farcy.**—1. ACUTE FORM.—The incubation is from three to five days, rarely two to three weeks, and is attended by depression, anorexia, nausea, headache, and leg pains. The infected point on the skin shows infiltration, which may heal only to break out anew. Lymphangitis and lymphadenitis are present, but are less marked than in the horse, and result in lymphangitic farcy-buds. Subcutaneous and cutaneous nodes or areas of diffuse infiltration develop. These swellings are classed with the infective granulomata, are composed of epithelioid, lymphoid, and white blood cells, and contain the bacilli, which are free, rarely intracellular, and most abundant in the centre of the node. Early sections are succulent and show central necrosis, which is followed by suppuration and ulceration. The ulcers are painful, deep, and crater-like, with sharply cut, everted borders, and with speckled bases; they have a thin, puriform, hemorrhagic, or ichorous discharge, and they often fuse. Thrombo-phlebitis, diffuse phlegmons, or gangrene may develop. The rise of the fever may closely resemble the ladder-like ascent of the temperature in typhoid. Chills are common. Polyarthritis and large muscular abscesses are frequent. In some cases a skin eruption occurs which is successively macular, papular, and pustular, and resembles that of smallpox except that it is rarely umbilicated; it may be scant and scattered, or very diffuse, and even confluent; and may affect the face, extremities, mouth, throat, and conjunctivæ. The patient is often delirious, and has an offensive diarrhœa; the pulse becomes rapid, and fresh skin nodes develop, which are accompanied by ulceration or perhaps gangrene.

The pulse is fast, small, and irregular; the urine contains albumin, or casts, or shows the diazo reaction, and death occurs in about two weeks from exhaustion or from lung inflammation. In the horse, acute farcy occurs with acute nasal localization.

2. CHRONIC FORM.—Chronic farcy shows the same skin changes, but they are very gradual in onset, slow in progression, are unaccompanied by essential inflammatory reaction or by lymphatic invasion. For the first month or two there are pains in the limbs and joints, and after a few weeks, indolent swellings in the extremities and in the periarticular tissues appear. These are followed by ulceration. The ulcers may cicatrize, but break out again after several months, and therefore somewhat resemble lupus, which heals on one side and develops on another. Muscular abscesses may appear, and are very often located in the pectoralis, biceps, brachialis, and gastrocnemius. The course lasts for months or years (two to eleven). Recovery occurs in 50 per cent. of the cases (Bollinger), or death results from exhaustion and acute dissemination. In horses the lymphvessels and glands are greatly swollen, and appear as sausage-shaped masses, on account of which fact the term "farcy" was used by Vegetius. In horses the skin eruptons and orchitis are more frequent than in man.

(*B*). **Glanders.**—1. ACUTE NASAL FORM.—The conjunctivæ are sometimes swollen, but the process generally begins in the nose after an incubation of three or four days. Miliary nodules develop. These are elevated, are yellowish-white in the centre, grow to the size of a pea, are surrounded by a pale red zone, and become ulcerated. New nodes form on the edges of the ulcers, which break down, fuse with them, and discharge a thin, puriform, dirty, sanguinolent fluid. Diphtheroid deposits or crusts form; desiccation may also occur. Ulceration may erode the bone or cartilage, and cause perforation of the septum. The nose is swollen and eroded externally, and the lesions may resemble those of erysipelas. The process reaches the mouth by the lymph channels or by thrombophlebitis. Perforation of the palate may occur; the tongue, gums, or tonsils are invaded; the angular lymphatic, parotid and submaxillary glands are swollen, and the larynx, and finally the lungs, are infected. Beregin found that microscopic nodes may develop in nearly all the parenchymatous organs, the liver, kidney, spleen, and brain. In the lungs the nodes may resemble miliary tubercles, or actual hepatization (pneumonia malleosa) may develop, and is frequently accompanied by hemorrhage, abscess formation, gangrene, a distressing cough, and raspberry, mucopurulent, or sometimes stinking sputum. Secondary suppuration in the bones, muscles, joints or skin, may develop. The course is that of acute farcy, though perhaps more rapid, and is attended by drenching sweats, diarrhœa, clouded sensorium, and convulsions; and death occurs in one or two weeks. The blood is thin and the leukocytes are increased.

2. CHRONIC NASAL FORM.—This is similar to acute glanders except that it is chronic. It is less frequent, and in forms which are limited

to the nose, confusion with other diseases is frequent. When nasal involvement is slight it may clinically and pathologically resemble pulmonary tuberculosis, chronic coryza, pyæmia, or osteomyelitis. Recovery is infrequent, though healing by cicatrization may result. According to Babes, recovery may take place or, at least, the malady may "slumber," possibly to be aroused later by trauma or some other infection, notably symbiosis with the staphylococci. Death occurs from exhaustion or from generalization by the lymphangitis or thrombophlebitis.

Diagnosis of Glanders and Farcy.—Typhoid, pyæmia, rheumatism, erysipelas, variola, syphilis, lupus, tuberculosis, and anthrax may cause confusion, but the surroundings, the history and the bacteriology of the case determine the diagnosis. The cultural findings are proven by intraperitoneal inoculation of a guinea-pig, in which orchitis should develop rapidly. Injections of mallein differentiate the disease in 90 to 95 per cent. of the cases for they produce a rise of temperature of three degrees or more. The bacilli are agglutinated by the serum of normal horses in a dilution of 1 to 200, and of those subject to glanders in a dilution of 1 to 1,000.

Treatment.—The prophylaxis of glanders is like that of anthrax. The principal measures indicated are isolation of diseased, and cremation of dead, animals, and the use of strong antiseptics in wounds in persons exposed to infection. Skin ulcers should be curetted and bandaged antiseptically; if they are obstinate, the live cautery should be applied. Infection of the nose should be treated by carbolic douches, zinc chloride paste, iodol, or iodoform. Arsenic, mercurial inunctions, iodides, and quinine and alcohol for the maintenance of strength are recommended. Injections of mallein may prove of therapeutic as well as of diagnostic value (Babes, Pilavios). Large doses of potassium iodide have been recently recommended.

TETANUS.

Definition.—Tetanus, or lock-jaw, is an acute infection caused by the tetanus bacillus and is characterized clinically by greatly increased reflexes, by muscular rigidity, and by tonic spasms, such as trismus, and opisthotonos. Tetanus is literally tension or stretching. The disease is described in Hippocrates' aphorisms.

Etiology.—In 1884, Carle, Rosenbach, and Rattone produced the disease by inoculations of pus, and in the same year, Nicholaier discovered the *tetanus bacillus*, of which Kitasato (1889) first obtained pure cultures from wounds and from earth, and found it an obligate anaërobe. The Bacillus tetani measures 3 to 5 by $\frac{1}{3}$ to $\frac{1}{2}$ μ, and is enlarged at one end, in which there is a spore, thus having a pin-head or drum-stick shape. It becomes thread-like in cultures. It stains readily by Gram's method, and is delicately flagellated and therefore motile. It is anaërobic and cultures emit a smell like that of an onion. It is very difficult to isolate but grows well in sugar solutions. The bacillus is very enduring, and lives long, even if dried or frozen; the spores may live eight years. It always exists in the primary wound and is thought to exist only on rare occasions in the tissues of the brain and cord, but recent figures show that it is present in 44 per cent. of the severest infections, so that tetanus no longer seems to be a simple toxæmia. The bacillus has been found in the blood and sputum. Tetanus is usually classified as traumatic or idiopathic (rheumatic); the so-called idiopathic form is similar to idiopathic erysipelas, for the *atrium* is either unseen, is microscopic, or is apparently through the pharynx. The bacillus produces *toxins* which are 100 to 400 times as toxic as strychnine and which in turn produce the symptoms. Brieger and Cohn speak of tetanin, tetanotoxin, and spasmotoxin; Ehrlich and Babes of tetanus spasmin and lysin, and Faber of a toxin which resembles the diastases. The bacillus does not produce inflammation, though mixed infections are usual. Tetanus symptoms result from the tetanus toxin alone, but both infection and virulence are enhanced by association with various putrefactive microörganisms, and innocuous solutions of tetanus poison become lethal if combined with sterilized cultures of these putrefactive bacteria. Mice, guinea-pigs and rabbits, are inoculable. In *nature* the bacillus is found in the alimentary tract of herbivora, whence it also occurs in manure, in garden earth, in street or house dust, in hay, in putrefying fluids, and in splinters. Though it is spread over the world, the disease is ten to twenty times as frequent in the tropics as it is in temperate zones, because the bacillus thrives in heat. More cases exist in Prague than in Berlin. It may lurk in certain districts, as it did formerly in the Eastern end of Long Island, or as it does in the West Indies where negroes are more often and more seriously affected than whites.

Mode of Infection.—Incised wounds are obviously less dangerous than punctured, gunshot, or contused wounds or hæmatomata. Tetanus occurred less frequently in our Civil War than it did in the Spanish-American conflict, in which many cases developed among the Spaniards. It may result from such minor operations as circumcision, hypodermic injections, the administration of antitoxin, tooth-extraction and vaccination (95 cases), and also from major operations. Parturition, lack of attention to the navels of the new-born, the use of imperfectly sterilized cat-gut, and leech-bites or pin-pricks may also cause its development. Chauffard (1903) collected 18 cases in which injection occurred from injections of gelatin for various acute hemorrhages, and in 6 cases it has followed similar injections for aneurysm. The germ is very frequently found in gelatin, which should therefore be sterilized three times and in very small amounts, each time for thirty to forty-five minutes at 212°. H. G. Wells has placed emphasis on the danger resulting from the use of toy pistols and blank cartridges for Fourth-of-July celebrations. He noted that 39 per cent. of the tetanus mortality of 1903 occurred near that date; 415

deaths from tetanus are listed in the United States for that year as against 105 in 1904; 75 per cent. of cases occur between the tenth and fortieth year of life.

Incubation.—This lasts from six to twelve hours in severe experimental inoculations, from two to nine days in less intense inoculations, and in man, from one to sixty days. The incubation in 66 per cent. of the cases lasts from five to fourteen days; Rose observed that in 33 per cent. of his cases the symptoms appeared in one, and in 45 per cent. in two, weeks.

Symptoms.—These appear suddenly, without any or with only indeterminate prodromes, and are due to toxæmia. This is proven by the fact that autopsies show no constant gross or minute alterations except those common to all convulsive conditions—*e. g.*, points of congestion or punctate hemorrhage. Ehrlich's side-chain theory is a fairly satisfactory explanation, for the anterior cells of the cord and medulla combine with the toxins. Babes believed that some third substance formed and produced the symptoms. These are, at first, *rigidity of the jaw* ("lock-jaw," trismus) and *stiffness of the neck.* In most animals the muscles become rigid near the seat of inoculation first, and sometimes, in injuries received in war, the muscles of the leg may be first involved. The probable explanation of this is that the toxin travels along the nerve trunks (Marie, Morais, Ransom, Meyer). The horse, like man, develops trismus first. The poison excites the anterior cells of the cord and medulla to tonic contraction.

The *facies tetanica* results from a spasm of the zygomatic muscles, which produces the *risus sardonicus*, and imparts a smiling expression to the upper and a sad expression to the lower part of the face (König); there are folds in the forehead; the eyebrows are raised and approximated, the eyeballs directed forward, the masseters prominent, the teeth set, and the nares dilated. The spinal extensors are tonically contracted, arching the head backward and the trunk forward (*opisthotonos*); the abdominal muscles are hard and retracted, and are occasionally even ruptured by their violent contraction; in rare cases the contracture is lateral (pleurothotonos), or forward (emprosthotonos), or the body is straight and rigid (orthotonos). The limbs are extended, the legs more so than the arms, and the feet and hands least. The spasms are chiefly *tonic*, but clonic exacerbations for a few seconds may result from peripheral stimulation of the sensory nerves or those of special sense, by the least touch, jar, breath of air, bright light or sound. The *reflexes* are *enormously increased*, particularly those of the skin. The spasms occasion violent muscular pain, oppression over the lower chest and epigastrium, speechlessness, insomnia, and, from participation of the medulla, profuse sweats and increased and irregular heart action. Fever may be present (even 105° to 111°), or the temperature may be normal or subnormal. After death a temperature of 114° has been observed. The *sensorium* is usually clear. Evacuation of the bladder and rectum is difficult, and erection or ejaculation may occur. The urine is decreased and sometimes contains albumin, sugar, acetone, or indican; but increased excretion of urea and creatin, which might be anticipated because of the muscular action, is not noted. In some cases the lymph glands adjacent to the seat of infection are enlarged. In very rare cases the toxins cause death without muscular spasm; *i. e.*, they combine with tissues other than the nervous system.

Death.—Sixty-six per cent. of the cases die in a paroxysm. The fatal factor is a respiratory spasm, an œdema or spasm of the larynx or an increasing heart weakness; death from collapse or in coma is exceptional.

Diagnosis.—The diagnosis is based on the nature of the wound, the presence of the Bacillus tetani, trismus (which is necessary for a diagnosis), the facies, substernal oppression, and opisthotonos. Serum from cases of tetanus, obtained by blistering, is fatal to white mice in twelve to twenty-four hours. In *strychnine poisoning* the history of the case, tests for the drug in the stomach washings and urine, the more abrupt onset of the spasm and of the reflex excitability, the more diffuse and severe clonic intermittence of the convulsions, their greater involvement of the hands and feet, and the later appearance of trismus and its interparoxysmal relaxation, are entirely distinctive. *Hysteria*, hydrophobia, and tetany (*q. v.*) are wholly different from tetanus. In *meningitis* the sensorium is affected. Trismus may be due to such other causes as dental caries or parotitis. Inflammation of the temporomaxillary joint usually results in a tender area in front of the tragus, produces unilateral masticatory cramp and is easily distinguished by the clinical evolution alone. Escherich's *pseudotetanus* is characterized by tonic contractures of the jaw, the back, and the limbs, which occur oftener after an acute infection than as an independent disease. Convalescence is usual after a few weeks.

Prognosis.—The death-rate ranges from 45 to 90 per cent., averages between 80 and 90 per cent., and is modified by the following factors:

1. **Incubation.**—The later after inoculation tetanus develops, the better is the outlook. The mortality is 91 per cent. if it appears in the first week, 81 per cent. if in the second, and 52 per cent. if later (Rose).

2. **Site of Infection.**—Infection in the arms results in a 70 per cent. mortality; in the legs, 90 per cent. (Norris); in cephalic tetanus (the *tetanus facialis* or *kopftetanus* of Rose, first described by Charles Bell, in 1830), the mortality is almost 100 per cent. J. H. Lloyd collected 100 cases of this type (1905); he finds the prognosis better than in the ordinary tetanus; Lloyd found seven cases of facial diplegia. In this type there is facial paralysis and involvement of the pontine and medullary nuclei, which sometimes causes dysphagia (hydrophobic type), or paralysis of the eye. The author observed these symptoms in a boy who had been kicked over the ear by a horse. *Tetanus* or *trismus neonatorum*, especially described by Beumer (1887), is an infection of the navel which occurs in the second week of life. It prevents nursing

and is fatal on the third or fourth day in 83 per cent. of the cases. In the West Indies 50 per cent. of the children died of tetanus at one time, in the Western Hebrides, 67 per cent., and in Iceland, 100 per cent. *Tetanus puerperalis*, particularly described by Heyse (1893), is a uterine infection and usually follows difficult labor, abortions, or ministration by midwives, and is generally fatal. In three years there were 232 cases in Bombay (Gowers).

3. **Course.**—Hippocrates observed that patients who survive the fourth day may recover; 68 per cent. of cases die in the first four days. According to Willard, 88 per cent. of acute and 25 per cent. of chronic cases die.

4. **Trauma.**—The death-rate in traumatic cases is over 80 per cent.; in the so-called idiopathic cases it is 50 per cent. or lower.

5. **Sex.**—More women recover than men.

6. **Symptoms.**—Slight trismus and response to antispasmodics are favorable; high fever and involvement of the pons or medulla, respiratory spasm, dysphagia, and ocular paralysis, are unfavorable.

Treatment.—1. **Prophylaxis.**—This consists of general surgical antisepsis and special care of punctured wounds, hæmatomata and injuries caused by blank cartridges. Iodine and silver nitrate are very helpful, but wide incision (in order to open the punctured wound, for the bacillus is anaërobic), thorough lavage and packing are most essential. Antiseptic conduct of labor, and care of the navel in the new-born, need but little emphasis at the present day.

2. **Antitoxin Therapy.**—Behring and Kitasato (1880) discovered an antitoxin and were able to immunize animals by cultures which were heated from 122° to 140°, or treated with Lugol's solution, which changes the toxins to toxoids (Ehrlich). The antitoxin was found in animals which are naturally immune to tetanus, such as crocodiles and chickens; and it was found in the fluids (the blood, milk, and bile) but not in the tissues. Tetanus antitoxin is not bactericidal,—*i.e.*, it does not act on the bacilli. It neutralizes the tetanus toxin in the blood, but *not after the toxin has become fixed in nervous tissue*, from which the antitoxin cannot dislodge it. Behring and Ehrlich think that the antitoxin neutralizes the toxin chemically, while Babes and others consider that cell metabolism is the most important factor, and that possibly a third substance is secreted. Tizzoni and Cattani (1891) produced a serum somewhat different from that of Behring. From the above it is obvious (*a*) that serum prophylaxis may be successful in suspected cases of earth infection or in dirty abortions, although the term "successful" is presumptive, as it implies that tetanus would certainly have developed; it has been successful in cases of wounds received on the Fourth of July, and it also succeeded in the Prague obstetrical clinic, in which latter instance every other measure failed; (*b*) that if used early it may neutralize circulating toxins which have not yet become attached to nervous structures and (*c*) that when the toxins have combined with the motor cells, it must be ineffectual. Babes estimates the mortality at 70 per cent. even

when the serum is used, which confutes the alleged lowering of the death-rate (34 per cent., Engleman; 43 per cent., Köehler, 16 per cent., Behring). The first dose should be 20 to 30 c.c., and later 15 to 20 c.c. should be given two to four times daily. It should be injected near the site of inoculation, and especially into the nerve trunks near the injury, in order to reach more readily the toxins. Subdural inoculations saved a severe case of cephalic tetanus (Holub). The technique is the same as in lumbar puncture (see MENINGITIS).

3. Clonic Convulsions.—These may be minimized by absolute *quiet* and seclusion, by darkening the room, and by careful handling of the patient. *Morphine* may be given hypodermically to relieve pain, insomnia, and cutaneous reflex irritability; one-quarter grain may be given every four to six hours for a day or two, but the pupils of the eyes and the respiration rate should be carefully watched. Occasionally a corrective hypodermic of atropine should be given. *Chloral hydrate* depresses the motor elements of the cord; twenty grains may be given every two hours for three doses, but even this amount may cause dangerous cardiac symptoms in alcoholics or in cases of arteriosclerosis. Digitalis may be given with chloral hydrate to steady the heart, and morphine and potassium bromide to enhance its depressomotor action and to lessen the cutaneous irritability. The bromides should be given ʒssj every three hours, for six doses, after which the interval should be lengthened. Inhalations of chloroform and amyl nitrate may inhibit the onset of severe attacks. Hot baths relieve tension, but the lifting of the patient apparently more than counterbalances their benefits.

4. Nutrition.—This must be carefully maintained by the nasal catheter (see TYPHOID) or by the rectum. In this way, the difficulties offered by trismus or dysphagia are overcome.

5. Other Measures.—(*a*) J. B. Murphy performs lumbar puncture and injects a weak solution of eucaine and morphine, with apparent results; (*b*) Baccelli, Ascoli and Babes secure better results with hypodermics consisting of 10 drops of 3 per cent. carbolic solution, than they do with serotherapy; no ill effects were observed from long-continued daily use; (*c*) Wassermann's proposal to inject subdurally an emulsion of sheep brains is endorsed by Babes and others. (*d*) Meltzer has recently been successful with intraspinal injections of a dram of a 25 per cent. solution of magnesium sulphate.

HYDROPHOBIA (LYSSA; RABIES).

Definition.—An acute specific infection, of unknown causation, peculiar to carnivora, the bites of which convey infection to other animals and to man. The disease is characterized by tonic and clonic spasms of the œsophagus and of the respiratory muscles, which spread to other muscles, and by an inevitably fatal evolution. It was described in ancient Jewish, Egyptian, and Indian writings, and was discussed by Democritus in the fourth and fifth centuries, B. C. The first adequate account was one by Youatt (1828, London).

Etiology.—In Germany it is infrequent, because of the strict laws; from 1887 to 1894 only 16 cases occurred. In England, France, and America it is far more common, and in Russia many cases develop. Dogs are most susceptible, are most frequently affected, and are the chief cause of dissemination (90 per cent.); next come wolves, cats, and cattle; and finally skunks, horses, pigs, rabbits and even fowls may be inoculated. The character of the *virus* is unknown. It is found in the nervous tissues most abundantly, in the saliva, and also, though in small quantities, in the blood, tears, sputum, semen, urine, and milk, and in the adrenals, lymphatics and muscles. Its propagation has been held to occur along the nerve trunk, but it must also reach the blood current. It resists putrefaction for fourteen to twenty-four days but, on drying, becomes attenuated in ten to fifteen days. The disease prevails chiefly in warm weather, particularly in the "dog days," and most laws are based on this assumption; but it is also fairly common in cool weather. In Paris most cases occur in March and May. Sixty per cent. of cases occur in men and 40 per cent. are in subjects under fifteen years of age. Inoculation occurs through the skin, and nearly always results from bites of infected animals, which may transmit the disease even during the incubation period. Punctured or deep wounds, and those in the nerves or muscles are most dangerous; 33 per cent. of those bitten by mad dogs, 62 to 90 per cent. of those bitten by wolves, and 60 per cent. of those bitten by cats, die. In exceptional cases, rabies results from being licked by an infected dog, from kissing an infected person, or from dissecting an infected body.

The incubation period varies greatly, averaging from six to ten weeks. The extremes are from one and a half weeks to five months, or in exceptional cases, one or two years. It varies with (*a*) the age, being shorter in children than in adults; (*b*) the site of the wound. If this is on the face or head, the incubation is very short and the symptoms are very severe. The incubation is longer and the symptoms less intense when the point of infection is on the body, because the clothing tends to clean the virus off the teeth.

Symptoms.—Three stages are described, which are somewhat arbitrary and variable.

First or Prodomal Stage.—The scar may become tender, paræsthetic or painful; the adjacent glands sometimes swell, or, if the wound is on the face, there may be sneezing. Distinct psychical alteration is usual, such as depression, irritability, or anxiety. Headache, insomnia, hyperæsthesia to light, sound and touch, anorexia, and some elevation of the temperature and pulse rate are usual. Sometimes speech is slightly disturbed, the voice is hoarse, the pupils are somewhat dilated, and premonitions of the later dysphagia are experienced. This stage lasts from one to two days.

Second or Excitation Stage.—(*a*) The *excitement* is more *motor* than psychical. The cutaneous and deep reflexes are increased.

There is such hyperæsthesia that the least sound or light, the slightest breath of air, or food and water in the throat, induce violent reflex spasms, whence the patient's dread of these excitants (photophobia, aërophobia, hydrophobia). Tonic spasms, following attempts at swallowing affect chiefly the muscles of the mouth, the hyoid elevators, and the laryngeal and respiratory muscles. Even without involvement of the larynx, the respiration is labored, with irregular, jerky, and often deep or sighing inspirations, during which the shoulders are lifted and the epigastrium is protruded. The spasms may extend to the trunk and limbs. (*b*) The excitement is also *psychical*, and is marked at the time of the spasm by restlessness and often late in the course by delirium, or even by violent mania. Delirium tremens may be simulated. The mind is usually clear between the spasms. The patient rarely attempts to injure attendants, although such an impression may be made by the patient's restlessness, the peculiar growling sounds occasioned by pharyngeal and laryngeal spasm, and the "frothing at the mouth," which is due to inability to swallow the increased flow of saliva. Other less constant symptoms are elevation of temperature from 100° to 103° or 105°, though some cases are afebrile; salivation and lachrymation; thirst; tremors, erections and ejaculations; increased pulse rate; scanty, albuminous urine (25 per cent.), often with casts and sometimes with sugar or acetone. The excitation stage lasts one and a half to three days.

Third, Paralytic or Terminal Stage.—This stage is less common in human than in animal rabies. In animals it may be marked from the beginning and the other stages may be lacking or rudimentary—the "dumb" rabies as contrasted with the furibund type. The spasms and excitement cease, the pulse becomes fast, small, and irregular, the pupils are wide and irregular, sweating is profuse, swallowing becomes impossible, and paralyses develop, mono-, para-, or hemiplegic, or of the acute ascending type of Landry. The sphincters are sometimes paralyzed. The patient dies, unconscious, of cardiac collapse, rarely of asphyxia or convulsions. The third stage seldom lasts more than eighteen hours.

Autopsy Findings.—Pathologists are not absolutely unanimous in regard to the specific autopsy findings. Minute hemorrhages and special congestion of the medulla and cord mark hydrophobia as they do other convulsive states. Babes's *rabic tubercles* are aggregations of leukocytes around the vessels and nerve cells. The latter may degenerate. Rapid diagnosis of rabies may be made, according to von Gebuchten and Nelis, by examination of the sympathetic and cerebro-spinal ganglia, under the capsules of which are found *accumulations of epithelioid and lymphoid cells*. Babes considers them less important than the rabic tubercles. The bodies described by Negri and Pavia (1903) in the cell processes, may be protozoal parasites or artefacts.

Course and Prognosis.—In twelve hours to ten days—on the average two to four days—all untreated cases die, 10 per cent. in the first day, and 60 per cent. in the first three days. Laveran, Chantemesse, Novi, Poppi, and others report recoveries, but Hogyes' is the only recovery verified by inoculations of the saliva.

Diagnosis.—The diagnosis is usually made with ease. After bites from supposedly rabid animals, microscopic examination can be made and also subdural inoculations in rabbits, which die in two to three weeks with the paralytic symptoms. *Tetanus* of the cephalic and hydrophobic type is easily distinguished by the invariable trismus. *Hysteria* (pseudohydrophobia, lyssophobia) in one bitten by a dog may at first cause suspicion but rarely any protracted doubt. The initial spasmodic dysphagia and irritability are not followed by the other symptoms and stages of hydrophobia, and the clinical evolution alone is distinctive.

Treatment.—1. Prophylactic.—In Germany, where laws concerning dogs are not only passed but are enforced, hydrophobia is almost extinct, while in Roumania over 100 cases occur yearly. In human rabies it must be remembered that the saliva is infective.

Preventive Inoculation.—Pasteur, in 1881, found the rabic virus in the nervous tissues. "The virus of the street," as Pasteur called it, varies in virulence, and subdural inoculations from the brains of rabid dogs, into rabbits, killed them in periods varying from fourteen to twenty days. By inoculating a series of rabbits, the incubation period is progressively shortened to a fixed period of seven days, this virus being called the "fixed virus." Pasteur attenuated this virus by suspending the spinal cord of the rabbit in a clean jar and desiccating it with caustic potash. Animals were inoculated with virus which had been desiccated for two weeks, then with virus a little stronger,—*i. e.*, with virus desiccated for ten or twelve days, until finally immunization was secured against inoculations which would previously have been fatal. In 1885, human beings bitten by rabid beasts were thus treated by two inoculations daily, for fifteen days, and immunization was secured, for the slow rabic incubation gives ample time for treatment. Early treatment secures the best results. Statistics show that the former mortality of 80 per cent. in untreated bites in the head or face has been reduced to 1.25 per cent.; the mortality of 60 per cent. in those bitten in the hands is reduced to 0.75 per cent.; the mortality of 30 per cent. in those bitten in the covered parts of the arm and trunk, and of 15 per cent. in those bitten in the leg, is reduced to 0.25 per cent. *The total mortality has been reduced to 0.5 per cent.* Babes (1905) very rarely found, as sequences of Pasteur's treatment, the following: neuroses, neuritis, bulbar or spinal paralysis and Landry's paralysis.

2. Primary Wound Treatment.—The wound may be sucked by the victim, but not by any other person. It should be washed with strong antiseptics, burned with caustic potash, pure carbolic acid, or nitrate of silver, and as soon as possible should be exposed surgically, treated with the live cautery, and packed with gauze, so that healing may be delayed for over a month.

3. Late Treatment.—In developed cases the mortality is 100 per cent., whence curative therapy is out of the question. The treatment of the *convulsions* is similar to that of tetanus, and nutrition is maintained by nutritive rectal enemata. Morphine and inhalation of chloroform are better than chloral and bromides, and should be given until they show their full physiological effects. Cocainization of the mouth and pharynx may overcome the pharyngeal spasm incident to swallowing milk and water, and to medication. Tracheotomy is useless for the respiratory spasm, which reaches further than the larynx, and involves the muscles of the chest and diaphragm; and besides, it is maintained by many that the larynx itself is not closed.

ACTINOMYCOSIS.

Definition.—An infection caused by the actinomyces (literally "ray-fungus"); it is mostly sporadic in cattle, and less frequently so in swine, horses, deer, or elephants; it occurs occasionally in man; it is rarely endemic or epidemic; it is marked by chronic inflammation (*a*) in the mouth, head or neck, (*b*) in the lungs, or (*c*) in the intestines or abdomen; and it usually runs a chronic course.

History.—Von Langenbeck (1845), Davaine (1850), Laboulbêne and Robin (1853), and Lebert (1857), observed the peculiar sulphur granules in the discharges of pus. Rivolta (1868) and Perroncito (1875), described the fungus, Bollinger (1877) first scientifically established its constant occurrence in cattle, and Hartz named it *Actinomycosis bovis*. J. Israel (1878) first found the parasite in human cases, but to Ponfick (1879) is due the credit of identifying the bovine and the human types. William T. Belfield, of Chicago, first found actinomycosis in this country. The monographs by Ponfick, Israel, Bostroem and Illich are the most important.

Etiology.—Its *distribution* varies. Statistics in Berlin, Breslau and Vienna show large numbers of cases. There are more cases in Scotland, Denmark, Italy, and Russia, than in America, where it is less frequent in the United States than in Canada. Seventy per cent. of the cases occur in males, and most of them are in subjects between twenty and forty years of age (33 per cent. between twenty and thirty; 82 per cent. between ten and fifty). The fungus enters the system chiefly by the mouth, in which ragged teeth, abrasions, etc., favor its access. Though almost nothing is known of it outside of the body, it occurs in straw, hay, barley, oats, and similar cereals, remnants of which are often found in actinomycotic foci. Most cases occur in young cattle at dentition; 89 per cent. in the last half of the year, when the hair and the grain which are ingested are dry. The drinking water may be the source of infection.

Bacteriology.—The *parasite* of actinomycosis is classed as a streptothrix and is found in small granules which measure 0.15 to 0.75 millimeters, are usually ovoid in shape, and show a "radiate" form when squeezed under the cover glass. The younger forms are transparent, sago-like, white-gray bodies which may be easily overlooked, and the older forms are more yellow. These suphur-like granules, on minute examination, consist of threads and coccoid bodies which some consider spores. The older yellow forms become clubbed, which is considered a degenerative change. The threads divide and branch. The fungus is therefore phleomorphic. It stains fairly well with the usual stains and by Gram's method. Cultures are successfully grown in 50 per cent. of the cases, and are best obtained by mixing the granules thoroughly with softened gelatin and bouillon, and then by spreading or inoculating them as usual. The colonies are thick, folded, membranous, resistant, and at first have a mucoid or crystalline appearance. Experimental inoculations succeed, but infection is very rarely direct either from animals to man or from one person to another.

Forms of Actinomycosis.—Incubation averages four weeks. Clinically there are three main forms: (1) That of the head and neck; (2) that of the lower digestive tract; and (3) that of the respiratory tract.

1. The *forms localized in the head and neck* constitute 50 per cent. of actinomycosis. The atrium is in the soft tissues of the mouth and throat. Entering these, the fungus is surrounded by a massive accumulation of round cells which soon degenerate centrally. The reaction of the connective tissue is marked and the proliferation shows numerous round or polygonal cells with large nuclei,—the epithelioid cells. Giant cells also form. The process is designated by Bostroem as a chronic inflammation, and by Israel as an infective granuloma, resembling the tuberculous, syphilitic, or leprous process. The further destiny of these granulations of the tissue is modified by the resistance of the tissues and by the animal diseased. (*a*) *In man* the granulation wall usually melts so that the fungus is surrounded by a mucoid, creamy, cloudy fluid, in which there are oil, fibrin, blood, pigment, and actinomycotic cocci and threads; the abscess is formed by the fungus itself which, unaided by pyogenic organisms, burrows and reaches the surface by circuitous fistulæ; it discharges a thin, puriform secretion like that of scrofulous glands but which contains the sulphur-like grains. (*b*) *In cattle and horses* the granulation tissue is more effectually obliterative and limitative, and hard, board-like, or ligneous tumors develop ("lumpy jaw," "wooden tongue"); suppuration and miliary dissemination are less frequent, and calcification is more frequent than in man. In human subjects the infiltration may involve the neck, face, tongue, pharynx, tonsils, larynx, and thyroid gland. In general, the most frequent involvements are inflammation of the soft tissues about the jaws, especially those under the lower jaw, submaxillary or submental infiltration, and superficial invasion of the bones. In the tongue, actinomycosis may resemble gummata or cancer but in cancer there is more pain and glandular invasion. In some few cases the maxillæ may be primarily invaded (Israel, Murphy). *Sarcoma* and *caries* may be simulated. The process sometimes *spreads regionally* along the bones to the base of the

brain and results in meningitis; along the spine, and causes vertebral erosion; and behind the sternum with mediastinal infiltration.

Symptoms.—These vary with the part invaded. The fever is irregular, rarely continuous or typhoidal. Pain and tenderness are not marked, for the process is indolent or chronic. Dysphagia accompanies retropharyngeal involvement, œdema occurs in laryngeal localization, and other local interference such as venous obstruction is frequent. In a recent case, the writer observed trismus and severe unilateral trifacial neuralgia from a primary focus at the base of the skull; recovery followed operation. Besides the regional invasion by contiguity, above named, *hæmatogenous extension* by metastasis may occur in this type as well as in the others to be described later. Unlike tuberculosis, extension to the lymphatics, or general dissemination by them, is unusual. Hæmatogenous (really pyæmic) deposits may occur in the brain, meninges, liver, spleen, kidneys, intestines, lungs, muscles, skin, bones, joints, or retinæ, in which they may simulate miliary tubercles. In the head-and-neck form the mortality is 10 per cent.

2. The *lower digestive tract* ranks next (20 to 30 per cent.) in frequency of infection. This may occur anywhere between the œsophagus and rectum, but 50 per cent. of this group occurs in the region of the ileum, cæcum and appendix (*perityphlitis actinomycotica*). Local pain, tenderness, induration and adhesions frequently follow the primary lesion of the intestine. In rare instances this lesion is superficial (*enteritis actinomycotica superficialis*, Chiari), but small nodes usually develop in the submucosa, over which the mucosa becomes somewhat pigmented. This is followed by central softening of the nodes and by the development of small ulcers, the undermined edges and uneven bases of which make them resemble tuberculous ulcers. Secondary pyogenic infections are frequent. The ulcers may cicatrize or fuse into larger ulcerations.

Symptoms.—As above indicated, the signs are often those of appendicitis; symptoms may be absent, but colic and vomiting are often present, and constipation is more frequent than diarrhœa or dysenteric stools. *Extension* of the process is usually *downward;* it involves the iliac bone, and occasionally the thigh, hip, bladder, ovaries, vagina, or rectum; the ray fungus is voided in the urine and fæces; and anteriorly preperitoneal, suprapubic, or muscular infiltrations occur, with external fistulæ. The process then extends *backward* into the retroperitoneal cellular tissue, in which it may burrow around the kidneys or even into the chest. These varied localizations may simulate perinephritis, peripsoitis or parametritis. In rare cases, freely movable, and therefore operable, tumors may develop. Scoliosis may result. Vascular metastases may occur as they do in involvement of the head and neck but suppuration of the liver is the most frequent development, and is secondary clinically and pathologically, though it is sometimes apparently primary. Forty cases are reported. In 71 per cent. death results, usually from exhaustion, from mixed infection, or from diffuse amyloidosis. Diagnosis must be made from gross detection of the yellow granules or from microscopic recognition of the fungus.

3. *Respiratory involvement* is still less frequent. It is (*a*) secondary to extension by contiguity from the neck or abdomen, or to miliary or larger infarcts, or (*b*) primary in the lungs, which is a less frequent form (14 per cent.). Primary pulmonary actinomycosis is rarely of the superficial type (*bronchitis actinomycotica superficialis*). Canali described such a case with seven years duration. This form may easily escape recognition. A case of actinomycotic fibrinous bronchitis is reported. More often it is deeper, and invades the alveoli. The lower lobes are its usual seat and they show a gray hepatization with the ordinary actinomycotic changes. Fusion of the foci may involve large portions of tissue, but demarcation is usual in the lung. The clinical picture varies. (i) In some instances *cavity formation* is obviously present. There is fever of a hectic or possibly of a typhoid type, an harrassing cough, and malnutrition; the sputum is puriform, sometimes fœtid and stained with altered blood like raspberry jelly, or contains even considerable quantities of blood and often reveals the actinomycosis rays. (ii) *Induration* of the lower lobes may occur from cicatricial development. (iii) *Pleurisy;* (iv) *peripleuritis;* (v) pericarditis; or (vi) mediastinitis may be the dominant finding. There may be much luxation of the intrathoracic contents, and involvement of the heart is characterized by great dyspnœa, dilatation and hydrops. Penetration may occur through the chest wall with characteristic discharge from the sinuses which run along the spine and ileopsoas muscle, or into the abdomen. Hæmatogenous generalization occurs as in other forms. The examination of the sputum is essential for diagnosis unless fistulæ develop. In 25 per cent. of cases the ray fungus is seen and *tuberculosis* is excluded by absence of tubercle bacilli and elastic fibers. Otherwise the resemblance may be very close or differentiation impossible. The *course* is usually subchronic (averaging under a year), less often chronic (two to three years), and very rarely acute. The outcome is fatal in practically 100 per cent. of the cases, though Schlange reports two recoveries.

4. *Other primary localizations* are rare; 23 primary skin cases are reported. They may resemble lupus or tubercle. Primary brain lesions are infrequent (Bollinger, Orlow, Keller, Grangee and Delvaine); they may simulate brain tumor or cause cortical epilepsy. Primary genito-urinary infection is possible, though very few cases are recorded (Stanton). The lachrymal duct or middle ear is very rarely the primary focus.

Diagnosis.—Similar fungi may cause difficulty in diagnosis, but their classification is still a mooted question. Eppinger's cladothrix asteroides may cause deposits in the cervical glands or brain, and miliary nodes in the lung. Rosenbach noted a similar growth in erysipeloid, and Vincent in Madura foot, which liquefies gelatin and produces a red pigment. The leptothrix of the mouth is a possible cause of confusion.

Treatment.—1. **Prophylaxis.**—Besides general measures employed in any contagion, care is necessary in regard to the holding of straws in the mouth or picking the teeth with them. Thorough cooking of cereals before eating is essential.

2. **Surgical Extirpation.**—This is indicated, if possible; or a sharp spoon should be used to clean accessible foci and fistulæ, which should then be packed with iodoform or sublimate gauze. Injections of bichloride of mercury ($\frac{1}{10}$ per cent. to 1 per cent.) are used especially by Illich and Hochenegg.

3. **Potassium Iodide.**—In cases wholly inaccessible to the knife, potassium iodide is excellent or even specific, according to some writers. It is not parasiticidal, but softens the tissues. It also prepares operable cases for surgical measures; 53 per cent. of bovine cases recover under its use. Thomassen and Netter use six grams daily for a month and then decrease the dosage. Rydygier injects large quantities of a 1 per cent. solution into the affected tissues.

4. **Tuberculin Injections.**—These were advantageous when used by Billroth, Kahler, and Socin. Bevan has seen benefit result from the internal use of copper sulphate.

GONORRHŒAL INFECTION.

Lightly spoken of as "the badge of virility," gonorrhœa is often untreated and neglected. It frequently becomes chronic, and in its so-called latent form may be conveyed after marriage to the wife, on whom it usually entails suffering, frequently sterility, and sometimes permanent invalidism. Its other dangers are:

I. *The regional invasion* of the genital tracts (*a*) in the male, of the prostate, seminal vesicles, and testes; (*b*) in the female, of the vagina, cervix, uterus, tubes, ovaries, and peritoneum; in institutions, repeated epidemics of vaginitis in children have occurred; (*c*) in both sexes, of the bladder, pelvis, and kidneys, in which extension, mixed infection is an important factor.

II. Gonococcic Septicopyæmia.

Hæmatogenous generalization has already been discussed under septic affections. The fever is toxæmic and septicæmic. Injection of sterilized cultures produces violent reaction, which hitherto has baffled attempts at animal immunization. The fever is intermittent, and is often marked by great variations. The gonococcus has been found in the blood (Amann, Petrone, Hewes, Thayer, Benmer and Lazear, in all 15 times); in the myocardium (Councilman); in the pleura (15 cases collected by Lemoine and Gallis, 1905); and in the joints (*v. i.*), kidneys, spleen, muscular abscess, lymph-glands (von Hausteen, Columbine) and skin (Hochmann, Horwitz, and Rendue). It has also been found in meningo-myelitic foci; in muscular abscesses; in myositis (7 cases collected by Harris and Haskell, 1904); in arteritis and phlebitis (25 cases collected by Heller, 1904; the author saw one in 1905 with Dr. Cuthbertson); in purpuric, erythematous, dermatitic and exanthematous eruptions; in felons; and in periostitis. It is reported to have been found in cases of pneumonia and of multiple neuritis. Lœb's collection of 62 cases of ulcerative gonorrhœal endocarditis has been referred to. This endocarditis may possibly heal. Septicopyæmic lesions may be due to Neisser's diplococcus alone, or to mixed infections.

Most cases of *gonorrhœal rheumatism* must be regarded as a mild septicæmic form. Occasionally it is part of a severe septicopyæmia. It was described by Selle and Swediaur in 1781, and the gonococcus was first found by Petrone (1883), and Kammerer (1884). According to Damasch it develops in 2 to 3 per cent. of gonorrhœal infections. If 80 per cent. of males contract gonorrhœa, König's assertion that it is the most common disease of the joints seems well established. Eighty per cent. show an acute urethritis; in the balance the urethritis has subsided or become chronic. In 18 cases reported by Lucas, the infection was primarily gonorrhœal conjunctivitis, especially in infantile cases. Northrup's series of 252 cases of gonorrhœal rheumatism gives 91 per cent. in males, though Brun and Bennecke find 62 per cent. in women, which is contrary to common experience. About 50 per cent. of the cases occur between twenty and fifty years of age. It has followed experimental gonorrhœa. Too active treatment and sexual indulgence during the florid stage of gonorrhœa are held to be predisposing causes.

Symptoms.—The symptoms are extremely variable. The joints alone may be involved, or there may be more severe evidences of blood invasion, such as endocarditis. While the synovial membrane is most often inflamed, gonorrhœal rheumatism is more than synovitis. The accessory bursæ, contiguous tendon sheaths, and the extracapsular structures are often involved, whence the name of *gonorrhœal arthritis*. It is often held that it affects one joint (monarticular) or at least few joints (oligarticular). Though fewer joints are usually involved than in genuine rheumatism, in Northrup's series more than one joint was affected in 78 per cent. of the cases, and three or more joints in 69 per cent.; and in Gerhardt's series two to nine or more joints were involved in 87 per cent., and one joint only in 13 per cent. Gonorrhœal arthritis frequently involves joints which are usually unaffected in acute articular rheumatism, *e.g.*, the sacro-iliac synchondrosis, sternoclavicular, temporo-maxillary, and intervertebral joints. The knee is affected in 70 per cent., the ankle in 27 per cent., fingers and toes in 19 per cent., hip in 13 per cent., wrist in 12 per cent., shoulder in 10 per cent., and the elbow in 9 per cent., of the cases. Within the joint serofibrin exudes, and without the joint there is œdema and infiltration. Suppuration is not frequent but may occur with or without coincident pyogenic infection, especially in the joints and tendons of the hand. The gonococci are found with variable frequency, the highest percentage of cases being that of Rindfleisch (60 per cent.).

König's *pathological classification* includes (1) *simple hydrops* of th joint; (2) *catarrhal* or serofibrinous *hydrops;* (3) *pyarthrosis,* and (4 *gonorrhœal phlegmon,* affecting the soft parts. A *clinical classificatio* may be made as follows: (1) *Arthritic form,* pain with exudation; (2 acute or subacute *polyarthritis,* by itself, or with septicæmia; (3 polytendosynovitis and polybursitis, often with periostitis,—this is th *periarthritic form;* (4) *acute monarticular form,* with rather intense loca reaction, sometimes with suppuration; and (5) the *chronic monarticular hydrarthrosis,* especially of the knee, a far less frequent form We might add (6) the forms in which simple arthralgia is present o the process localized in the heel.

The great clinical variations are explained by Pribrâm, as follows The joint involvement results from (1) gonorrhœa alone; (2) gonorrhœal infection plus mixed infection, with pus or tuberculous microorganisms; (3) genuine rheumatism, associated with an accidenta and essentially insignificant gonorrhœa; (4) gonorrhœa plus a relaps of rheumatism.

Course.—The course is essentially chronic, is sometimes relapsing and is marked by complications; iritis is fairly frequent; atrophy i the contiguous muscles may occur as in rheumatism; ankylosis ma result, which is far oftener plastic than osseous. In exceptional instances, sciatica, permanent arthritic changes resembling arthritis deformans (*q. v.*), exhaustion psychoses, or multiple neuritis, develop.

Treatment.—Recovery is a matter of time and nature, rather tha of therapy. The salicylates are almost wholly inert and the iodide highly unsatisfactory. In the acute stage, immobilization by plaste casts or splints, and in the chronic stage, blisters, Pacquelin cauter massage, passive movements, and extension, to overcome residu exudation and contractures, are useful. Some cases are helped b Roentgen rays and by superheated air (See ARTHRITIS DEFORMANS) Surgical incision and simple irrigation are indicated after a reasonable lapse of time and after the failure of expectant treatment. I some intractable cases, however, there is no fluid to drain.

SYPHILIS (POX, LUES VENEREA).

Syphilis is evidently an ancient disease as is indicated by probabl syphilitic bones belonging to the stone age and found in Japan. Th first clear outbreak occurred in 1494 among the troops of Charle VIII, who was then investing Naples. A full account of this diseas will be given because of its colossal importance.

Definition.—A specific chronic infection, found in man only, eithe hereditary or acquired by inoculation, in the seat of which a primar lesion (hard chancre) develops. This is followed in two or thre months by secondary lesions in the skin and mucosæ. Still later (afte months or years) tertiary lesions (gummata) develop in the viscera, bones, and skin, and these are finally followed in some instances by meta- or parasyphilitic manifestations, as tabes dorsalis, dementia, and perhaps aneurysm. This division of Ricord is most convenient but does not invariably hold,—*e. g.*, in hereditary syphilis and sometimes in the (malignant) acquired variety, where the stages overlap. Virchow finds a close histological resemblance between the secondary and tertiary lesions.

Stages According to Ricord.—**The Primary Stage.**—(1) Incubation lasts until the chancre appears, when the disease is already, according to most writers, a general disease; (2) the chancre develops, the lymph-vessels and adjacent glands enlarge, and there is a period of latency, sometimes called the "second incubation."

The Secondary Stage.—(1) Symptoms prodromal to the eruption, as fever or anæmia, develop and then (2) the eruption appears on the skin and mucosæ, with headache, "rheumatism," iritis, or sometimes jaundice, albuminuria, and splenic tumor.

The Tertiary Stage—This may follow very closely upon the secondaries or appear with them (syphilis maligna), but usually is not manifest for at least two years (even fifty or sixty). Tumor-like gummata occur in the skin and viscera, especially in the liver, scrotum, brain and cord. Unlike secondary manifestations, they are asymmetrical, non-contagious, and respond to different therapeutic agents.

The parasyphilitic manifestations of Fournier, Sigmund, Lancereaux and Baümler are not included in Ricord's division.

Acquired Syphilis.

Etiology.—**1. Transmission by sexual intercourse** (contact syphilis) causes the great majority of cases, but the term *lues venerea* is often incorrect and unjust since syphilis may occur in the innocent (syphilis insontium). Lustgarten's bacillus 3 to 4 μ in length, found by him in round cells of primary and tertiary lesions, is not generally recognized and may be confused with the smegma organism. Schaudinn and Hoffmann describe a *spirochæte pallida*, so named because of the difficulty in staining it: it is thread-like, refractile, actively mobile, pointed at its ends, and twisted like a corkscrew; it measures 4 to 20μ in length; it rather resembles the spirochæte of relapsing fever but is a flagellated protozoön. Though it is clearly present in nearly all cases, it is not positively known whether it is the cause of syphilis or only a concomitant infection. It is found in the chancre, in the papules, blood, viscera and in inoculated apes and mice. Siegel's staining technique; an old, "well-ripened" solution of 1 part methylene blue with 2.5 parts borax and 100 parts distilled water gives clear, well-defined views of the parasites after ten minutes contact with the sections, followed by simple rinsing under a strong stream of water. This stains the red corpuscles well, but leaves the blood plasma colorless, in which the parasites stand out distinctly. It is not necessary to examine sections of the organs, as the parasites are found equally well in the blood, which is much more convenient for examination. Siegel found the parasites constantly in the blood at the time of the exanthem. The parasites vanished from the blood in the third week of inunction treatment.

The sound skin and mucosæ resist infection, and hence a raw surface, erosion, wound, or hyperæmia is necessary for infection. Syphilis not only has a certain choice of place in its inoculation, but persists with peculiar obstinacy in certain localities, notably in the genitalia, under the breasts, in the axilla, in the navel, between the toes, in the angles of the mouth, on the tongue, iris, palate or tonsils, because of (*a*) irritation caused by the sweat, sebum, saliva or urine; (*b*) pressure on the parts by clothing; (*c*) trauma to the skin, or (*d*) irritation from smoking, from other skin lesions, and from various causes. These foci are of great moment in the awakening or reproduction of the disease. Syphilis is sometimes called a disease of cities; though more common in the city than in the country, syphilis very frequently escapes recognition in the country.

2. **Accidental infection** occurs in surgical or obstetrical practice, in skin-grafting, by postmortem infection (two cases of personal observation), in ritual circumcision, in nursing, vaccination, kissing, from use of pipes, in shaving, by scratches, from instruments used in glass-blowing, or from catheters. A few instances are known of transmission through a third healthy person.

Symptoms.—1. **Primary Stage.**—After an incubation of two to three weeks (ten to seventy days), the *initial lesion* occurs (the *ulcus durum*, Hunterian chancre, or initial sclerosis). Multiple lesions are rare. The induration may be irregular or flat, but is usually oval. It is always sharply localized, is of variable or even cartilaginous hardness, is freely movable, and is usually red and papular. *Hardness* is its most constant characteristic; it may last a long time, and heals only when the hardness disappears. It cannot be diagnosticated before the sixteenth, and is most characteristic on the twenty-first day or in the fourth week, when the glands become somewhat enlarged. Such retrogressive changes as *ulceration*, loss of epithelium, or necrosis are frequent. The ulcer varies in size, is round, elliptical, slit-like, or irregular; it is hard, with even surface, seldom with elevations or excavations; it has no areola, but a smooth, glistening base and there is some thin secretion. The color varies. It is gray when the exudate is removed, red, blue, or even gangrenous. On the skin the chancre may appear as an erosion. Contact ulcers around the corona are hard. The sclerosis may resemble herpes, but there is no itching. It has a copper hue. An herpetiform sclerosis becomes indurated in about sixteen days, while simple herpes lasts but three or four days. Herpes may be the atrium for syphilitic inoculation. If the sclerosis occur on a mucous surface there is little elevation; maceration and bleeding readily occur. (*a*) The *genital sclerosis in men* occurs on the urethral orifice; in the fossa navicularis, possibly producing some stenosis or ulceration, with a thin, "meat juice" secretion; on the glans, often as an erosion, usually not extensive, and difficult to diagnosticate because of the hardness of the glans; on the corona, circular and hard; on the (broken) frenulum or on the sulcus coronarius, usually with ulceration and much induration, giving the "split pea" appearance, one-half lying on the glans and the other half on the prepuce; on the prepuce, often with phimosis; on the dorsum. (*b*) The *genital sclerosis in women* occurs mostly on the labia; in the labia majora, as large, readily ulcerating contact ulcers with inflammatory œdema; in the labia minora as small, hard and circumscribed nodes; on the prepuce as hard fissures; in the urethral orifice with widening and induration which is felt from the vagina; on the posterior commissure, as erosions, which are especially prone to infect during coitus; on the hymen relics, as fissures, or large hard ulcers reaching into the vagina; rarely in the vagina, because of its paucity in glands and the thick epithelium, although sclerosis may develop on gonorrhœal erosions; in the vaginal portion of the cervix, usually in women who have been pregnant, in which location it produces chancres on the male meatus and is difficult to differentiate from simple and gonorrhœal erosions. (*c*) The *perigenital sclerosis* occurs on the scrotum, on the mons veneris, or on the inner thigh. (*d*) *Extragenital scleroses* (3 to 4 per cent.; 5.5 per cent., Montgomery and Bulkley; 6.3 per cent., Fournier) are especially dangerous in midwives, physicians, and wet-nurses. They occur on the scalp, conjunctiva, nose, eyelids, lips, tongue and tonsils (from sexual perversion), cheeks, fingers, arms, and mammæ. In rare cases of accidental syphilis the chancre may not be found. Among 9,058 extragenital chancres, 1,863 occurred from vaccination, 745 from cupping or phlebotomy, 179 from circumcision, and 82 from tattooing (Bulkley).

The primary sclerosis is always inoculated from a chancre or from an individual with secondary syphilis, and, in very rare cases only, from tertiary syphilis; Delbanchs reports five infections from gummata chiefly of the genitalia. When the sclerosis is recognized, the blood is already infected.

HISTOLOGY OF THE SCLEROSIS.—The round cells about the vessel walls increase their thickness ten- or twentyfold, and the vessel lumen is narrowed from external pressure and endothelial proliferation, even to obliteration, when retrogressive change, as ulceration, ensues. The lymph vessels and glands are involved later. The nerve fibers are altered. Epithelioid and giant cells, and proliferation of the connective tissue in and about the vessels, are noted. The spirochæte pallida is almost invariably present.

COURSE.—Retrogression usually occurs and the induration disappears in thirty to ninety days, generally without scar formation. In exceptional cases (1) the sclerosis lasts longer, especially on the corona or prepuce, and relics may be found after years (even 50) due to permanent vascular injury; (2) acute or (3) phagedenic inflammation sometimes intervenes. Recently anthropoid apes have been inoculated by Metschnikoff and Nicolle; they developed typical chancres but usually died in the secondary stage of the disease. Siegel states that he has succeeded in inoculating rabbits and white mice.

Diagnosis (According to Neumann).

Chancre or Sclerosis—*vs.*—Venereal Ulcer or Chancroid.

Incubation: two to three weeks.	Three days.
Form: erosion, ulcer, nodule, papule, all indurated.	Pustule, ulcer, etc., without induration.
Number: single, rarely multiple at first, also rarely successively inoculated.	Often multiple at beginning or successively inoculated.
Depth: erosion is superficial, flat, with little elevation, rarely deep excavations.	Through entire cutis or mucosa; "punched-out" appearance.
Border: gradual descent.	Abrupt, sharp, indented, undermined.
Surface: red, livid, sometimes pseudomembranous, thick, yellow-white covering or crusts.	White, gray.
Induration: marked, cartilaginous, sharply outlined, movable, sometimes thin, lasting weeks, perhaps months.	No basal induration; not sharply outlined; merges gradually into adjacent tissue; short duration.
Secretion: scanty, serous, autoinoculation difficult.	Rich, purulent, auto-inoculation easy.
Pain: little.	Much.
Destruction: rarely phagedenic (and then circumscribed).	More often phagedentic; diffuse.
Re-infection: extremely rare.	Frequent.
Lymph-vessels: inflamed frequently, hard and wide.	Painful; cylindrical.
Lymph-glands: regional, indolent, swelling hard, painless, movable; rarely pus formation.	Often absent or acute inflammatory swelling; often pus formation, capable of autoinoculation.
Nature of the disease: constitutional symptoms follow, usually inside of eight weeks.	A *local infection*, or involving at most contiguous lymph-glands.

Mixed infection may occur, whence the now abandoned idea of the unity of all venereal ulcers. The real practical lesson from this doctrine is diagnostic reservation, which is often imperative for some time in doubtful cases.

Lymphangitis syphilitica is marked in 70 per cent. of cases. The lymph vessels may become as thick as a lead pencil, bulging at the site of the valves ("bubonulus"); they become hard, are mostly painless, reddened, and are narrowed in their lumen from endothelial pro-literation, and from pressure inward of their infiltrated walls. The inflammation is best seen on the dorsum penis, extending toward the symphysis pubis; it is usually not over 6 to 8 centimeters in length, but may reach the glands. Resorption occurs in three to eight months. Œdema in women is particularly suggestive when the lymphatic system is involved and the initial lesion cannot be found. *Local lymphadenitis* (indolent buboes) is present in 97.3 per cent. of cases, and appears in three weeks if there be much, or in four weeks if little secretion from the chancre; and in six weeks reaches the contralateral inguinal glands and the iliac and lumbar group, and in seven weeks, the cubital glands. Just before the eruption of the secondary stage, the cervical and other groups are involved.

II. **Secondary Stage.**—Secondary symptoms usually appear toward the end of the eighth week (sixth to twelfth week). 1. Evidences of *general infection* occur. *Fever* is spoken of by the oldest writers, but is less frequent at the present time (20 to 33 per cent.). It may be low. It is far less common in tertiary than in secondary syphilis ("fever of syphilitic invasion"). It often develops just before the eruption, and occurs mostly in the pustular variety. It may be continuous, remittent, or intermittent. Solution by crisis has been observed by Gunz. Fournier described a typhoidal type. Janeway reports instances resembling tuberculosis pulmonalis. It may occur with chills, suggesting malaria. Sudden *anæmia*, vasomotor symptoms, psychical depression, (frontal) *headache*, vertigo, neuralgias, palpitation, night-sweats, boulimia, polydipsia, digestive disorders, *nocturnal boneaches*, synovitis, increased reflexes, icterus, swollen liver, albuminuria, or swollen spleen, may also occur as prodromata.

2. The *exanthem* is *polymorphous*, sharply marked, frequently grouped in a circular arrangement and is without pain or itching; it is highly infective, is not reddish as in other eruptions but *copper-colored*, a fact recognized in the fifth century; it appears where other eruptions are rare, as on the forehead or nasolabial fold; crusts may develop on an infiltrated basis. It is recurrent and responds to antisyphilitic treatment. The most frequent form is the *macule* or roseola, next the papular syphilide, and then the forms resembling pustules, varicella, pemphigus or impetigo. In the folds or damp parts of the skin the so-called *mucous patches* appear, as at the angles of the mouth, the groin, or the perineum.

3. *The enanthem* on the mucous membranes is observed on the tongue, lips and cheeks (*mucous patches*), and is often accompanied by pharyngitis.

4. The *lymphatic enlargement* becomes general, although in some cases it may remain more or less localized. Syphilitic rheumatism and iritis are frequent.

III. **The Tertiary Stage.**—This is characterized by the *gumma* which occurs in many tissues. Its specific symptomatology will be described below. The gumma may be microscopic, or an inch or more in diameter. On section it is usually hard, grayish-yellow, homogeneous, centrally caseous and peripherally fibrous. Baümler first remarked that tertiary syphilis, particularly of the viscera (liver, etc.), may be attended by fever. It must be borne in mind that in 20 to 40 per cent. of the cases of tertiary syphilis, no evidence of earlier lesions can be found. The author believes that this explains many instances of the so-called *mendacia syphilitica,* since there is no good reason why a patient should admit having gonorrhœa and lie about a chancre.

Special Symptomatology of Syphilis.—(*A*). **Blood.**—Oligocythæmia and oligochromæmia occur early and may be most marked when the glands are widely involved (Virchow). The anæmia disappears under administration of mercury to reappear if too much be given. A count as low as 1,700,000 may be observed. The "Justus test" is based on the loss of red cells after mercurialization; its value is still undecided. The hæmoglobin is reduced 15 to 30 per cent. and the loss may exceed that of the red cells. The white cells are increased, especially the lymphocytes; the eosinophiles, as in other skin diseases, are increased, but not constantly. The blood contains, especially during the secondaries, the syphilis virus, which is, however, not always inoculable; it has been held that the germ is a tissue rather than a blood parasite but recent studies have shown that the spirochæte is usually present in the blood. Pernicious anæmia and leukæmia may sometimes follow syphilis.

(*B*). **The Skin.**—The macular (roseolous) syphilide is the most frequent secondary eruption. It develops rapidly, lasts eight to ten days, varies in size from that of a lentil to that of a dime, is at first red, then livid, and pressure leaves it yellow. It comes out more clearly by chilling the skin, and sometimes after administration of mercury. It occurs mostly on the trunk. The face, hands, and feet are usually free except in severe cases. It is symmetrical, appears mostly on the flexor surfaces, is circumscribed, recurs more frequently than does any other variety, and is prognostically favorable. It is recognized with difficulty when sweat or other secretions cause maceration. On the scalp focal falling of the hair results. Annular or gyrate forms may occur. Some pigmentation is left.

The *papular syphilide* presents various forms: (*a*) The small miliary papule (lichen syphiliticus), usually develops on the forehead, chin, nose, shoulders, buttocks, back, and extensor surfaces of the extremities and usually corresponds to the glands of the skin. Irregularly distributed, it may appear in rings or circles. At first red, it rapidly becomes brownish-red. It may occur with the roseola or with acne in weak or alcoholic subjects. Fine scales or crusts may form, which, on falling, leave a lacquered appearance; vesicles may form. Recurrences are infrequent. It must be differentiated from psoriasis, the development of which on the scalp, ear, and extensor surfaces is not observed in syphilis, where the scales are smaller and less glistening; the hemorrhage on their removal is parenchymatous and not punctate; the glands are swollen; and the hair thinned. On the palms of the hands and soles of the feet differentiation may be difficult, but in syphilis the centre of the papule is likely to be sunken. (*b*) The large papular syphilide is often associated with fever, constitutional disturbance, or rheumatism, and may occur on all parts of the body, as the *corona veneris* on the upper forehead, around the nose, or in the furrows of the chin and neck. Though infrequent on the palm, it is often seen on the dorsum of the hand. This variety occurs most frequently at the junction of the skin and mucosa and its polymorphism is pronounced, as psoriasis palmaris, rhagades, onychia, or verrucose forms. (*c*) The *condylomata latum* (moist papule) is the papule altered by secretions or excretions, and is found about the genito-anal regions, axillæ, etc. It is the most important syphilide, and plays an important role in the propagation of syphilis by its occurrence on the lips, breast, and genitalia; it is flat, elevated, dirty, ill-smelling, and secretes a highly infective secretion. (*d*) The *pustular* syphilide includes the acne form, the varioloid, varicelliform, impetiginous, and other forms.

Gummata of the skin are asymmetrically disposed; are cutaneous or subcutaneous, involving deeper structures; are non-infective compared with the secondary eruptions; are prone to develop at the site of secondary involvements; occur in the skin more than in all other localities combined; follow incomplete mercurial treatment; are promoted by vicious habits and constitutional diseases; and are seen in 6 to 13 per cent. of cases, most frequently in the third year after infection. Small gummata may resorb by fatty degeneration, but the larger ones caseate, ulcerate, suffer secondary infection, and leave a scar with deformation. The ulcers may remain open and resemble lupus. Gummata are differentiated from malignant ulcerations by the absence of lymph nodes, by their slow growth, painlessness, and multiplicity, and by the results of therapy. In gumma, ulceration is more rapid than in lupus; it is deeper, less sharply marked, redder, and less symmetrical; recurrence after cicatrization is less frequent, the scars are less flat and smooth, and the bacteriological findings of lupus are absent.

Leukoderma syphiliticum is observed in recent, but rarely in late, syphilis—*e. g.*, after macular or lenticular syphilides—as white spots with peripheral brown pigmentation, appearing mostly on the face and neck. Leukoderma usually disappears in from two to twelve years, is rarely seen in either extreme of life, and occurs in 4 per cent. of syphilitic men and 45 per cent. of syphilitic women (Neisser).

The *hair* falls out except from the crown of the head. This occurs most frequently during the secondary stage, and is proportionate to the intensity of the eruption. It sometimes happens during the tertiary stage; is greater in poorly nourished cases, and its regrowth is often imperfect.

The *nails* are sometimes affected. *Onychia syphilitica* occurs particularly in women, in whom it may be the only secondary symptom (Jullien). The nails may hypertrophy, fall out, or fail to grow. Peri-

or paronychia rarely occurs without other specific signs and rarely causes pain, ulceration or suppuration.

(*C*). **Lymph Glands.**—The inguinal glands are first enlarged because they are nearest to the most common genital chancre. Some glands are more involved than others, yet adenopathy in secondary syphilis of any particular group, as the epitrochlear, is not pathognomonic. The posterior cervical glands are involved more than the anterior. Next in frequency are the axillary, epitrochlear, and those along the pectoralis. Inflammation and pain are rare, and suppuration occurs in but 3 per cent. Secondary infection sometimes causes painful inguinal swelling. In extragenital sclerosis, other glands are enlarged first,—*e. g.*, the anterior cervical, from chancre of the lip. The glands are freely movable; may remain swollen for years; and are not usually larger than a hazelnut, but occasionally become massive from trauma, scrofula, or gumma formation, and then may resemble Hodgkins' disease. Virchow describes three stages: (1) irritative; (2) medullary, with leukocytosis, chlorosis, and increase in the stroma and lymphadenoid tissue; (3) caseous, from loss of water and fatty retrogression, and much resembling gummatous formation.

(*D*). **Gastro-intestinal Tract.**—On mucous membranes the eruption (enanthem) is not necessarily characteristic, is less frequent and is less polymorphous than on the skin, with which it occurs synchronously.

MOUTH.—This is more involved than any other part except the skin. Mechanical, thermal, chemical, and bacterial irritation promote eruptions. Recurrence is very frequent. The eruption sometimes occurs earlier than that in the skin.

1. *Erythema* may occur as diffuse or circumscribed maculæ, which are dark red with slight swelling and elevation, especially in the cheek, pharynx, uvula, palate and tonsils—the acute syphilitic *angina;* it rarely passes in front of the border of the soft and hard palate, and it often escapes recognition in women, in drinkers, and in smokers. Desquamation, infiltration, fissures, with a slightly granulated and vesicular appearance, may develop. Erosions may last long in users of tobacco and of alcohol.

2. *Mucous patches* develop just before or with the exanthem, and are seen mostly on the tonsils, cheeks and lips. They are single or multiple, disseminated, round, elliptical or irregular, flat or slightly elevated patches which are circumscribed and of variable color, white, milky, or red. On the lips they may measure 6 to 17 millimeters. The desquamated epithelium may be renewed when little irritation exists, but the course is usually chronic; the patches swell, become granular, ecchymotic, and often ulcerated. On the tonsils, for instance, they may simulate diphtheritic patches, and in smokers a cure cannot be effected until the habit is stopped. Cauterization leaves scars. They are the most important syphilide because so highly contagious.

3. *Chronic angina* occurs in addition to the syphilitic patches. It is of a light red, granular appearance, and is accompanied by hyperæmia, some œdema, and erosion, a gray coating on the swollen ton-

sils, and the general appearance of pharyngitis granulosa. Psoriasis linguæ is usually central, with round or elliptical bright red, flat spots, with thread-like coating; fissures are frequent on the edges of the tongue, and result especially from the use of tobacco and from bad teeth.

4. *Pachyderma*, *ichthyosis*, and *leukoplakia* occur on the mucosa of the cheeks, and on the angles of the mouth, lips, and tongue, and are not always syphilitic, as Kaposi maintained. They are swellings with fissures, are gray in color, irregular, they vary in shape and size, and are thick and scar-like. They may occur early or late, especially in users of tobacco and of alcohol, and rarely in women. They may become carcinomatous. Leukoplakia in tobacco users usually occurs on the tongue, or under the lip or palate, while in syphilis it is mostly on the cheek.

5. *Gummata* of the oral cavity are tertiary. They appear on the lips, when the face is involved, as ulcers, nodes, or deformations. In general they are rare and without glandular swelling, and occur chiefly in smokers. Gummata of the tongue are most frequent, occurring (*a*) as a diffuse glossitis or diffuse infiltration and later as a cirrhotic shrinkage or (*b*) as circumscribed gummata on the dorsum or edges of the tongue, with nodes, swelling, or ulceration, and without pain or glandular involvement. Gummata of the palate or pharynx are mostly multiple, and ulceration may reach the nose, spine, base of the brain, Eustachian tube, or the large vessels. They are dangerous because they are frequently diffuse, deep, or phagedenic; they destroy the uvula, produce adhesions, obliterate the posterior nares or Eustachian tube, stenose the pharynx or draw the tongue back, thus stenosing the respiratory passage. In the tonsils they are very deep and may erode the carotid or palatine artery. Differentiation is required from tuberculosis and epithelioma, in which the glands are usually but not always involved. The diagnosis *ex juvantibus* is important. Carcinomatous or tuberculous change in oral gummata is infrequent.

Œsophagus.—Strictures result from deep gummata, from ulcerations in the larynx, or by extension from the bronchial or other glands. Differentiation is required from tumors of the mediastinum, from aneurysm, carcinoma, round ulcer, and nervous dysphagia. Diverticula may result as in other stenoses.

Stomach.—1. *Acute catarrhal gastritis* occurs in the secondary stage with dyspeptic disturbances and sometimes with great thirst. It is usually toxæmic, and is rarely due to early organic changes in the liver, and other organs.

2. *Chronic gastritis* is more common, in part explaining the syphilitic anæmia and tertiary malnutrition: it may result from early changes in the liver or pancreas. It differs in no wise from the vulgar chronic gastritis except in the influence of treatment. Amyloid change and glandular wasting are infrequent.

3. *Ulcers* of the stomach are not as infrequent as is usually stated. Some round ulcers are syphilitic and result from syphilitic arteritis;

Rosanow described an ulcer of eight years standing, with nocturnal exacerbations, which was cured by inunctions. Lafleur recently diagnosed an ulcer before operation. Lang's contention that 20 per cent. of gastric ulcers are specific is certainly overdrawn. Flexner has collected 16 definite cases.

4. *Gummata*, described by Wagner, Lancereaux, Klebs, Cornil, Chiari, B. Hirsch, Galliard, Andral, and Weichselbaum, are rare. They begin in the submucosa, are usually multiple, leave radiate scars, and differ from the ordinary round ulcer in that the latter is funnel-shaped and broader toward the mucous membrane, while the ulcer of gumma has undermined edges and is broader at its base.

5. *Hemorrhages* from the stomach are most rare, and Hayem's case is almost unique. They may also result from cardiac, splenic, hepatic, or renal lesions.

INTESTINES.—Except for the rectal involvement, little is known of intestinal syphilis in the adult.

1. *Acute catarrhal enteritis* occurs with gastric catarrh, and produces icterus when the duodenum is involved; it resists treatment other than specific.

2. *Chronic enteritis* results from stasis or from acute enteritis. In the syphilitic new-born, enteritis is manifested by the viscid meconium. It may occur with gummata, and disappears only with antisyphilitic treatment. Obstinate diarrhœa and stenosis may follow (Cullerier, Pillon, Mueller, Rieder).

3. *Ulcerations* may result from enteritis in secondary syphilis, from gummatous and diphtheroid changes in the tertiary, and from amyloid disease in the tertiary or quatenary stages. Meschede collected 54 cases of ulcer in the small intestines. They occur around the axis of the intestine, and are characterized by productive inflammation about the ulcer, which prevents perforation but leads to stenosis. They are not frequent in the jejunum or ileum but may reach the colon.

4. Intestinal *amyloid disease* is associated with amyloid of the liver, spleen and kidneys. The gut is pale, smooth, waxy, thick, and rigid, and there are atrophic folds, amyloid ulcers, obstinate diarrhœa, stinking stools, and hydrops. It is most frequent in the small intestine and is an essentially vascular change. Amyloidosis in general occurs in 67 per cent. of tuberculosis, and in 21 per cent. of syphilis (Hoffmann).

RECTUM.—Ulceration occurs in papules near the anus, or results from fissures (Johnson). Rectal disease is usually secondary by contiguity, except in cases of infection from unnatural coitus. (*a*) The presence of irritative *proctitis* in primary cases is not clearly proved, as it is secondary to fissure, catarrh, papules, or ulcerations. It is more frequently chronic than acute, is attended by evacuations of pus, which are voided alone or with the fæces, by itching, tenesmus, and external excoriations, by a long course, and occasionally by secondary tuberculous infection. (*b*) *Syphilitic ulcers* of the rectum are generally tertiary. They occur high up, extend to the colon, and often produce stenosis. In 14 per cent. of cases there are plaques in the rectum (Lang). The occurrence of strictures without previous ulcerations is rare, although Fournier declares it possible. Perforation is rare. The periproctal tissues are invaded. Fistulæ may develop, but the symptoms are less marked than in cancer or tuberculosis. They may increase when the ulceration is low, or when it is complicated with fissures. The fæces are covered with mucus and blood. Diarrhœa from catarrh of the colon, myositis of the sphincter with involuntary evacuations, burrowing of pus, and sepsis, necessitate at best a long course. Tuberculous ulcers rarely occur in the rectum alone, and are marked by the tubercle bacillus. In dysentery the involvement is higher up, colic is more frequent, diarrhœa is more profuse, and the resulting stenosis is higher in the intestine. In carcinoma, age, cachexia and local appearances are suggestive, but not positive criteria. Stricture is lower in syphilis, higher in carcinoma, and adhesions—*e.g.*, to the uterus—are more commonly carcinomatous.

PANCREAS.—Acute syphilitic pancreatitis was first noted by Rokitansky. The interstitial inflammation and sclerosis causing parenchymatous atrophy is rarer in acquired (22 per cent., Hecker) than in hereditary syphilis. The *peritoneum* sometimes participates in syphilitic disease of the organs which it envelops, while it is extremely rare as an independent disease; Lancereaux, Aufrecht and Laurenzi have observed gummatous peritonitis. Seropurulent, serous or localized adhesive peritonitis may occur, as perisplenitis, perihepatitis, peri nephritis, or perityphlitis. Perimetritis, salpingitis, and ovaritis are very rare.

(*E*). **Spleen.**—1. *Acute splenitis*, first described by Walter (1821) occurs in 31 per cent. of acquired and 61 per cent. of congenital syphilis. (2) *Interstitial splenitis* occurs less frequently alone than with syphilitic liver and renal disease. The organ is much increased in size and is hard, tender, and painful from perisplenitis. (3) *Gummatous splenitis* is very rare, and cannot be diagnosticated *intra vitam.* (4) *Amyloidosis* occurs very frequently as the "sago" spleen which is not necessarily very much enlarged; or as the diffuse amyloid spleen, which causes considerable swelling.

(*F*). **Liver.**—Syphilis of the liver occurs in two forms, interstitial and gummatous, which are often associated with perihepatitis, amyloid or fatty change, and sometimes also with such parenchymatous changes as acute yellow atrophy. (1) *Interstitial hepatitis* (syphilitic induration of the liver, syphilitic cirrhosis, or granulated liver) is more frequent in congenital (in 65 per cent.) than in acquired syphilis. The volume of the liver is increased in children, and sometimes decreased in adults. The surface is sometimes even, but is more frequently uneven, with furrows or nodules; the edge is thin, indented, and fairly sharp, whereas in other cirrhoses it is rounded. Perihepatic adhesions to the colon, the abdominal wall, etc., are frequent, and are clinically evidenced by lessened respiratory excursion. The connective tissue forms in Glisson's capsule, follows the branches of the portal vein into the liver (peripylephlebitis), and shows as bands of grayish tissue, the protruding granules representing normal tissue. The liver cells are degenerated, or even destroyed by the connective tissue, or by obliterating endarteritis of the small vessels. The atrophy is most marked in the anterior border of the liver and in the left lobe. The connective tissue is not only perilobular, but reaches into the lobes more than it does in alcoholic cirrhosis. In acquired syphilis the liver is not equally nor completely diseased, whence protrusions of the sound tissue are noted. New bloodvessels develop in the connective tissue from the hepatic artery, the hepatic veins are dilated, and thrombosis may occur in the portal vein and its radicles (pylephlebitis).

SYMPTOMS.—The clinical development is latent or insidious,—*e. g.*, without pain, ascites or œdema. The abdominal veins may be distended; the urine is often dark, possibly icteric, with heavy sediment, and often with albuminuria. The concomitant splenic tumor results from hyperplasia, stasis, or amyloid degeneration. Ascites is less frequent and develops late, herein differing from the vulgar cirrhoses. It usually increases gradually. Icterus is less frequent (33 per cent. Leube). More bile is present in the stools than in vulgar cirrhosis. Gastro-intestinal dyspepsia and vomiting of blood are not infrequent. This form develops more often in men than in women; is promoted by alcoholism; has a chronic course; and its prognosis is rather better than in non-syphilitic cirrhosis, since relative recovery is possible. When the liver is small, recovery is impossible. Death is more common from intercurrent pneumonia, from pleurisy, from cholæmia or hemorrhagic diathesis.

2. *Gummatous hepatitis* (syphilitic hepatitis *par excellence*) is more frequent than the first form, and occurs oftener in acquired than in congenital syphilis. The gummata are either miliary or large nodes. The surface of the liver is smooth or warty, thickened, or adherent to the diaphragm, or colon. The nodes vary from one to over fifty, and may be superficial or deep. The liver is distorted, mammillated or *lobulated* (*hepar lobatum*). Deep, irregular, radiating *furrows* are frequent, resulting from absorbed gummata, affecting either lobe, and occurring mostly on the anterior surface near the suspensory ligament (Virchow) or on the edge. In congenital syphilis gummata are often observed at the hilum, in the wall of the portal vein, or near the bile vessels.

SYMPTOMS.—Gummata are frequently latent—*i. e.*, are discovered only at postmortem. Considerable pain in the shoulder or over the liver often results, as well as pain on motion or tenderness on pressure in the epigastrium or hypochondrium. Pain is due mostly to perihepatitis; a friction rub is heard when the inflammation is fresh; adhesions and loss of respiratory excursion result, in older cases. Icterus is infrequent and results from accidental gummatous compression of the bile duct (Virchow), from perihepatitis (Lancereaux), or from cicatrices. When icterus exists the affection is often mistaken for cancer. The liver may be smaller, but usually shows furrows and nodules. Early enlargement is more frequent than in alcoholic cirrhosis but a final shrinkage has been observed. Ascites is rare except from coincident cirrhosis, amyloid liver, or cardiac insufficiency. When present, ascites is usually terminal, independent of the size of the liver, and is often associated with albuminuria. Splenic tumor is infrequent, except from gummata or amyloid degeneration in the spleen, from coincident liver cirrhosis, from gummata at the *porta hepatis*, or from splenic vein compression. Functional dyspeptic disturbances, bronzed skin (Lancereaux), hemorrhages, and inflammations of the serous membranes, rather infrequently complicate liver gummata.

DIAGNOSIS.—This depends upon other evidences of syphilis, and, while sometimes easy, it is often difficult or impossible. All forms of syphilis of the liver may remain stationary for a time (Bamberger), and in general a longer course is expected than in alcoholic cirrhosis. Oppolzer and Bachdalek mistook gummata for cancer, and the reported cases of cure of cancer by the earlier writers are clearly confusions with syphilis. Age is only a relative criterion, since cancer may occur under thirty years, yet most cases of syphilis of the liver occur under the fortieth year. Ascites, icterus, enlarged liver, and cachexia, may occur in both diseases, occasioning doubt and confusion. The nodules of syphilis are usually small but may be larger than those of cancer. Rapid growth is an indication of cancer; more constant size, of syphilis. According to Bamberger and Peiser, albuminuria and splenic tumor are more common in syphilitic liver. The best clinicians have confused syphilis with cancer, and the safest rule is always to think of syphilis when cancer seems the obvious diagnosis. Coincident amyloid disease renders the diagnosis difficult. The lungs are compressed in cancer more than they are in syphilis. The smooth edge of the liver, lack of respiratory excursion, or signs of fresh hepatitis, indicate syphilis (Lancereaux and Riedel). Riedel found similar symptoms in syphilis and gall-stones, such as pain, fever, and tenderness. Fever is not uncommon in hepatic syphilis, which may then be easily confused with liver abscess (*q. v.*). Final differentiation may be made *ex juvantibus*. (See DIFFERENTIAL TABLE OF DISEASES OF THE LIVER.)

PROGNOSIS.—This is more favorable than in the vulgar cirrhosis, for the disease lasts several years. Spontaneous recovery from gummata may result without specific treatment. Rupture into the peritoneal cavity is rare. Death from cachexia and amyloidosis may occur.

Amyloid liver (*q. v.*) may occur in the congenital type (Rokitansky), but is most frequent in tertiary acquired syphilis or as a parasyphilitic manifestation. Amyloidosis rarely occurs in the liver alone.

Syphilitic *pylethrombosis* (See PYLEPHLEBITIS).

Icterus, especially investigated by French writers, has been attributed to an intestinal enanthem, to swelling of the portal lymphatics, early hepatitis, cholangitis, injury to the liver cells, or to gastroduodenal catarrh. Its connection with syphilis is likely, according to Neumann, when no gastric symptoms precede the icterus; when the icterus develops synchronously with the exanthem; when the exanthem recurs with the recurrence of icterus; and when antisyphilitic remedies remove the icterus.

(*G*). **Kidneys.**—1. *Albuminuria* occurs in cases of recent syphilis or in the later ulceration. It is usually transitory and its connection with syphilis is not precisely known. It is thought to result from toxæmia or vascular changes. Its prognosis is usually good, since nephritis rarely follows it, but the later in the disease that albuminuria appears, the poorer is the prognosis. The use of small quantities of mercury probably does not cause it (Lang), though energetic mercurialization may produce albuminuria, cylindruria and even calcification of the convoluted tubules (Prévost and Saikowsky).

2. Paroxysmal *hæmoglobinuria* occurs relatively often, especially after mercurial therapy. The prognosis is better than in non-syphilitic cases.

3. *Acute parenchymatous nephritis* is focal and cortical. It occurs with the eruption, is toxæmic, and is distinctly rare (although some estimate its frequency at nearly 4 per cent. of the cases). When it develops late, from ulceration, the prognosis is unfavorable. There may be no symptoms; anæmia, vomiting, or other symptoms of any acute nephritis may be present, but uræmia is rare, since it is a focal disease (Bamberger), and the greatest danger is from œdema of the glottis and intercurrent inflammation.

4. *Chronic parenchymatous nephritis* may follow syphilis. Its actual relation to the disease and its percentage are difficult to estimate (0.5 per cent.).

5. *Chronic interstitial nephritis* differs from the ordinary form only in its multiple disseminated focal involvement, which leaves scars. Polyuria is rarely great, casts and albumin are often present in greater amount, and the course is more rapid than the ordinary course of from three to seven years, because of marasmus, amyloid, fatty, and other visceral changes. It often occurs from the twenty-seventh to the thirty-seventh year, when arteriosclerosis is rare. There is said to be no hypertrophy of the heart. Bamberger found syphilis in 3 per cent. of cases of ordinary contracted kidney, but the exact relation is difficult to determine.

6. *Gummata*, in sizes varying from miliary to large forms, which are found in the cortex and sometimes in the medulla, are usually multiple (20 to 80) and generally unilateral. The renal tissue is partly destroyed. They occur especially with liver gummata, amyloid kidney, and chronic parenchymatous nephritis. The symptoms are not well defined.

7. *Amyloid kidney* may occur alone, or is much more frequently accompanied by other renal lesions, especially contracted kidney (Rosenstein). It always occurs in the advanced stages with cachexia and amyloidosis in other organs. The specific gravity of the urine is higher than in the non-syphilitic amyloid kidney (Bartels). The albumin is due to vascular amyloid change, but may be entirely absent

(Ebstein). Globulin may occur in large quantities (Senator). The course is chronic.

8. According to Frerichs, Cantani, and others, syphilis is an etiological factor in *glycosuria*. Mercurial treatment is sometimes held causative. The *suprarenals* are rarely involved, except in congenital syphilis; swelling, hyperæmia, fatty degeneration, gummata, interstitial inflammation, and amyloid changes have been recorded. Gummata have been observed in Addison's disease. Amyloid occurs especially in the Malpighian bodies.

(*H*). **Circulation.**—The heart was found involved but six times in 100,000 autopsies (Mrâcek). Involvement of the heart-muscle and brain-vessels is frequently associated with sudden death.

1. Independent syphilitic *pericarditis* is unknown; it is invariably a complication of myocardial syphilis, occurring over the large vessels or anterior surface of the heart, as serofibrinous pericarditis or complete concretion. Gummatous pericarditis is always combined with the serofibrinous form, and sometimes with aneurysms of the coronary arteries. The symptoms are those of disordered compensation or of myocarditis, its invariable associate.

2. *Syphilitic myocarditis* is coronary arteritis; foci of interstitial involvement occur, as infiltration along the small vessels, which is especially marked in congenital syphilis, and which spreads into the intermuscular septa. Microscopically, the general picture of myocarditis prevails. It involves the front surface or apex of the left ventricle or the intermuscular septum. In congenital syphilis it occurs in the right more than in the left heart. Resulting changes are cardiac hypertrophy, aneurysm of the heart, and Dittrich's true heart stenosis (conus stenosis). *Gummatous myocarditis* seldom occurs alone, and is usually observed with a diffuse myocarditis. Only 97 (62 perfectly clear) cases of myocardial gummata are recorded (Stockmann, Goldfrank). Gummata may attain great dimensions in the interventricular septum. The disease is often latent until, in 50 per cent. of the cases, sudden death results, wherein it differs from myocarditis of other origin. Most fatalities occur in persons between thirty and forty years of age. The heart action may be incompetent or irregular, and there may be dyspnœa, asthma, angina pectoris, or cerebral embolism. Hydrops is rare. Amyloid of the heart is occasionally associated with gummatous myocarditis. Heart rupture is recorded.

3. *Syphilitic endocarditis* rarely develops. The nodules on the valves rarely retract, as they do in rheumatism, and therefore consecutive valvular disease is rare. Mural plaques complicate myocardial gummata. Endocardial gummata may occur.

BLOODVESSELS.—*Endarteritis Syphilitica.*—(*a*) In the *fibrous* form, the wall is thick, and opaque, and the intima and elastic tissue are hyperplastic, and the lumen is eccentric. When the muscular coat is not involved, thromboses occur; when both the muscular and elastic coats are diseased, aneurysms result; this type is known as the obliterating endarteritis of Heubner (see SYPHILIS OF THE BRAIN).

(*b*) Gummatous arteritis or periarteritis, occurring with Form *a* is very rare. (*c*) General arteriosclerosis involves the vessels more widely than does the syphilitic arteritis which is largely localized in the aorta and brain-vessels and exceptionally in the other vessels. Coronary involvement is held by some writers to be more frequent than brain involvement. It may begin in the first year after infection.

AORTA.—*Syphilitic aortitis* (Heiberg, 1877) is oftenest seen in the ascending aorta and its arch and thoracic segment. It begins in the adventitia or intima and ultimately involves all the walls, though in varying degrees. The early stages show gray, transparent, gelatinous patches of productive inflammation in the intima; older foci are gray-white, round or oval, elevated and stellate plaques. The process sometimes becomes deep and calcification occurs in the intima with thrombosis. It may extend down into the aortic valves. Its origin may be (*a*) in the intima, (*b*) in the adventitia and media together, or (*c*) in the intima and adventitia. The resulting loss of elasticity and resistance promotes aneurysm. Welch held that 50 per cent. of aneurysms were syphilitic; personally, the author believes 80 to 90 per cent. is a more accurate estimation. Syphilitic aneurysms develop more rapidly than those due to other causes. They may be single or multiple and usually occur in the arch of the aorta, and sometimes also in the abdominal aorta or its branches (see ANEURYSM). Straub found this aortitis in 98 per cent. of cases of paretic dementia, and Chiari in 50 per cent. of syphilitics. Benda describes gummata (of which only six cases are on record).

PULMONARY ARTERY.—Valvular gumma is very rare, as are occlusions of the pulmonary trunk, or its compression by scars. According to Neumann, syphilitic dilatation or aneurysm of the pulmonary artery has not yet been observed.

VEIN SYPHILIS.—Thirty-three cases of phlebitis were collected by Roussy in 1903. Schuppel has described a periphlebitis originating in the adventitia and seen especially in hereditary syphilis. Gummata sometimes exist in the veins or involve them secondarily.

(*I*). **Respiratory Tract.**—NOSE.—1. An *initial nasal lesion* is most rare, only 95 cases being recorded.

2. *Acute rhinitis* is much rarer in adults than in the new-born but is often not recognized. It is characterized by being less diffuse than an ordinary rhinitis, by its persistence, by erythematous and papular foci, by hemorrhagic erosions with persistent bleeding, by fetor, sometimes by acute sinus inflammation, and by hyperplastic rhinitis.

3. *Rhinitis atrophicans* in syphilis is the result of hyperplastic rhinitis.

4. *Gummata* usually develop late, though they have been seen in the seventh month (Mauriac); they occur on the floor and septum of the nose, and also in the periosteum, bone, and secondarily in the cartilage. They occur with diffuse necrosis or with circumscribed gummata in the skin, and involve the cartilaginous and membranous

septum and cartilages of the alæ; sequestra may be blown from the nose. Septal perforation may develop and a long course is usual, with discharge of pus and crusts; the bone is often denuded. The deformed "saddle-back" nose is due to loss of the triangular cartilage and vomer, and sometimes to simple atrophy. Difficulty in swallowing, meningitis, or disease of the antrum, are occasional sequences. Differentiation from noma, tuberculosis, carcinoma, and the ordinary perforating ulcer of Zuckerkandl (beginning as a hemorrhage and due to streptococcic and staphylococcic infection) must be considered. The diagnosis *ex juvantibus* is the best test.

LARNYX.—1. *Catarrhal laryngitis* occurs with the eruption, and at times is accompanied by swelling of the tonsils and by pharyngitis. The larynx is red and swollen, with a diffuse, thin secretion, which is at first thready and later purulent. Coughing and hoarseness are common.

2. In *papular* and *submucous laryngitis* the papules often ulcerate like ordinary catarrhal ulcers and are often associated with diffuse infiltration and nodes. The epiglottis is also involved. Deforming ulcers are neither so acute nor inflammatory as the non-syphilitic variety. Stenosis of the glottis results if ulcers invade the submucous tissue.

3. *Gummata* of the larynx (2 per cent.) are usually multiple, and vary from the miliary size to that of a pigeon's egg, though sometimes they are simply a diffuse infiltration. They may soften, ulcerate, and produce dysphonia, dysphagia, dyspnœa, hemorrhage, or suffocation. They always leave adhesions, stenosis, scars, or deformity.

Diagnosis.—In cancer the glands are usually, but not always, enlarged; the growth is harder than in syphilis; and the microscopic examination may be of value, but is often unsatisfactory. Syphilis and tuberculosis may occur together. Though the round form of syphilitic ulcer is not of absolute diagnostic value, it usually begins near the epiglottis, is rounder and larger, and has sharper edges, a more reddened border, and a whitish-yellow coating of its deeper base, while the tuberculous ulcer is less red, less infiltrated, less deep and less insidious in its development.

4. *Perichondritis* may affect any cartilage, the involvement usually being secondary. The cartilage is discolored and excoriated, and may be surrounded by œdema, and there may be submucous necrosis, or difficulty in respiration and deglutition. The prognosis is always grave. The lower the necrosis develops, the worse is the prognosis, for deformity and stenosis occur. Syphilis causes 13 per cent. of cases of glottis œdema; tuberculosis, 9 per cent.

5. *Cicatricial stenosis* of the larynx, described in the prelaryngoscopic days by Oppolzer and Türck, occurs mostly at the true cords, usually with adhesions between the cords anteriorly, and sometimes leads to absolute occlusion.

6. *Syphilitic vegetations* are rare, and histologically resemble other vegetations. They are due to irritating secretions; occur in the larynx,

epiglottis, and anterior parts of the cords; appear sessile or pedunculated; and cause dysphonia or even suffocation.

TRACHEA.—The trachea is irritated (*e.g.*, by secretions from the larynx) mostly in its middle third, and next in its lower third. Only two instances of condylomata have been recognized. There may be chronic inflammation in the submucous tissue. The trachea is red, swollen, and sometimes ulcerated and distorted. Tracheal stenosis, resulting from healing gummata, is usually angular (Mauriac); is rarely diaphragmatic, and involves mostly the middle third. The symptoms are a burning sense of constriction; dyspnœa at night, on exertion, or on lying down; a cough resembling that of pertussis, with sanguineo-purulent sputum, which may contain fragments of cartilage; and cyanosis or lobular pneumonia. The diagnosis may be made by the laryngoscope.

BRONCHI.—1. *Acute bronchitis* is observed, especially in tuberculous, cachectic and alcoholic subjects, and was held by Stokes to be an enanthem. It is frequent with the secondary symptoms but may last months.

2. *Chronic bronchitis* often accompanies laryngeal, tracheal, or bronchial affections. When there is much secretion, bronchiolitis or pneumonia may result with signs resembling phthisis. The breathing is disproportionately disturbed (Morton), which is caused by irritation of the vagus from enlargement of the bronchial glands; dyspnœa, fever, pain, and a thin, green, nummular, sometimes bloody, sputum, may be noted.

3. *Kidney-shaped ulcers* may cause stenosis and sometimes perforation into the pulmonary artery, mediastinum, or œsophagus. L. A. Conner, of New York, collected 128 cases of bronchial and tracheal syphilis; in 56 per cent. the trachea alone was affected.

LUNGS.—Syphilis of the lungs, as first recognized by Morton (eighteenth century), may cause symptoms like those of pulmonary tuberculosis, and, according to Sims, every case with tuberculous signs should be searched for syphilis.

1. The *white pneumonia* of hereditary syphilis may be described here although it presents no clinical symptoms. The lung is firm, heavy, airless, and grayish-white. This condition was called white hepatization by Virchow, the chief change being in the thickened and infiltrated alveolar walls. In the air cells, desquamation and swollen epithelium are found, with scattered miliary foci about the arteries.

2. *Diffuse infiltration* differs from tuberculosis in that it occurs in the centre of the lung, on one or on both sides, and exceptionally in the apex (Fournier). It usually travels downward rather than upward. In the beginning the lung is red, hard, large, airless, and smooth on section, and later becomes gray, uneven, and nodulated; the alveoli are filled with fibrin, epithelium, and blood-cells. Infiltration of the connective tissue occurs, particularly about the vessels impinging upon the air-cells. The bronchi are thick, narrowed by connective tissue, or else dilated. The origin of this infiltration is disputed. It occurs either

from desquamative pneumonia, from the alveolar connective tissue, from the bloodvessels, from the peribronchial connective tissue, or from the lymph-vessels. Necrosis, fatty change, and caseation, may result from occlusion of the vessels, which later may result in the formation of cavities or pneumothorax. Disproportionate dyspnœa is present. Temperature may occur with or without ulceration.

3. *Gummatous pneumonia* may occur alone or with diffuse infiltration. Gummata may be found everywhere, but largely in the lower lobes near the hilum, whence they spread to the pleura (dry pleurisy). Differentiation from tuberculosis is often difficult; the two lesions may occur together. Dulness and other signs of consolidation or cavity formation often result. The sputum is mucous, purulent, profuse, bloody, stinking, and contains masses of tissue but no tubercle bacilli. The course is usually chronic, and advanced cases may heal with appropriate treatment. Diagnosis is most difficult if the foci are small. Tuberculosis occurs more often on the left, and syphilis on the right side. Tuberculosis occurs more frequently in the upper, syphilis more often in the lower or middle lobes. The history alone may be misleading. Rise of temperature is more frequent in tuberculosis. In syphilis, hemorrhage is rare, as the vessels are obliterated, and night sweats are infrequent. In caseous tuberculous pneumonia, localization is of no diagnostic value.

4. *Syphilitic interstitial pneumonia* rarely occurs alone, but usually with the above affections. The lung may be lobulated like the lobulated liver, and adhesions may occur at the base; there is marked induration in the middle portions of the lung, as irregular branching bands, which dilate or distort the bronchi; diagnosis is impossible.

5. Syphilitic disease of the pleura is rare. It may occur with lesions of the lung alone or with pleural gummata.

(*J*). **Nervous System.**—The changes are partly toxæmic, partly anatomical. Syphilis of the nervous system occurs in men more frequently than in women (8 to 1), mostly between the ages of twenty-five and forty, in neuropathic subjects, and after trauma; in brain workers or those debilitated by worry, excesses, or from lead or nicotine; and in cases of acquired syphilis, or in children as perhaps the first symptom of parental disease.

The primary lesion often escapes recognition. In 20 per cent. of cases, there is no history of chancre. It occurs where the secondaries have been light and have had ineffectual or no treatment. Syphilis is said to occur in the brain in 16.5 per cent. of syphilitics, more, indeed, than in other viscera, excepting the liver. (Again, it has been held that only 10 to 15 per cent. of syphilitics have visceral, and only 3 per cent. brain, complications.)

Fifty per cent. of brain syphilis occurs within three years after infection. Formerly described as occurring many years after infection, instances are recorded where cerebrospinal syphilis developed even as early as five weeks after infection. Nervous symptoms may occur soon after an infection in those advanced in years (Kuh). Early nervous symptoms, as headache, neuralgia, spinal pain, increased reflexes, or fleeting paralyses, may occur in the secondary stage and are probably chiefly toxæmic.

In general, the symptoms are marked by—(*a*) *variability*, undulation, inconsistency and inconstancy, due to the characteristic growths and vessel changes which regress and recur; (*b*) *incompleteness*, as partial paralysis or partial disturbance of consciousness; (*c*) the symptoms are partly tumor-like, partly vascular, or partly inflammatory; they are partly meningeal, partly basal or cortical, and rarely occur in the centre of the brain except from secondary vessel lesions and gummata. The disease may begin with diffuse neurasthenic, meningeal, psychopathic or focal symptoms. As Wunderlich stated, syphilis produces no symptom not produced by other disease; therefore, no typical picture obtains.

TYPES.—1. BASAL GUMMATOUS MENINGITIS, the most studied type, and, after arterial disease, the most frequent form, begins in the dura, especially along the chiasma opticum, in the interpeduncular spaces, or along the cavernous sinus; less frequently in the fossa of Sylvius and the cortex; pathologically it consists of fibrinous exudate, granulation tissue, cicatrices and gummata.

General Symptoms.—Headache (in 66 to 75 per cent. of the cases) is the most important, constant, and early symptom. It is paroxysmal, or, if persistent, is increased at night, is sharp, boring or dull, is superficial, or deep-seated (Fournier) and but rarely circumscribed. Other symptoms are neuralgias, cerebral (projectile) vomiting, vertigo, and changes in mentality (somnolence, semi-intoxication, or impulsive, motiveless activity and loss of the æsthetic sense). Again, the brain may be normal, except during the attacks,—*e. g.*, coma may occur from which spontaneous recovery is possible. Persistent sleep has been observed, lasting for weeks or months and is usually ominous. The brain activity may be tardy or excited, while nocturnal automatism (Wood) or dementia, may develop, alternating with delirium, epileptic attacks, or paralytic seizures. The brain symptoms may, on rare occasions, resemble those of uræmia, meningitis or typhoid. Epilepsy may be typical or unilateral, frequent or violent; tetanic or cataleptic seizures may occur. Polyuria or polydipsia (33 per cent. of cases), diabetes insipidus (probably resulting from disease in the third ventricle), or diabetes mellitus (of which Oppenheim has collected 20 cases) may occur. Fever may be present; it is irregular, and usually accompanies the secondary manifestations.

The *cerebral nerves* are often affected, especially the second and third. Involvement of the nerve trunks or branches successively is most characteristic (85 per cent., Uhthoff); 80 to 90 per cent. of cases of nerve involvement results from syphilis of the brain, tabes, or less frequently, from general paralysis and brain tumor. (*a*) The third nerve is involved most frequently—50 to 80 per cent. of the cases, "the sign of syphilis" (Ricord). (*b*) Disease of the optic nerve occurs anatomically in 82 per cent. and clinically in 40 per cent. of cases. Choked disk (10 per cent.) is almost always bilateral. (See Retinal Plates under MILIARY TUBERCULOSIS.) Neuritis is unilateral in 4.6 per cent.; simple atrophy, with complete blindness (in 6.6 per cent.), is most frequent in tabes and next most frequent in brain syphilis and general paralysis. Involvement of the opticus rarely occurs as an independent syphilitic lesion. Amaurosis is common and often unilateral. Hemianopsia is homonymous. Temporal heteronymous hemianopsia is very often syphilitic while the nasal form is rarely so. In 5 per cent. of cases the field of vision is constricted. The eye changes, due to meningitis, bone disease, or gumma, are susceptible to treatment. (*c*) The fourth nerve, (*d*) the sixth, and (*e*) the fifth nerve are next in frequency of involvement. The fifth is unilaterally affected, and more in its sensory than in its motor branches. The olfactory nerve is rarely involved. The facial nerve may be paralyzed, nearly always unilaterally and peripherally. The eighth nerve is sometimes involved, and the Ménière symptom-complex results, which, according to Fournier, is ominous. The lower nerves, such as the vagus, or hypoglossus, may be affected. Together with the above changes gummata may grow into the pons, crus, or medulla, and may cause hemiplegia and crossed paralyses, as hemiplegia plus oculomotor paralysis (Weber's paralysis); plus facial paralysis (Gubler's paralysis) or plus abducens and trigeminus paralysis (Leyden's paralysis). In basal meningitis, arterial phenomena are often noted; in the cortical region they are evidenced by syncope or apoplectiform attacks from sudden variation in the blood pressure; toward the base (central ganglia) they produce encephalomalacia, hemiplegia, hemianæsthesia, and hemianopsia. These, however, are usually later symptoms than meningitis and neuritis. The course of syphilitic meningitis is rarely acute or chronic, the typical type being subacute with remissions and exacerbations, and lasts rarely more than a half a year, unless treatment has been given. Headache, convulsions, sopor, coma, delirium and neuritis are followed by irritative and paralytic symptoms with remissions, then with renewal of older, or advent of new, symptoms.

Differential Diagnosis.—*Carcinoma and sarcoma* at the base of the brain, which involve the meninges, run a continuous progressive course, with definite localization and with few changes in the vessels, while in syphilis the course is intermittent or remittent, localization varies and changes in the vessels may be marked. *Tuberculous meningitis* may exceptionally give identical symptoms, but there is less development of connective tissue, vascular changes are rarer, the nerves less frequently involved, the course more acute, febrile, and progressive, and remissions less frequent and less marked; stiff neck and general muscular rigidity occur, which are rare except in the congenital type of syphilis. Mental obscurity is more marked and sudden, while it intermits in syphilis. The irritation, in the initial stage, is followed by paralysis later, while paralysis may occur at once in syphilis. Tuberculous meningitis is a disease of the first, second or third decennium, while syphilis usually occurs after twenty years of age. Other syphilitic or tuberculous foci and the results of therapy determine the differentiation. The two affections rarely co-exist. Differentiation from Quincke's *serous meningitis* may be difficult, but the variations in the syphilitic variety and the results of the therapy are the final tests. Differentiation must be made from *primary syphilitic neuritis* and from multiple syphilitic *root neuritis*. It is questionable whether primary involvement of the second and third nerves may occur in syphilis. It is rare at the base of the brain and these cases are generally caused by an anatomical meningitis with but few symptoms. The multiple nerve root affections of Kahler, involving the seventh and third cerebral and the cervical and dorsal nerve roots, were observed without symptoms of meningitis or tumor, and caused neuralgia, and paralysis. *Optic neuritis* requires differentiation from similar changes due to tumor, alcohol, nephritis, diabetes and acute infections. *Periodic paralysis* of the third nerve is rhythmic in its attacks, usually involves the entire nerve, occurs in young children especially, and with attacks of migraine.

2. SYPHILIS OF THE CONVEXITY may occur as circumscript or diffuse meningitis or meningo-encephalitis, with symptoms like those of cortical tumor or with diffuse manifestations.

General Symptoms.—Headache is usually the first and most common, though convulsions may be the first, manifestation. *Mental* symptoms may occur independently of the localization. *Focal* symptoms are the Jacksonian attacks or epilepsy, which occurs with mono- or hemiplegia, sometimes with local tenderness, develops by starts or "installments" and occurs without an aura. Ninety per cent. of convulsions occurring in those over thirty years of age, not uræmic or alcoholic, are syphilitic (Fournier). The symptoms are more diffuse than in vulgar epilepsy. The convulsions may even number 400 in twenty-four hours. Pain, or paræsthesia occur, but anæsthesia is uncommon. Aphasia is frequent and is usually of the transitory motor type at first, word deafness being relatively rare; aphasia occurs less often from gumma than from vascular disease. Alexia and agraphia have been observed. When the process is diffuse it may be manifested clinically as an acute psychosis which can be cured only with antisyphilitic therapy; dementia is the most frequent type. Both the diffuse and gummatous forms recover more frequently than do other forms.

3. SYPHILITIC ARTERIAL DISEASE occurs alone or with the other varieties of brain syphilis. It is the most frequent form and (aside from paralysis of the nerves), is the most frequent cause of syphilitic paralysis. There are prodromal disturbances; headache is usual but less constant than in meningeal syphilis; vomiting, vertigo, dulness, psychical changes, dementia, convulsions, intermittent hemianopsia, and aphasia may occur; choked disk is rare; thrombosis and obliteration of the vessels is gradual in onset and is intermittent,—*e.g.*, it may involve the leg and, in a few hours or days, the arm. These changes are due to obliteration of the vessels and multiple softening which

(in 95 per cent. of cases) occurs in the Arteria Fossæ Sylvii; pseudobulbar paralysis may rarely result from involvement of the bulbar vessels.

Course.—The first attacks are mild and short; the later ones are more severe, and occur with marked somnolence, very frequently with bilateral, alternating, or crossed paralysis, with general or unilateral convulsions, which are rarely Jacksonian in character, or with partial or mixed aphasia, mental changes, and progressive bulbar phenomena, such as dysphagia. They result from arterial disease or hemorrhage, the latter of which is most rare except from rupture of a cerebral aneurysm.

It may be difficult to diagnosticate primary syphilitic disease from the ordinary *arteriosclerosis;* the latter in over 90 per cent. of cases occurs in late life, its progress is slower, the changes are more disseminated, all coats of the vessels are involved, the more frequent fatty and calcareous changes lead more often to widening than to obliteration, psychical changes and convulsions are less frequent and hemorrhage is more characteristic than softening; whereas in *syphilis,* encephalomalacia occurs at a younger age (See Brain Embolism, Differential Table), the changes chiefly affect the aorta and brain vessels, the intima is chiefly involved (the entire artery and tissue about the artery are exceptionally implicated), calcareous and fatty changes are rare and obliteration is usual. Syphilitic endarteritis is more acute in development and the headache is more severe and is nocturnal. There are such characteristic psychical changes as somnolence or apathy; the hemiplegia occurs by "epochs" or "installments," is associated with meningitis, dementia, or delirium; finally the symptoms are more diffuse than in senile arteriosclerosis. Syphilitic endarteritis sometimes occurs without softening. This type may occur with spinal syphilis or may produce the pseudo-apoplectic attacks in tabes (Minor, Kuh, Heubner, Dinkler). Without treatment, the average duration is one to three months (Oppenheim).

Dieulafoy has carefully described *aneurysms* in the basilar, the Sylvian, and carotid trunks; they are not necessarily tertiary but may occur eight months after infection. Their rupture produces symptoms of apoplexy or meningeal hemorrhage. Otherwise hemorrhage is rare in brain syphilis. They are often multiple. (See illustrations under ANEURYSM.)

While softening is usually ischæmic and not inflammatory, we sometimes encounter foci of *encephalitis syphilitica* as (*a*) disseminated foci in the brain and cord; (*b*) indurated sclerosis; (*c*) softening occurring apart from disease of the vessels, and very rarely observed in secondary syphilis.

4. GUMMATA are the least frequent variety; they may occur in any part of the brain, usually in the meninges, although sometimes in the medullary substance, central ganglia, cerebellum, pons or crus. If they occur alone the symptoms are the same as in brain tumor, except that there is often regression, which is either spontaneous or results

from antisyphilitic treatment. *Cortical gummata,* producing cortical epilepsy and monoplegia, require differentiation from other tumors as tubercle or glioma; in *cortical tumor* the headache is duller and deeper; the pressure symptoms, mental depression, and slow pulse are more marked; the disk changes follow focal symptoms and the process advances less by epochs; while in syphilis the headache is frequently local and the pressure symptoms are more diffuse, being those of more rapid extensive involvement; the disk, as a rule, is involved only when coincident basal meningitis occurs. Cortical paralysis may develop, and is often associated with Jacksonian epilepsy (*v.s.*). The symptoms are more undulatory, and finally success in treatment is permanent (though slight temporary improvement may follow antisyphilitic treatment in cases of genuine tumors).

5. CEREBROSPINAL SYPHILIS produces no characteristic spinal symptoms; suggestive, however, are (*a*) their asymmetry, (*b*) their less pronounced character, (*c*) their variability, (*d*) meningeal and nerve root symptoms, and (*e*) response to mercury and iodides. Syphilis is never a system-disease (Gowers). Syphilis of the cord is five to six times less frequent than brain syphilis.

The most frequent and characteristic form is cerebrospinal meningo-encephalitis, which is observed in both acquired and congenital syphilis. While the symptoms may be strictly spinal (spastic spinal paralysis, myelitis and meningitis), anatomical brain changes, such as gummatous meningitis or meningo-encephalitis are frequently found. Of the three sets of spinal bloodvessels, the peripheral are most involved and Heubner's findings (considered by some characteristic of arterial brain syphilis), are not noted in them. Brain changes usually occur first and may conceal the spinal symptoms, so that doubt often exists whether some symptoms are spinal or cerebral. The meninges are most involved in the cervical and upper dorsal region, as evidenced by pain, stiffness, or girdle sensation; while the cord itself is mostly affected in the lower dorsal region, and therefore involves chiefly the lower extremities, and causes weakness in the legs, paræsthesia, sphincter disturbance, and decubitus. Intercostal neuralgia, pains in the extremities (often most severe at night), spastic paraparesis, hemiparaplegia, triplegia, Brown-Séquard's syndrome, anterior poliomyelitis or amyotrophic lateral sclerosis, increased (varying) reflexes, involvement of the bladder and rectum, and general or selective sensory disturbances, are some of the symptoms of cerebrospinal syphilis. Erb has laid special stress on the spastic paraplegic type.

Varieties.—(*a*) Gummata of the bones. These are exceptional, as are also exostoses and caries.

(*b*) Gummatous meningitis. This is most frequent, and includes pachymeningitis (see p. hypertrophica cervicalis of Charcot).

(*c*) Meningo-myelitis. This is myelitis secondary to meningitis, with connective tissue and vascular changes. It is now maintained that the so-called syphilitic myelitis is only softening—*i. e.*, myelomalacia.

(*d*) Gummata of the cord. These are relatively infrequent.

(*e*) Vascular disease. Softening is rare and involves small areas only.

(*f*) Perineuritis gummosa of the sensory or motor nerve roots.

Differentiation from combined system-disease is often difficult. Some cases of ascending paralysis are considered syphilitic, and some have certainly been helped by mercury. In cerebrospinal syphilis and parasyphilitic tabes or dementia, the lymphocytes predominate in the fluid withdrawn by lumbar puncture.

Steady progression and constancy of symptoms characterize multiple sarcomatosis of the brain and cord.

Prognosis of Brain Syphilis.—According to Naunyn, only 48 per cent. of the cases recover. The later the disease, the worse is the prognosis. The prognosis is more favorable in meningeal (*i.e.*, extracerebral) involvement, when nerve symptoms are dominant, when epilepsy and other symptoms of peripheral brain irritation occur, or when early treatment is instituted. The prognosis is less favorable in specific arterial disease (paralysis from softening), in bulbar forms, in diffuse involvement, in great psychical alteration, and in proportion as the primary and secondary lesions were poorly developed; also in extragenital infections, in hereditary syphilis, and in complicating trauma, tuberculosis or alcoholism. Eighty-two to 88 per cent. of cases of syphilis receive insufficient mercurialization (Hjelman). Recovery is rarely complete and recurrence may be observed. The prognosis is relatively poor in spinal lesions.

6. SYPHILIS OF THE PERIPHERAL NERVES may cause facial and trigeminal paralysis, even in the secondary stage. The most frequently affected spinal nerves are the occipital and auriculotemporal. Other nerves are infrequently involved, as the crural or popliteal. Peripheral (multiple) neuritis is rare, yet isolated perineuritis and neuritis gummosa (nodosa) are recorded.

(*K*). **Eye and Ear.**—Syphilis comprises 2 to 3 per cent. of all eye diseases and at least 3 per cent. of all syphilitics have eye disease. Any part may be involved, except the lens, which suffers only from arterial disease in the uveal tract. The uveal tract is most involved; next in frequency come the retina, optic nerve and eye-muscles. Suppuration or inflammation may occur in the lachrymal duct; the gland is practically immune. The lids are not often involved; chancre, condyloma, and gumma, may occur, and are easily confused with chalazion.

CONJUNCTIVA.—Twenty-two scleroses (Guzeit) have been observed, as well as a few gummata; catarrh occurs usually in the secondary stage.

CORNEA.—The cornea is frequently involved: (*a*) as parenchymatous keratitis, which is mostly observed in congenital syphilis, and occurs even in the third generation, while in acquired syphilis it occurs in but 2 per cent. of cases. "Hutchinson's triad" in congenital syphilis consists of keratitis, notched teeth, and congenital or early acquired deafness. The cornea is diffusely clouded and milky, and on close examination shows patches or streaks of inflammation and sometimes new-formed vessels; (*b*) as keratitis parenchymatosa, the circumscribed or the punctate variety; (*c*) as gumma, which is infrequent; (*d*) as keratomalacia, which is observed in the congenital variety. The sclera is involved secondarily to disease in adjacent parts.

IRIS.—Of all inflammations of the iris, 23 per cent. (Cohn) to 75 per cent. (Mauthner) or 90 per cent. (Klein) are syphilitic. The symptoms are those of other varieties of iritis—ciliary injection with discoloration, narrowed pupil, slow reaction, and synechia or occlusion of the pupil. It is usually plastic, or exudative with tiny papules (Fuchs), and is rarely purulent; gummatous iritis with nodules, if it is not the result of trauma, is strongly indicative of syphilis (in 12 to 30 per cent. of such cases, Klein). Iritis is usually secondary and rarely tertiary, or if so, it occurs in foci of an old iritis. Without cyclitis it is rare in congenital syphilis. As it rarely heals completely, atropine should be administered early while mercury is being given, for expectant treatment results in severe lesions and inflammation tends to recur. Serous iritis may occur in the form of fine brown spots on the posterior surface of the cornea. They are often triangular in arrangement, with injection, disturbance of sight, and a few synechiæ. *Ciliary* involvement is usually secondary, rarely tertiary and may be the sole symptom of syphilis. Injection and exquisite tenderness to touch are characteristic. Because of their intimate vascular connection, cyclitis is often associated with iritis. Gummata usually heal; recurrence is not infrequent.

THE CHOROID is involved late in the secondary period; it may be involved secondarily to iritis or cyclitis. There are several forms but all terminate with much the same appearances—*e.g.*, the exudative choroiditis with bright yellowish, bluish, or even rose-colored spots, which are small, oval, or irregular, and are usually peripherally pigmented. The pigmented type is without spots. Both the spots and pigment lie behind the retinal vessels, which branch over them in the ophthalmoscopic picture. (See Retinal Plates under MILIARY TUBERCULOSIS.) Disseminated choroiditis consists of spots of pigment, especially in the peripheral eye ground, and is usually an indication of syphilis. Choroiditis may occur suddenly, with great pain and reduction in the tension of the eye, and with synechiæ and disturbed vision. When occurring with inflammation of the retina and choroid (choroiditis totalis) the prognosis is usually bad, as blindness may result from recurrent attacks. Though no objective signs positively point to a syphilitic origin, syphilis is always suspected. The *vitreous humor* is frequently involved after other lesions, especially those of the uvea, and is rarely involved alone. As it usually occurs late, it results merely in opacities or dust-like bodies; in the absence of myopia and hemorrhage, it rather indicates syphilis.

THE RETINA is involved chiefly in its anterior layers, becoming cloudy about the disk, with opacity of the nerve and blurring of the disk outlines. The course of the arteries is lost or broken and the veins are tortuous and engorged. Hemorrhages are not common. Most idiopathic cases of retinitis are syphilitic, although no unique morphological characters exist. Nodules, either papules or gummata, constitute the specific retinitis. This is usually late or tertiary and its prognosis is usually good. Central recurring retinitis (v. Graefe), commencing at the macula, is of great importance. Retinitis with pigment formation occurs mostly in congenital syphilis. The *optic nerve* is usually involved with the retina. It may be involved anywhere from its central origin to the disk. It is involved less frequently than is the uveal tract. So-called rheumatic neuritis is often syphilitic. Sometimes the change is simple blue atrophy; again the appearance is nearly normal, or again choked disk occurs. The causative lesions are gumma in the brain, basal disease or independent syphilitic inflammation of the intracranial portion of the tract. Neither its syphilitic nature nor its location can be diagnosticated by the ophthalmoscope. The nerve alone may be involved—*e. g.*, atrophy may occur, as it does in tabes or progressive paralysis. Amblyopia or amaurosis may result from transitory, circulatory, or permanent, organic changes. Hemianopsia is often central, results from lesion in the optic tract, and, in most cases, is transitory.

THE EYE MUSCLES are involved as frequently as is the iris. Syphilis is a more common cause than is usually recognized, explaining many so-called rheumatic cases. More than half the paralyses of the eye muscles are syphilitic. Ocular paralyses may be (*a*) peripheral; (*b*) intracranial as at the base of the brain; or (*c*) central, comprising nuclear, fasicular and cortical localization. Anatomically the following lesions occur: diffuse syphilitic basal meningitis, basal gummata and periostitis, cellulitis of the orbit, diffuse arterial disease of Huebner, nuclear disease, poliencephalitis, gummata of the hemispheres or cortex, and rarely hemorrhage. Other causes, as tabes, general paralysis, tumor, tuberculosis and cancer must be considered. Thirty-six to 50 per cent. of nuclear paralyses are syphilitic. According to Gowers, syphilis frequently produces progressive paralysis of all the muscles of both eyes. The *oculomotor* is involved in at least 65 per cent. of the cases (Alexander, Uhthoff). Total paralysis is rare; if it occurs, the lesion is usually at the base, is rarely orbital, or may be nuclear with localization in the third ventricle, especially if one twig after another become involved. If it is partial, it is usually nuclear, rarely neural or muscular. Ptosis occurs very frequently, and is highly suggestive of syphilis. Abducens and trochlear paralysis may occur. Ophthalmoplegia interna, involving the sphincter iridis and the ciliary muscle, produces mydriasis and paresis of accommodation, and is sometimes an unfavorable symptom. Thirty-six per cent. of all mydriases are syphilitic (Alexander). Its origin is purely nuclear (Mauthner). Ophthalmoplegia externa may occur with facial paralysis, involving the orbicularis palpebrarum, or with trigeminal paralysis involving the sensory filaments of the eye (in 25 per cent. of cases of brain syphilis, Uhthoff).

ORBIT.—Cellulitis and periostitis occur frequently. Periostitis is indicated by deep pain on pressure over the eyeball, or over the edge of the orbit, which is sometimes prominent; by pain on movement of the eye; more rarely by blindness, exophthalmos, and thrombosis. Periosteal gummata may often be mistaken for tumors.

EAR.—Four instances of primary sclerosis on the external ear have been noted (Politzer). The ear in general is involved in 0.6 per cent. in acquired syphilis; 33 per cent. of children with hereditary syphilis are deaf. Secondary eruptions, as maculæ or condylomata, mastoid periostitis or gumma, sclerosis or gumma of the Eustachian tube, suppurative otitis media, sclerosing otitis media and labyrinthine disease (rapid deafness, vertigo, and tinnitus), may be observed.

(*L*). **Gentalia.**—Some primary lesions occur in the urethra. Secondary manifestations are infrequent, though gummata have been occasionally observed in the urethra and bladder.

PENIS.—Gummata may occur at the site of the primary lesion or elsewhere, and have often been confused with the primary ulcer, chancroids, pustules, or carcinoma.

TESTES.—(*a*) The diffuse interstitial form (orchitis fibrosa syphilitica) occurs with great enlargement; the swelling is oval or pyriform, with flattening of the epididymis; it is more often nodular than smooth, and is due to periorchitis (the albuginitis syphilitica of Ricord), or sometimes to gummata. Serous effusion and partial or total synechia occur. Recovery by absorption of this sarcocele syphilitica may result. Some changes may occur in the epididymis or spermatic cord. The connective tissue is increased and the canals are compressed, separated, sparse, fatty, pigmented, and infiltrated with round cells. (*b*) Gummatous orchitis is less frequent, and usually involves one testis, and also the epididymis and cord, more than the first form. Great swelling may occur, may be symmetrical or irregular, and frequently involves the entire organ, which is elastic, firm, and studded with miliary or giant gummata, has thick bloodvessels, and shows perforation with fungus-like granulations, or suppuration. It is often mistaken for sarcoma. The testes atrophy and sclerose. The symptoms begin gradually, or develop after strain or trauma. The anterior portion of the testis is most involved, with single or multiple, hard, irregular nodules, which are usually not painful. The veins are dilated and adhesions occur. The scrotum is infiltrated and nodular, and red or violet in color. Perforation is frequent, with a crater-like opening and irregular edges. Oligospermia, azoöspermia or impotentia may occur, though this is not always the case, because islets of functionating tissue frequently remain. Differentiation is required from gonorrhœal orchitis, which is more rapid, painful, and which involves the epididymis first. Plastic orchitis, resulting from stricture, traumatic orchitis, and mumps orchitis, must be considered; also tuberculous disease, which is often bilateral, or which at least occurs in several foci, and frequently involves the cord, vesicles, epididymis and often causes perforation with pain, fever, and prostration, in which case tubercle bacilli may be detected. Differentiation is also necessary from sarcoma, which is harder, develops more rapidly, is more nodular, produces lancinating pains, and frequently affects the epididymis and cord, which are less often involved in syphilis. Sarcoma also invades the glands, and, when perforation results, is more rapid and painful, and occurs with fever, necrosis, hemorrhage, and cachexia. Carcinoma, which is often difficult to differentiate from the interstitial form of syphilis, does not respond to specific treatment.

VAS DEFERENS.—This is rarely involved, and is seldom affected alone, though gummata may form. Syphilis of the seminal vesicles may convey the disease to the mother and child. Prostatic involvement is rare.

VAGINA.—In the vagina, primary infections are relatively rare, because of its thick epithelium and the paucity of glands; secondary eruptions are rarely observed. Condylomata occur which may be confused with colpitis granulosa. They must disappear under mercury to make certain the diagnosis. Gummata rarely occur alone and seldom affect the whole circumference.

VULVA.—The vulva is very frequently the seat of secondary lesions, upon which tertiary lesions develop from microscopic residua of secondary eruptions. These may extend to the urethra or bladder. Enormous swelling, sometimes resembling blisters, is a common result of lymphatic involvement. Phagedenic ulcerations or strictures may occur. Gummata are readily differentiated from carcinoma and tuberculosis.

CERVIX.—Chancre occurs in 5½ per cent. of the cases (Fournier, Mrâcek and Neumann). Scars may result with stenosis, and sterility. Chancre in this location is promoted by erosions and by a low position of the uterus. Tertiary lesions may occur and recur. Diagnosis may be difficult.

Syphilitic *endometritis* is frequent; its symptoms are the same as those of the non-syphilitic type. Previous endometritis may be increased by syphilis; metrorrhagia may respond only to mercury. Virchow describes a papular and a tuberous variety. Endometritis may produce sterility. *Metritis* syphilitica has been less studied, but some clear cases are recorded, in which large tumors have disappeared on administration of iodide. Specific perimetritis or simple perimetritis is secondary to rectal disease, and is frequently located in the pouch of Douglas, with gummata, adhesions, dislocations or with sterility.

Effect on Pregnancy.—The ovum may be infected without infection of the mother, if abortion occurs in the first two months, before development of the placental circulation. Later the child infects the healthy mother in direct ratio to the advance of gestation. The child may escape infection, only to become infected later by preëxisting or post-conceptional syphilis of the mother. Fraentzel has found peri- and endarteritis in the placental villi with infarction and necrosis from occlusion of the vessels. Thrombosis in the umbilical vessels results in the death of the child, and in abortion. Adhesions may occur, so that only part of the placenta is expelled, and these are, according to Neumann, the foci of the specific disease. Miliary gummata have been found in the maternal portion of the placenta.

Tubes and Ovaries.—Salpingitis gummosa has occurred. Oöphoritis syphilitica has been described and also gummata, which disappear on administration of iodide.

(*M*). **Bones, Joints, and Muscles.**—Secondary *periostitis* produces pain, tenderness, and considerable swelling, which is hard at first, and later is elastic even to fluctuation. It occurs especially on parts subjected to trauma as on the forehead, temporal bone, sternum, ribs, tibia, or clavicle. Osteophytes develop in the cambrium layer, which later becomes adherent. Fugitive pain may occur with the secondary eruption. The pain of periostitis is fixed; it may radiate but rarely migrates, is increased by pressure, is largely boring in character, appears at night—the *dolores nocturnæ*—and disappears at one or two o'clock in the morning with sweating, but otherwise resembles other varieties of periostitis. In 28 per cent. of cases it occurs in the frontal or temporal bones, and forms circumscribed, smooth, elastic, and immovable tumors. It may also occur on the ribs, causing pain on coughing, neuralgia over the sternum, or possibly dyspnœa. Tibial nodes occur in 16 per cent. of cases. Periosteal changes last for five or six weeks and are sometimes associated with arthritis. *Gummata* of the bones were the first well-studied form of gummata. They occur on the exposed parts largely as: (*a*) Diffuse subperiosteal infiltration, which causes molecular destruction of the bone, and possibly reaches the marrow or meninges (perforating type). Osteophytes develop, and are the result of an effort of nature toward regeneration. The bone may rarefy or show foci of sclerosis; multiple areas exist in the pernicious forms of gummata. In large bones sequestra may form, especially in the femur; osteophytes may develop in the diaphysis, and sometimes resemble arthritis deformans. (*b*) Circumscribed gummata, the most important form, develop chiefly in the periosteum; they form elastic, immobile tumors which are not especially tender, and which afterward soften and, at a very late stage, may develop connective tissue and calcification.

Symptoms.—Gummata of the cranium are single or multiple; are attended by dull pain, especially at night; have a slow growth; are at first soft, and later become hard and have a wall-like, hard edge. They rarely ulcerate and often disappear spontaneously, leaving a somewhat elevated periphery and a deep centre. They last for a month or two, and are very often mistaken for supra orbital neuralgia, or migraine. Tuberculous ulcers rarely occur in this location. Pyogenic infection may produce ulceration; or meningeal symptoms such as epilepsy, coma, paralysis, or dementia, may develop. Gummata may occur *at the base*, where periostitis is observed infrequently; they occur chiefly in the middle fossa and involve the nerves or bloodvessels. Diffuse gummatous infiltration develops very slowly, with little pain, possibly with ulceration and exposure of the bone and is diagnosticated with difficulty. The *spine* is rarely the seat of gummata; but when it is involved, the affection is largely cervical, rarely dorsal, very rarely lumbar, and includes the bodies of the vertebræ less than their processes and arches, in contradistinction to tuberculosis. Pain and immobility are observed. The head is held in the hands, if the atlas is involved; recovery is possible, but death may result from ulceration, pressure on the medulla, or luxation. Paralysis of the upper extremities has been observed. The *humerus, radius,* and *carpus,* are less frequently involved. The phalanges may be involved, but this usually affects one finger only, the *spina ventosa,* the parts being swollen, but soft, with periosteal thickening, shining appearance, and rarely pain or ulceration. The clavicle, from its slight protection and its exposure to trauma, is very frequently involved; the course is slow, often without pain. Ulceration of the sternum is frequent; that of the ileum and sacrum is rare. Involvement of the tibia, patella, femur, tarsus, or toes, may occur.

JOINTS.—(*a*) *Acute synovitis,* of recent syphilis, occurs alone or with bone changes at the time of the eruption; pain alone may be observed or the swelling may be multiple, constituting the pseudorheumatism of Fournier, with tenderness, nocturnal pain, immobility, temperature, fluctuation and involvement of the periarticular structures, as the muscles and tendons. The acute form is polyarticular, while the subacute variety is oftener monarticular. The knees are most frequently involved, and, according to Fournier, more than three joints are never affected. The affection may last for weeks or months, does not lead to cardiac complications, and responds to mercury and iodide, and rarely to salicylates. (*b*) *Chronic synovitis* or arthritis is usually monarticular, indolent, with little or no pain or fever, and affects chiefly the knee and elbow, with slow loss of function and fluctuation. It responds to antisyphilitic treatment. (*c*) Joint disease of late syphilis originates in the bone or cartilages (*tumor albus syphilitique* of Richet), and affects chiefly the knee and elbow and next in frequency the ankle. The central portion of the cartilage is involved, and not its edge, as is the case in arthritis deformans. This is probably due to gummata originating in the bone and is most frequently seen in congenital syphilis. The symptoms begin slowly with moderate pain, swelling and localized tumors; no temperature is observed, the symptoms vary, and contracture may result. There may be a close resemblance to tuberculous lesions, diagnosis from which depends upon other stigmata of syphilis or tuberculosis; while tuberculosis is usually inflammatory, incipient syphilitic lesions are non-inflammatory; in syphilis, sequestration and ulceration are infrequent; therapy is the final test.

MUSCLES.—Early involvement may occur, even before the eruption, from (*a*) *toxæmia,* or (*b*) *myositis.* The latter is either a diffuse or circumscribed infiltration, which possibly results in muscular contractures and limited motion, especially in the biceps, arm flexors, finger flexors, sphincter ani, masseters, deltoid, and sternocleidomastoid. The course is subacute or chronic, depending on treatment. The affection is most often observed from two to twelve months after infection. Diagnosis from muscular rheumatism may be necessary. *Gummata* of the muscles are important because of confusion with neoplasms, especially when they occur in the arms and neck; they may be as large as the fist, single or multiple, usually with a firm connective-tissue capsule; they involve the long muscles, especially at their attachments. The course is long, and regression may occur with induration. *Myositis ossificans* is rare. Syphilitic *atrophy* of the muscles may follow arterial disease and, less frequently, central nervous lesions. The *tendons* may be involved, partly from changes in the bones or muscles. (*a*) The irritative form, occurring early as acute bursitis, or as tendosynovitis (Fournier), is sometimes confused with rheumatism. There is more tension than pain. It occurs most frequently in sewing women and washerwomen, and the exudate is usually absorbed. (*b*) The gummatous form, occurring in the flexor tendons, is characterized by slow growth, little pain, rarely by diffuse infiltration, and usually a favorable prognosis.

(*N*). **Mamma.**—The mammæ may be involved (*a*) by diffuse infiltration, by syphilitic mastitis, with increased size, and possibly by tumor formation. The affection occurs largely in the secondary stage, and often resorbs spontaneously. (*b*) Gummatous mastitis is deep-seated and is not very sensitive. Rapid growth may be observed with central necrosis, fluctuation, or even external ulceration. It requires differentiation from adenoma, fibroma, and carcinoma; no glands are involved, it occurs frequently on both sides, without cachexia, but possibly with gummata elsewhere, and it responds to antisyphilitic treatment. (*c*) 206 cases of mammary chancre are recorded (Dimey).

Abnormal Course of Syphilis.—Of special importance is *malignant syphilis,* (syphilis precox, acute syphilis, or galloping syphilis, which are synonymous with early development of tertiary syphilis). The cause may be increased virulence of the virus, or decreased physiological resistance, as in cases of tuberculosis, alcoholism and other cachexiæ. Early ulceration, grave general symptoms and hemorrhage are noted.

Relation to Physiological and Pathological Conditions.—The mortality is great in congenital syphilis (*v. i.*); tuberculosis, retarded development, idiocy, and epilepsy, may result later. On *pregnancy* the disease has a bad influence and mercury is less universally successful. *Exanthematous diseases* may cause syphilis to disappear but their curative influence is short. The effect of coincident measles and smallpox is unfavorable. *Chronic diseases,* as tuberculosis, are unfavorable and often induce the ulcerative form with early tertiary symptoms. Syphilitic cachexia may be complicated by tuberculosis; glaucoma may become aggravated, and syphilitic psoriasis may become carcinomatous. *Trauma* is an important and well-studied subject. In the treatment of fractures in syphilis, mercury is indicated. Pyæmia is seldom observed after operations in syphilis. Syphilis is important in plastic operations. Wounds may become the seat of specific ulceration. In *life assurance*, the outlook is uncertain; it has been estimated that 11 per cent. die before their expectancy.

Hereditary Syphilis.

That syphilis descends only to the first generation is the general belief. Fournier (1905) finds 115 cases recorded in the second generation, of which many are very doubtful.

If both parents are diseased the child mortality is 68 per cent. and the morbidity is 92 per cent.

1. **Syphilis of the Child Acquired from the Father at the Time of Conception.**—The possibility of purely spermatic (paternal) infection is established. It is the simplest and most frequent variety, in that 97 per cent. of cases of syphilis occurs in men (Fournier), although a larger ratio occurs in women of the lower classes. It rarely occurs after the second year of infection has passed. The explanation is difficult, except on the theory that the otherwise non-infective sperma is infective for the ovum only. This form of transmission is more probable, the more recent the paternal disease. The effects are shown by abortion, premature birth, less frequently by syphilitic children at full term, or all of the above successively (the virulence of paternal syphilis decreasing with time). The child mortality is 28 per cent. and the morbidity 37 per cent., this being the most frequent and the most favorable form. The mother may remain uninfected.

2. **Syphilis of Child from Mother, Syphilitic at the Time of Conception.**—Infection is most dangerous in the first year of the disease, and generally persists longer than in man. Abortions occur first, then premature deliveries, and finally, dead or diseased children at term. The mortality is 60 per cent. and the morbidity 84 per cent.

3. **Infection of the Mother and Conception Occurring Simultaneously.**—This form is dangerous to the child, though early treatment may modify the result. According to Neumann, in 29 per cent. of cases, the children are not viable, and 39 per cent. die soon after birth (*i. e.*, 68 per cent. die).

4. **Postconceptional Syphilis.**—The child is less liable to contract the disease, the later the infection of the mother; it may even escape direct infection and only suffer inanition. The virus may pass through the placenta to the child, although there is a theory that the placenta, if not diseased, may protect the child from infection.

Influence upon the Sound Mother of the Child Infected by the Father.—Colle's (or Baûme's) law is as follows: The child is held to make the mother immune to infection; she cannot be infected from her infected child; she can nurse her syphilitic child without danger.

Some exceptions exist and 30 cases are reported in which the mother was infected after the birth of a syphilitic child. Caspary and Neumann have widened the application of Colle's law to the statement that the mother cannot even be infected by inoculation from an initial sclerosis. In such cases it is not true, as Fournier held, that the mother is already syphilitic. Sound children of syphilitic parents have apparently some immunity against syphilis because of action of toxins (Profeta's law,—*i.e.*, the syphilitic mother is said not to infect the sound child, which theory has many exceptions). If the children acquire syphilis, it is said to run a mild course. It is, nevertheless, a fact (Grunfeld) that the sound child may become infected *intra partum*.

Influence on the Child.—Abortions and miscarriages have been overestimated as evidences of syphilis. They are frequent in animals. Abortion, atrophy, maceration, retarded development, congenital syphilis or evidences of the disease after birth, are frequent. Many children, born first after infection, die, though those born later may be sound. A difference exists between the congenital syphilis with signs at birth, and the hereditary type, which develops later (possibly very late, syphilis hereditaria tarda). Even when specific lesions are absent, pathological cell degeneration, rhachitis, scrofulosis, slow intelligence, neuroses, psychoses, and a high percentage of mortality to all diseases, may result.

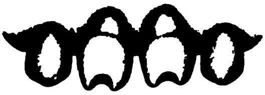
Symptomatology.—The primary, secondary and tertiary stages are not usually present and lymphatic infection does not precede that of the entire organism. Secondary and tertiary manifestations may occur in the most intimate and synchronous connection. The symptoms are usually recognizable at birth or soon afterward, usually after a few days or weeks, and almost always within the first three months.

Appearance.—The *atrophic* child presents a characteristic appearance, with relaxed yellow-gray skin, poorly developed hair and bent or undeveloped nails (onychia). The palms of the hands and soles of the feet are lacquered, the voice and muscles are weak, and the child does not nurse well. Such children, especially when several are born in succession, are suggestive of syphilis, even when no lesions are found. Other *suggestive symptoms* include the heavy placenta whose weight compared with that of the syphilitic child is 1 to 5, 4, or 3 (normally 1 to 6); increasing loss of weight for one to two weeks (normally three days), which may amount to 13 ounces; and enlargement of the spleen.

Pathognomonic Symptoms.—The *exanthem,* as in adults, is polymorphous, but varieties occur which are absent or infrequent in the adult, such as pemphigus, hemorrhages, furuncles, and diffuse infiltration of the skin. There is a general tendency to severe forms. The maculæ are dirty, brown-red, and often confluent, and occur on the face, body, extremities and in the genital region. The papules occur principally in the folds of the joints, in the axillæ, groin and gluteal region and they tend to recur; papules of various ages co-exist with desquamation, though never with condyloma, which is always a sign of acquired syphilis. They tend to ulcerate. Psoriasis of the hands and soles of the feet with desquamation is common. Pustular lesions occur in severe syphilis and are prognostically unfavorable. They occur not only in the new-born but also in the immature fœtus; pemphigus is a subtype of the pustule; gangrene and necrosis may occur. The copper-colored syphilitic infiltration especially on the nates and lower extremities, may often be mistaken for erysipelas. Coryza ("*snuffles*") is very characteristic and almost invariable, the secretion being bloody and purulent, with crust formation. Fissures occur about the mouth, with an infiltrated base and cicatrix formation. Papular eruptions (erosions) occur in the mouth, especially on the tongue and the palate, and the tendency is toward necrosis of the bones. Sequels result from secretion reaching the larynx, lungs or intestines. Eruptions and *rhagades* occur about the genital and anal regions.

Teeth.—Erosions may occur upon the surface and furrows of the teeth and notches may develop. The teeth are small, often irregular and poorly developed. Hutchinson held that the semilunar covering on the free edge of the upper middle incisors was pathognomonic of tardy hereditary syphilis, but *Hutchinson's teeth* occur in other affections, such as scrofula and rickets; they are therefore expressions of constitutional derangement and not necessarily syphilitic, though they are suggestive of it.

Fig. 19.



Notched teeth. Malformation of permanent teeth found in hereditary syphilis. (Jonathan Hutchinson.)

Bones.—The bones, as in adults, may present characteristic findings. There may be periostitis, tophi or perforated palate. *Osteochondritis* occurs in various bones but especially in the lower end of the femur; the diaphysis and epiphysis may be separated. There is (*a*) overgrowth of the cartilage of the diaphysis which produces the wavy line of white cartilage cells; (*b*) the line of cartilage cells becomes thick and foci of decalcification occur; (*c*) the cartilage is as prominent as it is in rickets, the part nearest the bone being soft. The immobility of the leg may be suggestive of paralysis.

Viscera.—The changes are vascular or gummatous. There are characteristic changes in certain organs which are rarely involved in acquired syphilis;—*e.g.*, in the thymus, umbilical cord, pancreas, lungs, intestines and adrenals. To avoid repetition and to conserve the classification, these changes have been described under acquired syphilis (SPECIAL SYMPTOMATOLOGY). Vascular changes are especially marked, as in the umbilical vessels, and the tendency is toward hemorrhage—*syphilis hæmorrhagica neonatorum*. Hemorrhages may occur in the skin, mucous membranes and viscera. They may be profuse, especially in the liver and the lungs, where the circulation is precipitately altered after birth. Some vessels may be thickened or infiltrated (disease of the vasa vasorum). There is often deficient brain development and the posterior cerebral fossa is especially involved; the head may be retracted, and there are other signs of meningitis.

ACQUIRED SYPHILIS IN CHILDREN——*vs.*——HEREDITARY.

Less mortality and less malnutrition.	Marked.
After third month (are exceptions).	Before third month.
A primary lesion occurs.	Absent.
Secondary lesions, then tertiary.	Secondary and tertiary together.
Lymph glands enlarged.	Absent (except from accidental causes or suppuration).
Usual sequence of primary, secondary and tertiary symptoms.	Snuffles, anæmia, diffuse pigmentation, pemphigus, involvement of soles and palms, epiphyseal changes, etc.

Syphilis hereditaria tarda develops its first symptoms in later years, as for instance, at puberty. This variety is recognized by some writers such as Neumann, if (*a*) the parents are syphilitic, and if (*b*) the individual has presented no previous signs of syphilis. Most cases begin between the tenth and eighteenth years (rarely before the tenth and even after the fortieth year). Most cases are seen in private practice. The symptoms are mostly those of infantilism, in regard to puberty, bone development, teeth, and brain.

Syphilis usually confers *immunity*, but this is neither absolute nor life-long, because (1) reinfection may occur (53 instances, Koebner); (2) in hereditary syphilis, a later infection may be acquired (27 cases known) and (3) a sound child of syphilitic parentage may acquire syphilis (29 cases reported).

Treatment of Syphilis.

Prophylaxis.—The disease prevails in spite of the numerous opportunities for free treatment, for the sexual instinct is even stronger than that of self-preservation. Irregular intercourse is possible because of class differences, lack of barriers among the ignorant and destitute, and is promoted by the struggle for existence which often makes early marriage impossible. Prostitution, hidden or open, is the basis of syphilis. Sixty-nine per cent. of women in open houses have the disease for months before treatment is instituted, while hidden prostitution among waitresses, for example, is much more dangerous. In prostitutes, condylomata last as long as seven years and repeatedly recur about the genitalia. Continence and enforced medical inspection with control are the sole prophylactic measures. American sentiment is against licensed prostitution, which, besides, is incomplete because it does not regulate men nor unlicensed hidden prostitution. Many infections are acquired under the influence of liquor. Where ethical considerations fail, an appeal to fear may be effectual.

The importance of the disease lies in its long course, in the involvement of vital viscera, in its transmission to the wife and offspring whose mortality and morbidity are enormous, in its wide dissemination (13 per cent. of the population acquire the disease, and 1,650,000 persons are infected annually), and finally in our possession of specific remedies.

Marriage should be forbidden for at least two, or, better, for three years. Coitus must be avoided when there are recurrences. In cases of doubtful infection of the wife or husband, both should be treated. Inunctions should be given the pregnant mother, whether she be infected in the first or second part of pregnancy. If the child be diseased and the mother healthy, the facts should be laid before her and some would then allow her to choose whether she would nurse her child. Mercury should be given her. The physician should never countenance employing a healthy wet-nurse for the syphilitic child. Circumscision is thought to lessen chances of infection.

I. Initial Lesion.—Extirpation is inefficient. The early use of mercury does not prevent the secondaries nor the recurrence of the disease; it may lessen secondary manifestations, but it is thought that the tertiaries are more apt to follow weak secondary development. We may err in the diagnosis of hard chancre, if we employ treatment in the initial stage. Patients may become immune to mercury when results are most needed—*e.g.*, late in the course of the disease. Nevertheless, the writer believes early treatment is often indicated when the diagnosis is certain. Local treatment includes the use of antiseptics, iodoform, aristol, mercurial plasters, iodoform in suppository in rectum or vagina, bichloride washes for mouth, and calomel for condylomata (calomel 6 parts, boric acid 3, and salicylic acid 1 part).

II. Seconday Stage.—In the secondary stage mercury is a specific. It must always be used with care; the teeth should be cleaned first, and bad teeth should be filled. Calomel acts especially on the lesions of the mouth, and is prone to produce salivation. Mercury is indicated for all of the irritative (*i.e.*, secondary) phenomena of syphilis, and Gowers always uses it with the iodides, even in the tertiary stage, especially in nervous lesions. Mercury may produce bad effects (*v.i.*) in tuberculous, cachectic, alcoholic, malarial, or anæmic subjects, in bleeders, and in cases of galloping syphilis, in which latter, iodides are considered by some better than mercury. Mercury must be used with great care in nephritic subjects.

METHODS OF ADMINISTRATION.—1. *By Mouth.*—Ninety-five per cent. of cases are treated by this method in this country: (*a*) Calomel (Hydrargyri chloridum mite) is especially adapted to children with hereditary syphilis, who are rarely salivated under three years of age; gr. ¼ to ½ t. i. d. plus opium p. r. n. It is insoluble, and is probably converted by the alkaline carbonates in the intestines into the gray oxide which is absorbed with the fats. (*b*) Blue mass (Massa hydrargyri), gr. ½ to 1, is milder than calomel and less irritant to the stomach, but produces salivation oftener than any other mercurial. Women are less subject to ptyalism than men. (*c*) Hydrargyrum tannicum oxydulatum (Lustgarten) contains 50 per cent. mercury, is non-irritant, does not salivate, produces less diarrhœa and is easily assimilated; gr. 1 to 1½ plus pulv. opii gr. 1/10; (*d*)—

℞

Hydrargyri protiodidi	gr. xv.
Extr. opii	gr. v.
Extr. gentianæ	q. s.

M. et ft. pilulæ no. 60
S.—One after meals.

The Hydrarg. iodidum flavum or protiodide is mild and analogous to calomel. (*e*) The bichloride of mercury is irritating (Hydrarg. chloridum corrosivum); it is not prone to salivation, is absorbed by the intestine, and stimulates the liver. Mercury, administered internally, produces more irritation and is less likely to be absorbed. It is used especially in children and in lighter forms of the disease.

2. *Inunctions.*—These must be thorough and should be given where the skin is soft and free from hair (flexor surfaces, and never in the axillæ, the English method). The mercury is rubbed into the sebaceous or sweat-glands and is probably absorbed by the lymph-vessels. The skin must be cared for by daily baths. A simple diet and mild laxatives should be given, though, as a rule, mercury somewhat loosens the bowels. The rubbing should continue for twenty to thirty minutes. Hairy regions, the navel, the nipple, and areas of accidental eczema, abscess, psoriasis, or furuncle, are to be avoided. On the first day the inunction should be given on the abdomen, on the second day on the chest, on the third on the calf, then on the thigh, forearm, arm, etc. If an attendant gives the rubbing, his hand should be protected by a rubber glove. Written instructions should always be given to the patient.

The number of inunctions necessarily varies with the case; twenty to thirty should be given for the macular and papular types; more than thirty, for all pustular or ulcerating eruptions, but not more than thirty should be given continuously. Inunctions must be interrupted for ulceration, diarrhœa, or stomatitis. Salivation is not desirable, nor is it a sign that enough mercury has been given. The *unguentum* contains 50 per cent. of mercury, and the more elegant *oleatum* but 20 per cent., which should be considered, as about ʒss of mercury should be incorporated daily.

℞

Unguent. hydrarg.	℥ iv.
Div. in dos. æqual. no.	xxx
Dent. ad cartam cerat.	

S.—One as directed daily.

In children, mercurial ointment may be applied on a binder (Westlander) and allowed to remain for many days. The ointment is some times also applied to the feet, and the friction of walking rubs the mercury in. The advantages of inunctions are as follows: they cause almost no digestive disturbance; they are especially good in children; they are the most rapid method, and are attended by the least general bad effects. The disadvantages are publicity, troublesomeness, and dirtiness, all of which, however, are but relative objections.

3. *Injections.*—A great diversity of opinion exists as to the value of injections. Other methods should at least be used first. Their advantages are prompt action and exact dosage. Intravenous injections may be imperative in cases with coma and other urgent symptoms. The disadvantages are the risk of embolism, suppuration, sloughing, and pain; once given, their action cannot be regulated or decreased. The soluble salts are best:

Hydrarg, chlorid. corrosivi	1.00
Sodii chloridi	10.00
Aquæ dist.	100.00

One c.c. daily equals 0.01 gm. or gr. $\frac{1}{6}$ of $HgCl_2$; five to forty injections are usually necessary.

4. *Baths.*—ʒij to iij. of $HgCl_2$ to a full bath is useful for skin lesions, such as syphilitic psoriasis.

5. *Fumigations.*—Four ounces of calomel should be vaporized, and the fumes held to the exposed body by heavy coverings. The method is impracticable.

Mercury is incompatible with nearly everything. Coincident anæmia is treated by iron, strychnia, sarsaparilla, and arsenic. Syphilitics tolerate mercury well, in large doses, and for a remarkable period. Indeed, they sometimes seem to develop an immunity to its action. It is not out of place, however, to consider briefly the general action of the drug; in the smallest doses it is tonic, for it increases the number of red cells. In small doses there develops a symptom-complex known as salivation, of which peculiar fœtor is first noted; then sore teeth, metallic taste, red gums which are swollen, spongy, and bleed easily, and increased saliva, follow. The drug is never given beyond this point. In larger doses the tongue becomes swollen, the gums inflamed with a red line and great vascularity; the teeth become loose; the saliva is increased greatly, is viscid and stringy; the salivary glands become intumescent; there is low fever; fugaceous eruptions appear, resembling those of eczema, scarlatina, measles, and more rarely, purpura and pemphigus. Severe salivation results in loss of teeth, great stomatitis, necrosis of jaw, ulceration of the mouth and contiguous parts, marked changes in the blood, and profound exhaustion. The treatment consists of potassium chlorate as an antiseptic mouth wash, and such astringents as tr. myrrh and tannic acid.

III. **Tertiary Stage.**—Iodides are specific in this stage. They are sometimes used in the secondary stage (*v. s.*) especially in febrile cases, in secondary osseous and nervous manifestations, and in weakly persons with a marked macular eruption. Iodides do not operate very specifically in the tertiary stage when no mercury has been given during the secondaries. It acts more slowly than mercury; it acts not only as a specific, but also is useful in removing mercury from its loose, protoplasmic union with the gummata. It is especially indicated when the bones, nervous system, and viscera, are seats of gummata, and when the general health is broken. Patients may acquire immunity to iodides, and its use should therefore be intermittent and should alternate with mercury. Mercury is conjointly employed when organs of great importance are involved, such as the larynx, brain, and eye.

The therapeutic tests for syphilis are (*a*) cure with mercury and iodides; (*b*) tolerance by the patient of very large doses of iodides. Regarding their diagnostic value, Gower's criteria are: (1) that the influence of the drugs must be unequivocal and fairly immediate. (2) That no other remedy be used. (3) That spontaneous remissions which are seen in other diseases (such as brain tumor), must be excluded. (4) That the remedies used must not operate on any disease other than syphilis. (5) Before negative conclusions are drawn regarding the syphilitic nature of the lesion, it must be remembered that loss of tissue, which occurs in arterial occlusion, and paralysis from brain softening, cannot be restored.

The different iodides contain the following percentages of iodine: KI, 75.5 per cent.; NaI, 84.6 per cent.; LiI, 95.5 per cent.; AmI, 87 per cent.

Sodium iodide is milder, produces less iodism, has a less bitter taste, and has less effect on metabolism than potassium iodide, which, however, is better than other iodides (Schmiedeberg), though it irritates the stomach and bowels, depresses the muscles, and disturbs the motor nerves.

The following proportions for a single dose may be employed when no urgency demands large doses:

℞

Potassii iodidi	gr. xv (gm. 1.0)
Hydrargyri chloridi corrosivi	gr. 1/32 (gm. 0. 002)
Tincturae opii	♏. 2 (gm. 0. 13)
Syrupi aurantii corticis	ʒss (gm. 2. 0)

M. et S.—After meals in a glass of water.

Large doses of iodide are sometimes given in urgent cases ʒss–ʒj t.i.d., to break down more rapidly the new-formed granulation tissue. The author believes that enormous doses are unnecessary and are not without danger (*v.i.*). Iodide dissolves in its own weight of water; hence one minim of a saturated solution equals one grain of iodide. It is best disguised by Syr. sarsaparillæ co. dr. ss and Tr. iodi ♏ 1; also by peptonized milk, aërated waters, currant, orange, or raspberry syrups, beer, sassafras, licorice, or anise oil.

Iodism is often produced by small doses, and usually ceases with larger ones. It is obviated by intermittent administration, by daily baths, by stimulating with water the kidneys, the inactivity of which is the usual cause, by giving aromatic spirits of ammonia, or arsenic and belladonna. Iodism is likely to occur in renal disease. Its leading characters are: (*a*) Increased secretion from the eyes, nose, mouth, salivary glands, bronchi, stomach, bowels, skin, and kidneys. The eyes, throat, larynx, and salivary glands swell. The writer once nearly suffocated from œdema of the pharynx and uvula after ingestion of 10 grains of iodide for a cold. Laryngeal and pulmonary œdema and albuminuria, or even nephritis, may develop. (*b*) Gastric irritation. (*c*) Nervous depression, mental derangement, or excitement. These result especially from the potassium salt. (*d*) Acne, hemorrhages, blebs, and telangiectases in the skin. (*e*) Cachexia and anæmia, especially in goitrous subjects; emaciation, wasting of mammæ and testes; fever, increased pulse, nervousness, and neuralgia of the fifth, and other nerves which pass through bony canals.

TUBERCULOSIS.

Definition.—A specific infection, characterized (*a*) etiologically by the Bacillus tuberculosis, (*b*) pathologically by nodes or tubercles, or diffuse tuberculous infiltration, and (*c*) clinically by symptomatology varying with the tissues or organs invaded. The term tuberculosis has special reference to the tubercles, but in the light of our present knowledge, to tubercles caused only by the Bacillus tuberculosis.

History.—The term phthisis (wasting) was first used by Hippocrates. The history of tuberculosis includes several epochs: *Epoch I.* The anatomical tubercle was recognized by Sylvius (1614–1672), Morton (1689) whom some consider the real pioneer, Mangetus (1700), Bayle (1810) who specially described the miliary tubercle. Laennec, the pupil of Bayle, in 1819, spoke for the unity of phthisis. Virchow threw the weight of his influence for Laennec's views, but in 1847, showed that caseation was only a retrogressive change and not necessarily tuberculous. In 1857, Buhl described a case of miliary tuberculosis arising from old foci of caseation. Cruveilhier, Rilliet, and Barthez identified scrofula and tuberculosis.

Epoch II was opened by Villemin (1865) who positively established the inoculability of tuberculosis by experiments on animals. Klenkpe (1843) made successful inoculations, but Villemin absolutely proved his point. It is interesting to note that from the time of Hippocrates, Aristotle, Aretæus, and Galen, tuberculosis was feared as a contagion, and the older pathologists, as Morgagni, dreaded to section phthisical subjects. Villemin's formulations met great scepticism and opposition, but were supported by Fox, Charcot, Grancher, and Thaon, and were corroborated by Cohnheim and Salamonson. The histology of the tubercle was developed by Wagner, Schueppel and Virchow; Langhaus fully described the giant cells previously seen by Virchow and Rokitansky; tubercles were found in lupus (Friedländer, 1875), in lymph-glands (Schueppel, 1871) and in fungus joints (Koester, 1876). The firm and ancient belief in its contagiousness led to the successful search for the cause. It seems strange that some, as Aufrecht, Baumgarten and von Ziemssen, still practically deny the infectivity of tuberculosis.

Epoch III began with Koch's announcement, in 1882, of the tubercle bacillus. His report was so complete that scarcely a single essential point has since been added to his initial communication. The bacillus was found in all tuberculous lesions, such as chronic phthisis, miliary tuberculosis, intestinal ulcers, scrofulous lesions, bone and joint disease, lupus, and in the sputum, in cattle, etc.; it occurred in no other disease; it was cultivated by him and inoculated into animals with reproduction of tuberculosis.

Bacillus Tuberculosis.—1. Morphology.—It is a thin, narrow rod, measuring ¼ to ½ of the red blood-cell (1½ to 4μ long) and more often slightly bent or curved than straight; it contains no spores, the light unstained areas in its body being vacuoles or spots of degeneration; it is generally considered immotile; in young cultures it is shorter than it is in the sputum, or in older growths; in old cultures and in lung cavities it is longer and is frequently thread-like; it occurs singly or in groups and bundles. Pleomorphism: variations in form are often noted, as bulging sides or ends, thread-like or branching forms and even forms said to resemble actinomyces. [See Figure under PULMONARY TUBERCULOSIS.]

2. Staining Reaction.—It stains slowly, except at body-heat, and once stained gives up the dye reluctantly. Ziehl's solution is the best for clinical purposes (Fuchsin 1, Absolute alcohol 10 and 5 per cent. carbolic acid solution 100). The sputum is spread out in a dish over a black background and the yellow-green areas selected are thinly distributed on the slide which is held in the hand, and is heated until dry at some distance from the flame; it is then stained by the carbol-fuchsin, being passed repeatedly but lightly through the flame (without boiling) to deepen the stain; it is then washed, treated with Gabbett's stain (1 part methylene blue to 100 parts of 25 per cent. sulphuric acid) for ten to thirty seconds, washed well, and dried. If the sputum is thick or if previous examinations were negative, quantities of sputum (15 c.c.) with twice their bulk of water (30 c.c.) are treated with caustic potash (8 drops of a 10 per cent. solution), are shaken and allowed to settle for some hours, after which the supernatant fluid is decanted, and the sediment is examined directly or after centrifugation. Spengler digests the sputum with pancreatin instead of using potassium hydrate. The bacillus resists destaining because of its fat, an almost pathognomonic feature. For clinical purposes only (*a*) the bacilli found in bronchiectasis and gangrene of the lung (*q. v.*), (*b*) the smegma bacillus and (*c*) the leprosy bacillus, need be considered; the leprosy germs are intracellular, cover the nucleus of the containing cell, and are stained in six or seven minutes with alcoholic fuchsin, while tubercle bacilli are extracellular, stain slowly, and require more time (by the cold method) than that specified; the smegma bacillus causes many errors in genito-urinary examinations; it grows rapidly in twenty-four hours, does not show the beaded appearance which is seen in the Bacillus tuberculosis, and when stained and counterstained in the usual way, becomes tinged with methylene blue (Weichselbaum).

3. **Cultures.**—These develop best at body heat in one or two weeks; blood serum is the best medium, but growths are also successful on bouillon, glycerine-agar, and potato; they are dry, thin, grayish, and scale-like. The germ is a facultative aërobe and multiplies by transverse fission. It seemingly does not lead a saprophytic life in nature, outside the bodies of animals and of man.

4. **Chemistry and Metabolism.**—It contains 2 to 16 times as much fat as other bacteria. Aronson holds that a waxy substance envelops the bacillus; its other constituents are proteids, a glycogen-like substance, cellulose, nucleic acid, water, and ash; it also develops tuberculin whose chief components are peptone, albumose and alkaloidal substances; it is soluble in glycerine.

5. **Resistance.**—Its resisting powers are as a rule limited. (*a*) Drying is endured by the bacillus for a few months (six or even ten months). In decomposing media its average life is one to one and a half (possibly six) weeks. (*b*) Heat (106°) kills it in a few minutes. (*c*) Cold is fairly well resisted and it may survive 14 below zero for weeks. (*d*) Sunlight kills the bacillus in a few minutes to a few hours.

6. **Virulence.**—Koch was inclined to assume that it had an equal virulence independent of the source of the germ, while the Arloing school finds that it has an attenuated virulence in glandular and osseous lesions, and Theobald Smith holds that bovine cultures are more virulent for rabbits than is human sputum.

7. **Agglutinating Properties.**—Arloing and Courmont (1898) found that serum from inoculated rabbits or goats, agglutinized tubercle bacilli in a dilution of 1 to 5 or 20, in two to twenty-four hours; eight to twelve day cultures, grown on 6 per cent. glycerin-bouillon with 1 per cent. peptone, gave the best results; agglutination was noted in 95 per cent. of the cases of lung tuberculosis and in 50 per cent. of the cases of surgical tuberculosis. Its differential value has not, however, been generally conceded.

8. **Associated or Mixed Infection.**—This has been much emphasized by Ortner, Weichselbaum, Struempell, Roger, Dieulafoy, Mosney, Marfan, Osler, Wolf, Spengler, and King, though Straus, Leyden, Baumgarten, Fraenkel and Troje lay but little stress upon it. Symbiosis has been noted with the streptococcus, staphylococcus and pneumococcus, to a lesser degree with the Bacillus pyocyaneus, influenzæ, prodigiosus, pseudodiphtheriæ, Friedländer's bacillus, proteus vulgaris, micrococcus tetragenus and sarcinæ, aspergillus, streptothrix, amœbæ, and monads.

9. **Number of Bacilli.**—Large numbers are found in fresh tubercles; few are found in chronic articular, osseous, or glandular lesions, and inoculations may be necessary to demonstrate the nature of long-standing lesions.

Prevalence in Man.—Tuberculosis is the greatest foe of man; it causes 14 per cent. of all deaths, and 33 per cent. of deaths between fifteen and forty-five years of age; it causes more deaths than war, famine, plague, cholera, yellow fever, and smallpox combined; in Germany, in 1894, the deaths from diphtheria, croup, measles, scarlatina, pertussis, and typhoid, together, were 7,000 less than those from tuberculosis. Five million die yearly from this plague, one million in Europe, seventy thousand in England, 150,000 in France, 150,000 in the United States, and 170,000 in Germany. Postmortem and hospital statistics do not exactly depict the tuberculosis situation; in Austria, the general death-rate from tuberculosis is 11 per cent.; the hospital rate is 25 per cent., the patients being drawn from the poorer classes. The true total is obtained by multiplying the annual death-rate by the average duration of life (three years).

Quiescent (latent) and healed tuberculosis is found in many postmortems upon those dying of other diseases; Heitler, of Vienna, found obsolete pulmonary tuberculosis in 4.8 per cent. among 16,562 autopsies. Latent tuberculosis occurs in 25 to 33 per cent. of the population (Baumgarten, Birch-Hirschfeld and Bollinger), 39 per cent. (Harris) and 50 per cent. (Cruveilhier, Roger, and Déjérine). O. Naegeli found gross or microscopic evidence of tuberculosis in 97 per cent. of all necropsies. Recently Franz obtained a tuberculin reaction in 61 per cent. of apparently healthy recruits.

Prevalence in Animals.—(*a*) *Cattle.*—In Germany, it is found in from 4 to 20 or 30 per cent.; in Leipsic, it even reached 20 per cent. in bullocks and 48 per cent. in cows. Calves are less frequently diseased. In New York, the tuberculin test was positive in 17 per cent. In 1898, Theobald Smith, and later, Koch, contended that bovine and human tuberculosis were separate infections; opinions are still divided on this subject, though most writers incline to the view of Smith and Koch. In cattle, tuberculosis is less often attended by softening of the tubercle, and the serous membranes are more often involved; the lungs and pleuræ are involved in 40 per cent., the lungs alone in 20 per cent., and the pleuræ and peritoneum in 15 per cent. of tuberculous cattle. (*b*) *Swine* are affected more often in Europe than in this country; in Saxony tuberculosis occurs in 1 per cent. and in Copenhagen and Dantzig, in 11 per cent.; because their infection commonly results from eating tuberculous offal, the type is usually alimentary (90 per cent.). (*c*) *Other mammals.*—Dogs and cats are seldom diseased except from contact with man; the type in dogs is respiratory (83 per cent.). Horses, sheep, and goats, are rarely diseased. Monkeys in captivity die frequently (43 per cent.) from tuberculosis, referred by Dieulafoy and Krishaber to foul cages. (*d*) In *reptiles,* it is rarely seen, except in those in captivity. (*e*) In most *birds,* tuberculous lesions are more frequent in the liver, spleen, intestine, mesenteric glands, bones, and joints, than in the lungs, though parrots suffer from skin and lung localization. In avian tuberculosis the bacilli grow rapidly, present greater morphological variability, and live longer, which characters seem to justify their separation from the human type. Man may apparently become infected (Pancini), and there are those who still contend for the unity of the human, bovine, and avian bacillus. Tubercles are not uncommon in animals from nematodes, protozoa, and bacteria, and are designated pseudotuberculosis. In man, Flexner has described a streptothrix pseudotuberculosis causing caseous pneumonia and peritoneal nodes; the streptothrix differed both from the tubercle bacillus and the ray fungus. An aspergillus tuberculosis has been described (*v.* diagnosis TUBERCULOSIS OF LUNGS).

Atrium.—The details will be especially considered under the different clinical localizations of the disease.

1. **By the Respiratory Tract.**—The bacillus enters the respiratory tract by inhalation. In the vast majority of cases the sputum of diseased persons is causative both of the disease, and its great frequency of localization in the respiratory organs. It has been estimated that a phthisical person voids 7,200 millions of tubercle bacilli daily. As the expired air is proven germ-free, the sputum must be the chief factor in dissemination. Cornet holds that the greatest danger lies in the drying of the sputum in the room, on the handkerchief, on the lips, beard, hair, fingers, and clothing, though Fluegge contends that dried sputum is less dangerous than the moist expectoration. Fluegge demonstrated that in coughing, which seemingly voids no secretion, in singing, in sneezing, or talking, small and almost invisible drops of moisture containing the bacilli are voided, and he has cultivated bacilli from glass plates which were covered with solid culture media and held near the patient; he believes that minute particles remain in suspension in the air and thus infect other individuals. Drying of the sputum voided in the street or other open places is less significant than was formerly thought, since the sun soon kills the bacilli. Hence the ubiquity of the tubercle bacillus is generally exaggerated though it is of great importance in poorly ventilated, dark dwelling-rooms, hospitals, cloisters, hotels, or prisons, which are frequented by victims of tuberculosis. Cornet found the bacilli in the dust of such rooms in large numbers, yet in about 10 per cent. of tuberculous wards no bacilli were found; he experimentally demonstrated that animals confined in rooms with dried tuberculous sputum readily acquired tuberculosis. Straus, of Paris, collected the germ from the nose in 31 per cent. of the attendants in tuberculous wards. According to Cornet, infection is less frequent than might be expected, because the mucus and the bacilli absorb moisture from the air, and their hygroscopic properties thus make them resistant to drying. The closer the contact with tuberculous subjects, the greater is the danger of infection, as tuberculosis is surely an infection. In crowded prisons, the death-rate is three or four times that of the general community; in Austria, for example, the general mortality is 11 to 15 per cent., and the prison mortality is 40 to even 60 per cent. In nursing sisterhoods, Cornet found the death-rate ranged from 63 to over 75 per cent. Six cases were observed by the writer, in two years, in one small, closely

cloistered community. In Philadelphia, Flick's studies showed that 33 per cent. of infected houses contributed more than one case. In Falkenstein, Germany, and Brompton Hospital, London, acquisition of tuberculosis by attendants is infrequent, while in Paris, Straus puts their mortality at a high figure. In our dispensary service, seven rapidly fatal cases were observed in one family which had no hereditary stigma nor previous disease (Harpole). Oral breathing, caused by adenoids, inhalation of coal-dust or stone-dust, syphilis, cancer, and leprosy, are favoring factors.

Dusting, sweeping, or shaking the bed clothes, disseminates the virus; there is less danger of infection when the windows and doors are open and when dusting is done with moist cloths rather than with feather-dusters. In hospital wards there are more germs in the air during the cleaning hour than during the other twenty-three hours. However, if bacilli are in the dust, they are not necessarily in the inhaled air; if inhaled, they do not necessarily reach the lungs, for they may be caught in the upper air-passages whose secretions antagonize their development, and are often waved back by the ciliated epithelium of the lower air-passages. Cornet holds that the statement that "every man lives in the environment of tuberculosis" is surely exaggeration. Married persons, because of their intimate association, may contract the disease from each other, as was noted by Ettmueller and Morton, in the seventeenth century; the husband more often conveys infection to the wife than the wife to the husband; in Weber's series of cases, a husband lost four wives, another three wives, and in four other cases two wives contracted tuberculosis. The danger of sleeping and living together is somewhat overdrawn, as the family members are less together than is at first apparent. When bacilli are inhaled into the respiratory tract (*a*) no lesions may result, (*b*) the lungs may become diseased, or (*c*) the germs may pass the mucosa of the mouth or bronchi and cause glandular tuberculosis—*e. g.*, cervical or peribronchial adenitis tuberculosa.

2. By the Digestive Tract.—Milk is most dangerous (Gerlach, Bollinger, Bang, and others), though lately attempts have been made to minimize the danger from this source of infection. It was formerly thought that, in cows, a tuberculous mastitis was necessary to milk infection, but Ernst and others demonstrated that milk may be infective from cows with tuberculosis which does not invade the udders. Pigs, fed on tuberculous milk, develop intestinal tuberculosis. Butter may contain tubercle bacilli. Meat is less dangerous, as it is usually well cooked, but tubercles in raw offal frequently infect swine. Bacilli may pass the mucosa, especially in children, and produce the first lesions in the mesenteric glands.

3. Direct Inoculation.—Inoculation through the skin, eye, genito-urinary tract, by trauma, or operations, is far less significant and will be considered under the various tissues occasionally involved. The point of entrance is often hard to find.

Predisposing Factors.—These are important, but if practically considered, tuberculosis is best prevented and treated as an infection, communicated and acquired by direct personal contact.

I. Heredity.—1. *Hereditary Predisposition to Tuberculosis.*—This is shown by the frequency with which a tuberculous family history is obtained in infected individuals, and is estimated at 25 per cent. (10 to 85 per cent.). It is remarkable what statistics can be made to establish, and if moved by theory alone, one might prove by figures a formidable hereditary influence in measles, pneumonia and other common diseases. Kuthri found essentially the same percentage of family tuberculosis in non-tuberculous as in tuberculous cases. Tuberculous parents may infect their children (50 per cent.), as they may infect others, but the children usually (97 per cent.) escape the disease when removed from their parents. According to Rieffel, 3 per cent. of children acquire tuberculosis when both parents are sound, 22 per cent. when both parents are tuberculous, 10 per cent. when the mother and 7 per cent. when the father is tuberculous. Whatever stress may be laid on statistics, hereditary predisposition is no obstacle to prevention and treatment.

2. *Congenital Tuberculosis.*—It is thought by Baumgarten that children actually inherit the bacillus, which remains latent, possibly for years, and then develops an active tuberculosis. If his views are correct, we are confronted with a difficult problem. Considering the possibilities of bacilliary inheritance, congenital tuberculosis can be acquired only through (*a*) the spermatozoön, (*b*) the ovum or (*c*) placenta. (*a*) The spermatozoön is a nuclear structure which is not proven to be attacked by the tubercle bacillus, though the bacillus is sometimes found in the semen in genito-urinary tuberculosis. (*b*) The ovum has, in rare instances, been infected, but Virchow, among others, questions this possibility. Mafucchi found that tuberculosis developed in chicks, weeks or months after the ova, from which they were hatched, were infected. (*c*) Placental tuberculosis has been observed clearly in about 22 human cases (according to Warthin and Cowie in only 5), and slightly over 100 cases in animals (Friedman, 1905). The first indisputable case was that of Schmorl and Birch-Hirschfeld which was confirmed by the inoculation and finding of the bacillus. The maternal and fœtal circulations are quite distinct, but toxæmias may occasionally allow the virus to pass to the fœtus, that is, in typhoid, scarlatina, smallpox, or anthrax. Placental transmission is observed more often in animals (½ to 1 per cent., Klepp). Usually there is a placental focus before the fœtal blood is infected, but the bacillus may sometimes pass directly to the fœtus. Placental infection is possible, but is so infrequent that many experienced observers, like Virchow, have never seen a case. However, Schmorl and Geipel recently found tuberculosis in nearly 50 per cent. of placentæ of tuberculous mothers.

The arguments against hereditary tuberculosis are (1) that children born with the disease die very early; (2) that tuberculosis in the first months of life is extremely rare; (3) that the disease seldom remains latent until adult life, as most cases of infantile tuberculosis develop during the period of special growth, namely from the second to third year; (4) in congenital infection, the liver is chiefly diseased because of the nature of its vascular relations to the placenta; in tuberculosis of children, the lungs, glands, and other organs, are more often diseased,—*i.e.*, the infection is respiratory, and therefore, extra-uterine. (5) When removed from their tuberculous parents, children thrive well.

II. Environment.—This covers many points which were mentioned under ATRIUM. Dampness and poor drainage, emphasized by Bowditch and Buchannan, are factors which reduce the physiological resistance. Trudeau's experiments show that inoculated animals, kept in dark, damp cellars, die, while others, allowed to run at large, outlive the infection. Density of population raises the proportion of tuberculous cases. In recent years the large cities have shown an apparent decrease of tuberculosis, as shown by Edward F. Wells, of Chicago, and Jas. B. Russel, of Glascow. It has also decreased in New York and London. In Massachusetts the total is less by half than it was fifty years ago. The decrease is attributed to more intelligent hygiene. The figures show a decided decrease in Chicago (*v.* PNEUMONIA); it is possible, in recent years at least, that this is explained by the fact that pneumonia carries off the less resistant of the population. In Munich, the death-rate from tuberculosis was 12 per cent. at a time when typhoid was epidemic; when typhoid was practically abolished, the tuberculosis rate increased to 16 per cent.,—*i.e.*, 4 per cent. of those with tuberculosis had previously died from typhoid.

III. Occupation.—Occupations involving work in close, damp rooms, in dust, in crowded quarters, particularly when associated with poor food, alcoholism, worry, and over-exertion, promote tuberculosis when exposure to infected individuals exists. Clerks, stenographers, or waiters, are naturally more easily infected than are those working in the fresh air, for example, cab-drivers. It is rather surprising that miners do not suffer more frequently, but the explanation of this is the dampness, which prevents the drying of infected sputum.

IV. Age.—Cornet's figures show the following death-rate per 10,000 of population: first year 2 to 3, the child being in close contact with the mother. These figures grow smaller, until the lowest figure is reached, namely, ½, between the fifth to tenth years; fifteenth to twentieth years, 17 to 19; twentieth to thirtieth years, 32 to 36; thirtieth to fortieth years, 44; fortieth to fiftieth years, 55; fiftieth to sixtieth years, 76; sixtieth to seventieth years, 100; seventieth to eightieth years, 70; over eightieth year, 20. In the first decade of life, glandular, meningeal, osseous, cutaneous, and articular tuberculosis are more frequent; the mucosæ are more permeable and the lymph channels wider, so that infection is very often first evidenced in the lymph glands.

V. Sex.—Between five and twenty years of age, girls are more prone to tuberculosis, because their life is more sedentary. After twenty years males more often contract tuberculosis, because of their greater exposure to infection. Repeated pregnancies and protracted lactations may favor infection by lowering physiological resistance. Skin lesions are more frequent in women; lupus, for example, occurs in 68 per cent. (in males in 32 per cent.); tuberculosis of the nose, mouth, and throat, is less frequent in women (4 per cent.) than in men (81 per cent.).

VI. Climate.—Warm, dry climates, high altitudes, and lack of sudden variations in temperature, are generally held to reduce tuberculosis. Of late they are apparently considered less beneficial. The French Alps were long free of tuberculosis until crowded by infected subjects, and the same seems true of other localities. Some writers even hold that the chief advantages of many resorts is their sparse population and inaccessibility.

VII. Race.—In this country the Irish and negroes are most frequently diseased. The Indians have justly come to dread the "coughing white man," and they die in large numbers from consumption, particularly in their Eastern reservations. Infection among Jews is peculiarly infrequent.

VIII. Individual Predisposition.—This was considered important by the oldest medical writers, who described the winged scapulæ, the paralytic thorax, and the depressed sternum; but poor thoracic and general development is very often an expression of already existing tuberculosis; phthisis often occurs in splendidly developed chests, exposure to infection being the most important factor. Tuberculosis confers no immunity to relapses or to new localizations; a lung lesion may heal while a coincident intestinal lesion progresses.

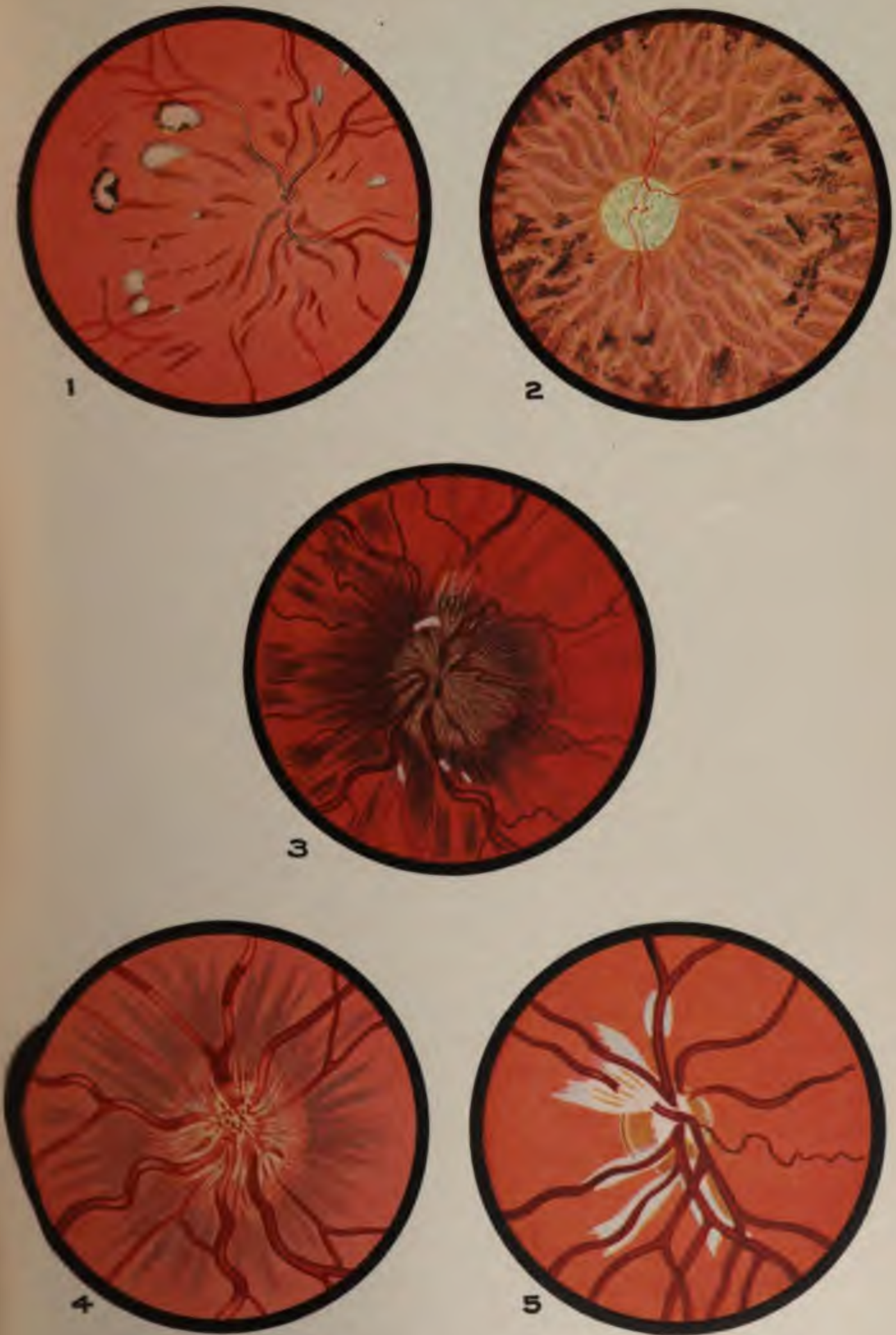
IX. Trauma.—This is a somewhat determining factor. Bacilli in the blood may be localized in a bone or joint by trauma; a blow on the head or spine may initiate meningitis in a tuberculous subject; pleurisy or pulmonary tuberculosis may be initiated by injury to the chest, which is thought to awaken latent lesions in the peribronchial glands.

X. Other Factors.—These are (1) diabetes, in which 23 to 50 per cent. die of tuberculosis of the lung; (2) psychoses, especially of the depressive type, as melancholia; (3) anæmia, which, however, is more often the result than the cause of tuberculosis; (4) inflammation of the mucous membranes, acute processes, such as heavy colds, measles, and pertussis, and less often scarlatina, influenza, or diphtheria, may predispose to infection, or they probably more often awaken slumbering glandular tuberculosis by the irritation of the mucous membrane by products absorbed from the bronchi or lungs. Enterocolitis in children, or typhoid in adults, may in a similar way awaken or initiate abdominal tuberculosis. Smallpox and eczema sometimes seem to produce similar effects on the skin. (5) Contracted kidney, cirrhotic liver, sclerosed arteries, stenosis of the pulmonary artery, aortic aneurysm and narrowed hypoplastic aorta are predisposing factors. As Rokitansky first pointed out, valvular disease of the left heart prevents or checks infection, because it congests the lungs; Bier's treatment of surgical tuberculosis, by inducing congestion through ligatures, is based on this point; Birch-Hirschfeld, in 4,359 autopsies, found chronic pulmonary tuberculosis in 21 per cent., and in 107 autopsies of valvular heart disease, it was present in but 2.8 per cent. Potain, in 54 cases of mitral stenosis, found pulmonary tuberculosis in 16.6 per cent. (see VALVULAR DISEASE OF THE HEART). (6) Syphilis, pleurisy (*q. v.*), and hæmoptysis (*q. v.*), are occasional predisposing factors.

General Histo-Pathology of the Tubercle.—The only absolutely pathognomonic feature of the tubercle is Koch's bacillus, as similar infective granulomata are seen in strongylus in sheep, in actinomycosis, and in syphilis. Baumgarten described the changes following inoculation of the anterior chamber of the eye: (*a*) On the first day the bacilli increase and spread somewhat along the lymph-spaces. (*b*) By the sixth day, the fixed connective-tissue cells proliferate under the stimulus of the tubercle bacillus, which acts as a foreign body; these cells, known as epithelioid cells, are large, rounded or polygonal cells with distended nuclei, and sometimes contain tubercle bacilli; in another day or two they show karyokinetic figures; the bacilli multiply and the endothelial cells of the vessels proliferate. The tubercle of this stage is sometimes called the epithelioid tubercle. (*c*) On the tenth or eleventh day the leukocytes emigrate. This inflammatory reaction results from the toxins secreted by the bacillus; at first most of the white cells are polymorphonuclear, many of which degenerate; later they are mononuclear, which do not suffer retrogressive changes; the mononuclears crowd the epithelioid cells and dominate the histological picture, especially with complicating trauma (lymphoid tubercle). (*d*) The reticulum of the tubercle consists of the stretched fibers of the infected tissue or of coagulation necrosis. (*e*) The epithelioid cells increase in size and are often multinucleated. From them or from the extravasated leukocytes, by growth, or by fusion, giant cells develop; they contain many large, oval, vesicular nuclei and tubercle bacilli; the bacilli may number 50 to a cell in very active processes but are few in chronic tuberculosis; the giant cells are particularly abundant or may even be the chief histological finding in chronic forms, such as lupus, scrofulous glands or fungoid joint-disease; they are few or absent in acute processes, as in miliary tuberculosis; there seems to be a certain antagonism between the nuclei and bacilli, for if the nuclei lie on the margin of the cell, the bacilli are centrally grouped, and if the nuclei lie at one pole of the cell, the bacilli concentrate at the other pole. The cellular inclusion of the bacilli is considered a phagocytosis by Metschnikoff, which Baumgarten, however, denies. The completed miliary tubercle is non-vascular, cellular, millet-seed (miliary) in size, gray in color, and translucent. It is the pathological unit of all tuberculous lesions, however extensive. In the diffused inflammatory tubercle, the diffuse areas result from fusion of myriads of miliary tubercles (see CASEOUS PNEUMONIA).

The tubercle then undergoes one of two *degenerations*, caseation or induration: (1) *Caseation* results from degeneration of the tubercle, be-

PLATE V.



Retinal Findings in the More Important Diseases.

1. Retinitis diabetica. 2. Atrophy (and pigmentation) of retina and optic nerve. 3. Choked disk in brain tumor. 4. Optic neuritis in meningitis. 5. Medullated nerve fibers (normal).

ginning at its centre, from ischæmia (vascular obliteration) and from coagulation necrosis (action of the bacilli and its toxins). The area of coagulation necrosis then gradually becomes more yellow and granular, and finally results in an amorphous, homogeneous, cheesy debris. Caseation is a dangerous, destructive degeneration. (2) *Induration,* sclerosis or encapsulation, results from growth of connective tissue around the tubercle or conglomeration of tubercles; it limits the tubercle, is the measure of the resisting power of the surrounding tissue, and is a conservative process. It occurs most frequently in the peritoneum, but also in the lungs and other tissues. *Inflammation,* varying with the tissue involved, may cause other histological findings. Suppuration may result from mixed infection with streptococci, though Koch held that pus may be formed by the tubercle bacillus itself; the ordinary tuberculous "cold abscess" is not really pus, but merely thick, amorphous, granular debris.

Extension.—(1) Tubercles develop at the point of infection and also in the nearest lymph-glands, which permanently or temporarily arrest the process. (2) Infection may occur through the uninjured mucosa or skin, but the process rarely passes beyond the tributary lymph-glands. (3) The later advance of the process occurs interruptedly or gradually, so that it can be traced clearly at autopsy. (4) Extension usually occurs along the lymph vessels and glands.

I. Tuberculosis Miliaris Acuta.

Definition.—An acute general, sometimes almost universal, embolic dissemination of the tubercle bacillus in great numbers, by the blood or lymph stream. It results pathologically in the formation of miliary tubercles, which are wide-spread in various tissues, and it produces, clinically, acute toxæmia, pulmonary, meningitic, or other symptoms, and is invariably fatal in a few weeks.

Etiology.—**1. Development.**—Acute miliary tuberculosis *always develops from a tuberculous focus.* This is found in direct ratio to the carefulness of dissection; von Buhl, who first fully described the affection (1856–1858), recognized that miliary dissemination occurred by absorption from old tuberculous foci, just as pyæmia results from local suppuration; and he found the primary focus in 90 per cent. of cases. It may be small or even microscopic and may easily escape detection (Pizzini, Cornet, Spengler), but Simmonds found the initial lesion in 100 per cent. As Cornet remarks, it is curious that miliary tuberculosis occurs from foci so small as to be easily overlooked, and that large tuberculous lesions so seldom result in miliary dissemination. A description of all primary foci would be an enumeration of every possible localization of tuberculosis.

2. Lesion.—*Pulmonary tuberculosis* is the most frequent lesion, and causes over 50 per cent. (Ruehle, Orth, Litten) of the cases of acute miliary tuberculosis (which occurs in 3 per cent. of the cases of pul monary tuberculosis). Glandular lesions rank next, and articular, osseous, genito-urinary, and serous-membrane tuberculosis, cause most of the balance. The more the tubercle is indurated, the less is the danger of local or general dissemination.

3. Mode of Access of Bacilli into Blood Stream.—Sir Astley Cooper, in 1798, found tubercles in the thoracic duct, and this was later confirmed by Ponfick, Weigert, Koch, and Meisels. In 1877, Weigert demonstrated that tubercles invade the vessel walls, which Rokitansky had declared to be immune. Tuberculosis reaching a vessel usually causes its gradual obliteration and in this way keeps the process local. In some cases bacilli reach the lumen of the vessel and are distributed in the direction of its current. Two views are held as to the nature of the involvement of the vessels: (*a*) that of Weigert, who thought the most frequent process was periangitis, by which, for instance, a caseated gland became adherent to and emptied into a vein; and (*b*) that of Benda, who contended that the common lesion was endangitis, by which tubercle bacilli lodged in the vessel's intima, and, grouping there, further disseminated the infection. One of the most common methods is periadenitis of a tuberculous bronchial gland, its adhesion to a pulmonary vein, and final rupture into it. Tuberculous, lesions may rupture into the vena azygos, pulmonary artery, aorta, thoracic duct, jugular, thyroid, supra-renal, or renal veins, into the cavæ, or into the brain sinuses. From the pleuræ and peritoneum infection may travel directly to the thoracic duct.

4. Organs Involved.—(*a*) Rupture into a pulmonary vein floods the arterial circulation with bacilli, whence miliary deposits occur in the brain, choroid, liver, spleen, kidneys, thymus, thyroid, heart (myocardium near pulmonary artery), bones, serous and mucous membranes, and in the blood-vessels themselves. (*b*) Rupture into a portal venous radicle affects chiefly the liver. (*c*) Into the pulmonary artery, the lungs, and (*d*) into a peripheral artery, the tissue supplied by it.

5. Degree of Lesion.—Acute generalization only follows the introduction of large numbers of bacilli, for they do not multiply in the blood. A few bacilli reaching the circulation cause a solitary tubercle in some organ—for example, in the brain. Repeated ruptures of moderate numbers of bacilli lead to the so-called chronic miliary tuberculosis (*v. i.*).

6. Predisposing Factors.—Though miliary dissemination may occur at any age, it is most frequent in children at the developmental period, especially between one and four years of age, because the circulation is faster, metabolism is quicker, thrombosis of the vessels is less frequent, tissue reaction is less marked, and the lymph-vessels and spaces are wider. Depleting factors, pregnancy, the puerperium, trauma, bone operations, measles, pertussis, mixed infections, and the spring and summer months, are somewhat predisposing causes.

Symptoms.—The clinical picture varies greatly, depending on (*a*) the quality (virulence) and quantity of the bacilli; (*b*) the toxins thrown into the circulation from the primary ruptured focus; (*c*) the toxins developed elsewhere by the bacilli; and (*d*) the localization of the tubercles formed. Naturally toxæmic symptoms precede local symptoms.

.1. **Temperature.**—Continuous fever may be observed in very acute cases with wide-spread localization, which do not involve the brain; as a rule it is lower than that of typhoid. Remittent or intermittent fever is more common; miliary tuberculosis following pulmonary phthisis may change neither the hectic fever of the latter nor its euphoria. The *typus inversus* may occur, though it usually lasts but a few days (Bruniche). In 17.5 per cent. of cases (Rheinhold) the course is afebrile; involvement of the brain or meninges lowers the temperature or renders it variable; hemorrhages, vomiting, or diarrhœa, may depress the fever (Litten); low temperatures are common in the aged, in the ambulant, and in bronchitic or cardiac types.

2. **Circulation.**—(*a*) The pulse is faster than is proportionate to the degree of fever, being 120 in the early stages and 140 to 160 later; the blood-pressure is low but dicrotism is infrequent; a pulse under 100 is very rare and nearly always occurs in old people and in cases of meningitic localization. (*b*) The heart is somewhat dilated and an apical systolic murmur, with accentuation of the second pulmonic sound, is fairly frequent. (*c*) A pericardial rub is heard on rare occasions, and is due to roughening of the serosa by the miliary eruption (Litten). (*d*) Tubercle bacilli were first found at autopsy in the blood by Weischselbaum and Meisels, and clinically by Lustig and Ruetimeyer, Sticker, Ulacacis, and Doutrelepoint. In the death agony from lung tuberculosis, bacilli may enter the blood. (*e*) The leukocytes are usually not increased.

3. **Respiration.**—Early symptoms are frequent. (*a*) There is very often a severe *continuous cough*, which is always a suspicious symptom. It is due to the toxins or to irritation of the vagus twigs by abundant miliary nodes in the lung; it causes considerable muscular pain, but little voiding of sputum and ceases later if the brain becomes involved. (*b*) The *breathing* is *increased* to 40 or 60 in adults, and to 80 or 90 in children. (*c*) *Dyspnœa* and *cyanosis* develop from irritation of the vagus (Ruehle) or from diaphragmatic involvement (Rheinhold). The tachypnœa, dyspnœa, and cyanosis, are peculiarily significant diagnostically in that they have no adequate explanation in the physical findings. (*d*) The sputum is scant, foamy white, and mucous, in acute cases. It seldom contains dots of blood, and is rarely rusty without the presence of complicating pneumonia. It may be more abundant in subacute cases. (*e*) The physical findings are not as marked as the urgent respiratory symptoms would indicate. A *tympanitic note* is very frequent, resulting from relaxation of the lung, or from acute emphysema from coughing. Dulness only results from older tuberculous foci in the lung, or from complicating atelectasis or consolidation. Auscultation often reveals *fine rales*, which are especially suggestive when they occur over the upper chest, where stasis is

uncommon. Juergensen notes a fine pleural rub, which is due, he thinks, to pleural tubercles. A terminal pulmonary œdema is frequent. At autopsy innumerable translucent nodes are scattered through the lung, and are most apparent on oblique illumination.

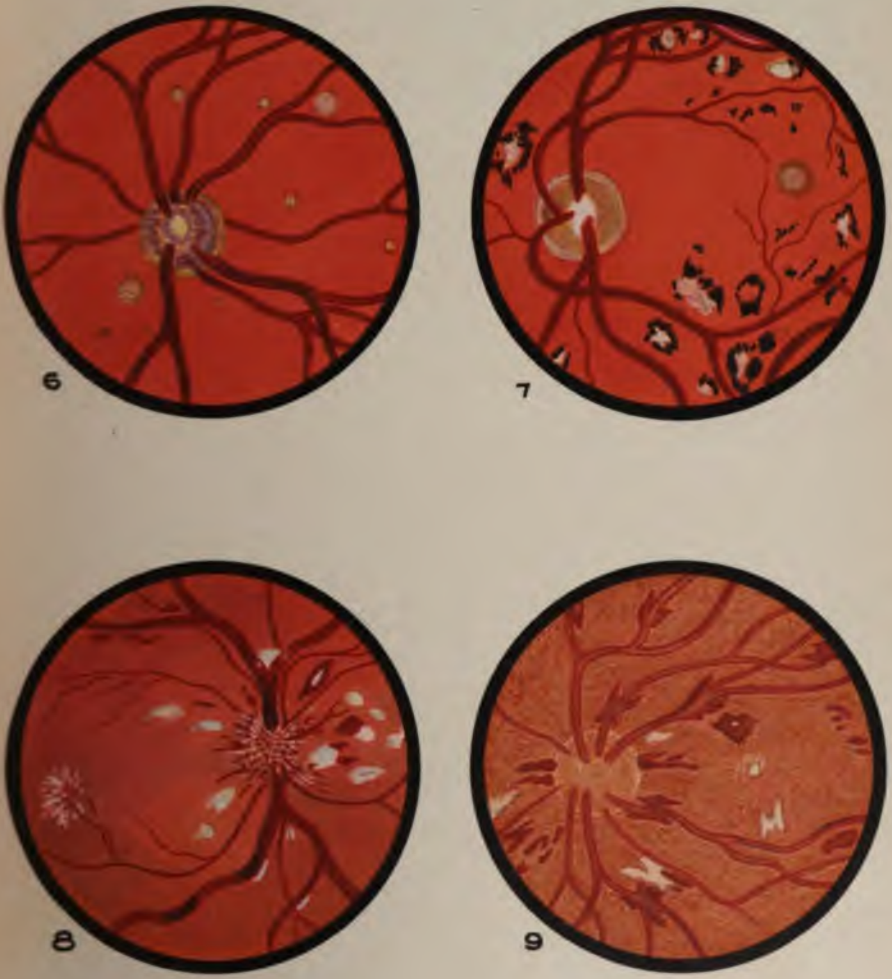
4. Nervous System.—Nervous symptoms may predominate, and may result from toxæmia (like the symptoms following tuberculin injection), or from central organic changes. (*a*) The toxæmic symptoms are headache, dulness, vertigo, and tinnitus; these are followed by delirium, muscular adynamia and tremor, and result in sopor, which is broken by fits of delirium, and finally in coma. In rare instances coma may develop early in an apoplectiform fashion, or the sensorium may be normal. (*b*) *Meningitic symptoms* (see DISEASES OF MENINGES) may develop, such as headache, variable pulse and respiration, tenderness of the spine with retraction of head and spinal rigidity, narrow and then widened pupils, strabismus, paralysis of other cranial nerves, Kernig's sign, clonic contractions, convulsions, and coma. The findings at autopsy are essentially those of tuberculous meningitis(*v.* MENINGITIS).

5. Digestive System.—The tongue is febrile and there is anorexia. Nausea and vomiting are usually meningitic. There is usually constipation and the abdomen is normal. Diarrhœa is infrequent, but bloody stools may occur with tympany and without ulceration (Sée, Rheinhold). Litten once heard a friction rub over the liver. Ascites is infrequent in miliary tuberculosis of the peritoneum (as distinguished from tuberculous peritonitis). Miliary tubercles may occur in the stomach and bowels, mostly in children.

6. Choroidal Tubercles.—These were first found at autopsy by Nanz (1857), Nusch and Cohnheim, and clinically by von Graefe (1857) and Leber. They are said to occur in 16 per cent. (Rheinhold), 22 per cent. (Demme), and 75 per cent. (Litten) of the cases. They are most likely to be found when the tuberculosis is very general. They occur singly or in large numbers (52, Cohnheim), usually in both eyes as whitish-yellow protuberances with irregular washed borders. They become quite large, for they cause so much atrophy of the retinal pigment layer which covers them that they can be seen through it. Daily examination for them is necessary, as they frequently develop in half a day. They are seen only after one or several weeks. (Tubercles of the choroid are depicted in PLATE VI, Fig. 6.)

7. Other Symptoms.—Emaciation is often pronounced; one of Wunderlich's cases was reduced to one-quarter of his ordinary weight. The *spleen is enlarged* in 70 per cent. (Litten), but is usually smaller than it is in typhoid, except in children, where it may assume great dimensions. Collin observed swelling of the lymphatic glands, which in one instance became enormous. The urine is concentrated and dark, from the fever and from sweating, may contain albumin or peptone, and quite often shows the diazo reaction. Rosenstein observed anuria, especially in children. Hemorrhages into the gums, nose, retinæ, and intestines, are infrequent and possibly result from mixed

PLATE VI.

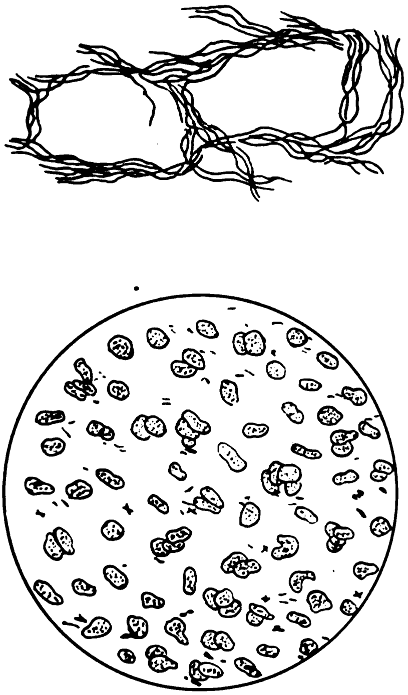


6. Tubercles in choroid. 7. Syphilitic choroidoretinitis. 8. Albuminuric retinitis. 9. Hemorrhages in pernicious anæmia. (Figures 1 to 3 are from Jaeger and the balance are from Sahli.)

tains sago-like bodies which are composed of alveolar cells transformed into a myelin substance. It later becomes yellow from admixture of pus, or yellow or red from saprophytic organisms, such as sarcinæ, or the bacillus aureus or squamosus. A green tinge may result from the bacillus pyocyaneus or fluorescens. Blood may give it a red, rusty, flesh-like, or chocolate hue. It tastes sweet and smells slightly fœtid. Its amount varies from a few globules to over a pint. It is more abundant in cavities than it is in extension of tuberculosis by the blood- or lymph-vessels; it stagnates before death, because of the patient's muscular weakness; women and children are prone to swallow the sputum. Nummular sputum from cavities is flat, "coin-like," greenish-gray, extremely abundant, and sinks in water, as it is airless; it occurs in other than tuberculous cavities, as in bronchiectasis. The *only characteristic* of the sputum is the *tubercle bacillus* (*v. s.*), which is found only in tuberculosis; it may be seen before the advent of any physical finding, but in other cases it must be sought repeatedly, as it appears only when ulceration communicating with a bronchus, develops. The tubercle bacillus is found in only one-third of the cases of incipient tuberculosis. Other bacteria and saphrophytes are frequent (*v.* BACTERIOLOGY, 8). If the patient expectorates into water a crumbling precipitate, rich in bacilli, is obtained (Virchow). *Elastic fibers* are found in 70 to 90 per cent. and originate from the vessels, or from the parenchyma of the lung. The elastic fibers from the vessels are elongated and twisted, and sometimes considerable shreds of the vessel-wall are seen; the elastic fibers from the bronchi are also long and twining; those from the lung substance often show its alveolar arrangement. Elastic fibers can often be located by flattening the sputum between two plates of glass and picking out the yellowish areas for microscopic examination. Treatment with caustic potash, and centrifuging, facilitate their detection when they are scanty. They merely denote a destructive process, hence are also found in abscess, gangrene, infarction, and in pneumonia. Detritus, pus cells, red cells, alveolar, bronchial and buccal epithelium, myelin droplets and pigment, are also found. The so-called calculi of the lung are sometimes voided; they are calcified particles from old areas of caseation, from bronchial obstruction, or from peribronchial lymph glands; from the glands large calcarious masses may cause fatal obstruction of the trachea, or suffocation may be averted only by immediate low tracheotomy. Chemistry: 88 to 98 per cent. of the sputum is water; 3 to 11 per cent. is organic, and less than 1 per cent. is inorganic matter; tyrosin, glycogen, urea, lecithin, cholesterin, fatty acids, nuclein, and a pepsin-like ferment, may be found. (See Plate VII for tubercle bacilli and elastic fibers).

3. HÆMOPTYSIS.—Hæmoptysis (hæmoptoë, spitting of blood) occurs in a variable percentage. Prior saw it in 37 per cent. of 1,000 cases; others place it at 66, 80, or even 90, per cent.; it is more frequent in adult males than in females and is rare before puberty. (*a*) *Early hæmoptysis* is due to erosion of the mucous membrane, or often erosion of a small vessel by tubercles in its perivascular lymph sheath. The

PLATE VII.



Tubercle Bacilli and Elastic Fibers.

toxins usually prevent extensive early hemorrhage by causing coagulation in the vessels in and near the affected lung area. Hæmoptysis is the first symptom in 4 per cent. of phthisis cases and is fairly often (16 per cent.) the only symptom, the patient never having future trouble; in most cases, however, other symptoms develop; bacilli and elastic fibers may be seen at the time of bleeding. Reiche recently corroborated the old statement that early hæmoptysis is not unfavorable. The bleeding often occurs without cause, coming on suddenly with a warm, salty taste in the mouth and the evacuation of a dram or more of bright frothy blood, either clear or mixed with mucus; small particles of darker blood are brought up, for a few days, after which it disappears; in 4,125 of the Brompton Hospital cases, 69 per cent. evacuated less than half an ounce. In other instances hæmoptysis is excited by coughing, laughing, bathing, exertion, or excitement. Bubbling rales may be heard on the side of the lesion, and moulds of the bronchioles may be voided. F. Stricker collated 900 cases of hæmoptysis in the German army among 1,728,505 soldiers, between 1890 and 1895; in 480 cases there was no cause, and of these 87 per cent. were tuberculous; of 43 cases brought on by exertion, 74 per cent. were tuberculous; and of 24 cases due to trauma, half were tuberculous. The occurrence of early hemorrhage, before other symptoms of phthisis, led Hippocrates, Boerhaave, Van Swieten, Morton, Graves, Niemeyer, and others, to think that hæmoptysis caused phthisis, while Laennec, Louis and Traube, correctly regarded it as a symptom of already established phthisis. The earlier idea is explained by the fact that in bleeding, blood and bacilli may be aspirated into other parts, in which the process develops anew (*v.i.* Acute Forms); fever following hæmoptysis is therefore rather ominous. (*b*) *Late hæmoptysis* may be slight or profuse. Profuse bleeding is usually due to erosion of bloodvessels running across, or in, the walls of cavities, which are frequently aneurysmatic (Rasmussen); occasionally larger vessels, such as the pulmonary artery, are ulcerated. Amounts as large as a pint or a quart may sometimes spurt out of the mouth, causing acute anæmia, suffocation, or rapid exsanguination. Blood may accumulate in large cavities, never reaching the air passages. While hæmoptysis is usually a sign of tuberculosis, it may occur from other causes (*v.* Diseases of the Lungs).

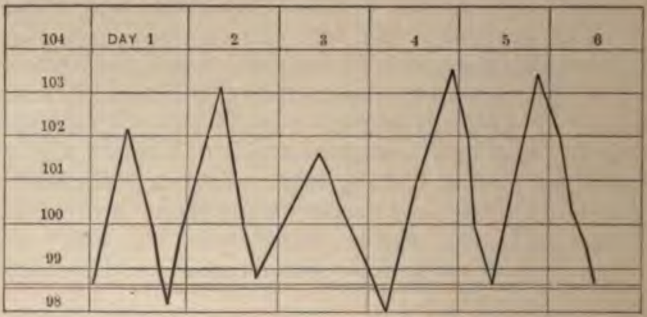
4. Dyspnœa.—This is infrequent, even when the respiratory area is enormously contracted, just as it is absent in pleurisy of gradual onset; it is often absent even with great infiltration, great cavity formation, or induration of two-thirds of the respiratory space, whereas in sudden miliary tuberculosis the dyspnœa is great. In some cases dyspnœa is neurotic, or it may follow pressure of swollen glands on the vagus, anæmia, stagnating secretion, extensive vicarious emphysema, or massive pleural adhesions. It is most often due to the rapid heart action. In the last stages it may cause orthopnœa and can result from intercurrent bronchitis, pneumonia, or pneumothorax. Germain Sée has described a tuberculous pseudoasthma.

5. Pain.—Pain is moderately frequent. Its location may be apical, basal, diaphragmatic, or on the anterior part of the chest from the clavicle to the third rib or under the scapula. Its character is usually sticking and intermittent, but not intense; the skin is often hyperæsthetic to pressure and palpation. Its cause is chiefly acute pleurisy, for lung disease causes no pain, and chronic pleurisy is generally painless; other causes are muscular fatigue from coughing, intercostal neuralgia, and less often intercostal neuritis. Pain may be intense in pneumothorax.

(*B*). **General Symptoms.**—These result from absorption of the *toxins* secreted in the local lesions.

1. Fever.—This is present in the great majority of cases and is almost invariable in the early stages. Its cause is somewhat disputed; probably the dominant factor is the tuberculous toxin, whose febrile action is analogous to that of tuberculin; secondary infections undoubtedly may cause fever, though not necessarily, for it is proven that the associated streptococci are usually far from virulent. Fever marks the tubercle formation, fusion of foci, advance of the disease, degeneration of the tubercle, and probably secondary infection; its cessation is synchronous with temporary or permanent arrest of the disease as manifested by improved health, strength, and weight. Its height and course are somewhat influenced by the individual; labile individuals possess labile temperature centres (de Renzi); fever cannot be classified by the clinical stages, as incipient fever, ulceration fever, etc.

Fig. 20.



Hectic-fever curve in phthisis.

Types.—Intermittent fever may exactly resemble malaria and can be detected only when registered every two or three hours; remittent fever most often occurs late in the clinical course; continuous fever occurs early or in acute consumption, though it rarely resembles the typhoid curve; in some cases the fever falls from high to subnormal registers, sometimes with collapse, which was regarded as unfavorable by Ruehle; the *typus inversus* with morning exacerbation and vesperal solution is very uncommon. Fever may be absent in cases of old cavities and of fibrous induration. Chills have no relation to the fever nor to its intensity. As fever is indicative of toxæmia, it is often associated with anorexia, emaciation, and other symptoms to be immediately described.

2. THE BLOOD.—(*a*) The red cells rarely number less than 2 million, though Limbeck records a case with but 730,000; the anæmia may antedate all other symptoms, whence the frequent confusion of chloranæmia and incipient phthisis. Again, anæmia may be inconspicuous or late. In some cases the red cells are increased, notably in individuals prone to hæmoptysis (Mircoli). (*b*) The hæmoglobin may fall to 25 per cent., but averages 70 to 80 per cent.; it may be decreased more than the number of red cells, therein resembling chlorosis (*chloranæmie tuberculeuse*). (*c*) The leukocytes may be normal; again, in cavity formation (secondary infection) their number may rise to 12,000 or 15,000; they are increased in cases of caseous pneumonia or protracted pyrexia; after injection of tuberculin the eosinophiles show a decided increase. (*d*) Tubercle bacilli are seldom found in the blood except in the death agony.

3. SWEATING.—This is more important than in any other disease. It occurs in 90 per cent. of cases (Louis). The most likely cause is the action of toxæmia on the sweat-centre in the medulla, though there are many other theories. Heavy coverings may sometimes cause sweating. It is more frequent in acute than in chronic phthisis and in younger than in older subjects. The term "night sweats" is used because nearly all of them occur in the night, chiefly between 2 and 5 A.M. They are most frequent when cavities are forming, and in the last stages. They have no constant relation to the fever but are more frequent when it is variable.

4. PULSE.—The pulse is rapid, digestion is frequently disturbed, muscular strength is reduced, and nutrition is depreciated (*v.i.* COMPLICATIONS, for details).

(C). **Physical Signs**—In adults, the apex of an upper lobe, more often the right than the left, is first affected (Laennec). The reasons ascribed are various, such as a poor apex blood supply, slower lymph current, and less expansion,—which are scarcely proven. It is more correctly held that coughing drives infection upward from the bronchi, and that the clavicle or first rib compresses the bronchi passing to the apex. The summit of the apex is not invaded first. Birch-Hirschfeld, in early cases which died from causes other than tuberculosis, found that in 24 per cent. the disease began in the fourth or fifth order of bronchi in the upper lobe and that the posterior were affected earlier than the anterior divisions.

1. INSPECTION.—(*a*) The nutrition may be good, but, later, emaciation is marked. The face is pale, the cheeks hectic, the nose pointed, the eyes hollowed and glistening, the neck thin, and the chest is altered in various ways; in some cases it is long and narrow, with wide interspaces, sharp costal angles, prominence of Louis' angle, and more vertically directed ribs; in others it is flattened anteroposteriorly and the ribs are closely apposed; in still others the funnel-chest may be

seen. Laennec recognized that the "paralytic chest" was the result and not the cause of phthisis. Examining the patient in a good light, the following may be noted: (*b*) *deficient expansion* above or below the clavicles, especially when examined from behind and above; in the lower chest, an impaired or absent excursion of the diaphragm (Litten's phenomenon). Deficient expansion may be due to pleural adhesions, to atelectasis of the lung resulting from caseous nodes obstructing the bronchi, to a cavity, or to gradual induration of the focus, which is an effort towards encapsulation and recovery. (*c*) Distention of the chest may be due to vicarious emphysema, effusive pleurisy or pneumothorax. (*d*) The cardiac impulse may be increased by retraction of the left lung away from the heart.

2. PALPATION.—Palpation shows (*a*) *decreased expansion,* elicited by standing behind the patient with the thumbs above either clavicle and the fingers below it, or by standing in front of the patient and applying the spread fingers and hands over the anterolateral parts of the thorax; (*b*) *increased vocal fremitus,* obtained when the lung is infiltrated (caseous foci or pneumonia), indurated or is the seat of cavities, all of which increase conduction of the voice-sounds from the bronchi. The depth of the focus is a factor, superficial lesions showing more clearly than deep ones; thick pleural adhesions or plugging of the bronchi with caseous material decrease the fremitus. (*c*) The systolic distention of the pulmonary artery and the second pulmonic closure are better felt when the lung retracts from the heart or when it is infiltrated in its vicinity.

3. PERCUSSION.—(*a*) Incipient infiltration of the apex gives a short percussion note, which later becomes *dull.* According to Grawitz the apex is first involved in 88 per cent. of cases. Dulness is detected in the apex or under the middle of the clavicle, at which level Birch-Hirschfeld found the anatomical beginning of tuberculosis. The apices are best percussed when standing behind the patient, and it should be remembered that the right apex is normally less resonant than the left. The lung tissue lying behind, or near, the clavicle can be well tested by gently tapping the clavicle with one finger. The apex reaches 1 to 2 inches above the clavicle, and behind to the *vertebra prominens.* Piorry, Friedreich, and Eichhorst, demonstrated that impaired resonance is produced only by consolidation measuring 1½ by 2½ inches in area and within ¾ inch of the surface, and that foci of these dimensions lying deeper than 2 inches escape detection. Oesterreich holds that a focus as large as a cherry may cause dulness. Small multiple or deep foci give a tympanitic note. To detect slight early alterations in note, percussion should be gentle, symmetrical parts should be compared and the patient should hold his breath in full inspiration; the muscles must be relaxed, and it should be recalled that each patient has his own percussion note, which varies with the individual's muscles, fat, and bones. Often the first dulness of consolidation is found behind, in the supra- or interscapular region. The dulness may extend from the clavicle to the second and third costal

cartilages, then to 1.5 inches below the apex of the lower lobe at the level of the fifth dorsal vertebra; this area is almost always involved when the upper lobe shows distinct consolidation; extension occurs outward and downward along the interlobar fissure; then it extends to the apex of the upper lobe on the other side. (*b*) Shrinking, or fibrous induration, and a thickened pleura, give apical or other dulness. Sometimes the lung becomes vicariously emphysematous about an old focus which is thus hidden clinically and pathologically. (*c*) A tympanitic, or cracked-pot resonance, may be obtained over cavities (*v. i.*); percussion over a cavity sometimes induces sudden evacuation of its contents.

4. AUSCULTATION.—This is the most important aid, next to the microscopic examination of the sputum. (*a*) The vesicular murmur, especially at the apex, is altered,—for detection of which the patient is best examined without deep breathing or coughing; the jerky "*cog-wheel" breathing* may be due to small tubercles in the pleura or bronchi, somewhat stenosing their lumina; it is only suggestive and occurs in normal individuals; Potain considers it due to pounding of the heart on the lung during inspiration (also a cause for the cardiorespiratory systolic whiff, which is heard in the front, sides, and back, of the chest during deep inspiration, in healthy as well as tuberculous subjects). The breathing may be *weak, indeterminate*, or *puerile*. A rude, rough inspiration may be heard, which is due to peribronchial tubercles compressing the bronchioles. The *expiration* is frequently *prolonged, rougher*, and *higher pitched*, in apical "catarrh"; it is long, sharp, and bronchial, in older foci. It must be remembered that the breath-sounds are physiologically higher pitched on the right than on the left side, and that what seems clinically to be catarrh is often consolidation, pathologically. (*b*) *Bronchial breathing* is caused by various consolidations (tuberculous, pneumonic, neoplastic, or gangrenous infiltration) and by cavities, tuberculous and otherwise. Bronchial breathing of moderate degree is physiological in the interscapular region, but is considerably less intense than that of consolidation. (*c*) *Rales* are rather an evidence of infiltration (consolidation) than of "catarrh". If they are heard in the apex, the presence of tuberculosis should be suspected, though they may occasionally occur there physiologically. The vibrations of the accessory muscles of respiration, when the patient takes a long forcible breath, may be confused with rales, but are easily differentiated after a little experience. Rales may not be heard unless the patient breathes deeply or coughs, and auscultation should be made before, and then after, coughing and deep inspiration. In incipient consolidation, small crackling crepitant rales are heard over the alveoli and smaller bronchioles, at the end of inspiration; they later become larger, ringing and resonant, which proves that the alveoli and bronchioles have become airless over a considerable area, or that softening or formation of cavities is in progress; scattered, clear, large, crackling rales are heard over small cavities in the apex or in the smaller bronchi, and indicate a viscid secretion. Clicking rales

near the heart may occur in the systole, as the heart beats upon an adjacent infiltration. In emphysematous patients, whose chests exhibit sibilant and bubbling rales, auscultation and diagnosis of developing tuberculosis may be most difficult. (*d*) *Bronchophony* (a bronchial quality imparted to the voice-sounds) is heard when the patient counts or speaks during auscultation; it indicates infiltration or cavity formation but is absent when the bronchi are plugged with secretion. *Ægophony* is merely a higher grade of bronchophony and occurs, as Skoda demonstrated, in cases of consolidation, in middle-sized cavities, and above pleural exudates and transudates. (*e*) *Pleural friction* may result from rough tubercles in the pleura, or from fibrinous pleurisy. (*f*) A systolic bruit over the subclavian artery is probably due to pleural adhesions to the artery.

5. ROENTGEN RAYS.—The rays show shadows which may be due to consolidation, thick-walled cavities, calcified foci, gangrene, pleural callus, or pleural exudate. The excursion of the diaphragm can be followed; shadows toward the apices, due to the muscles, may be readily eliminated, by moving the arms; the results are often confusing.

The findings of inspection, palpation, percussion and auscultation are grouped together, because they are found together clinically, one part showing the signs of consolidation, another those of cavity formation or of induration. To summarize, (*a*) the *signs of consolidation* are decreased expansion, increased vocal fremitus, initial tympany followed by dulness, high-pitched expiration, then distinct bronchial breathing, crepitant rales and bronchophony; (*b*) the *signs of induration* are retraction, or lagging behind of the fibrous area on breathing, pleural adhesions, increased vocal fremitus, dulness, usually large rales, bronchial breathing and bronchophony. (*c*) The *signs of cavity formation* are: (i) increased vocal fremitus, if it is near the surface; (ii) tympanitic note, which may increase in intensity when the cavity is percussed with the mouth open (Wintrich's change of note; it is also observed in cases of consolidation lying over a bronchus), when it is percussed during inspiration (Friedreich's change of note), or when it is percussed with the patient in the sitting posture (Gerhardt's change); the note may be metallic; the cracked-pot note, Laennec's *bruit de pot fêlé*, is obtained in cases of superficial cavities measuring 2½ inches with thin walls, which communicate with an open bronchus by a small opening; variation in the percussion note from accumulation (dulness), or emptying of secretion (tympany), is most suggestive of cavity formation; (iii) the breathing, which is loudly bronchial, or, if the cavity is quite large, is metallic, amphoric or metamorphosing; metamorphosing breathing is breathing which begins normally and suddenly becomes bronchial (Seitz); (iv) bronchophony and ægophony; (v) large, resonant rales, which are the most frequent and important finding; metallic rales in the apices are most suggestive of cavities; they may acquire an amphoric character in very large, thin-walled excavations; (vi) nummular sputum; (vii) systolic murmurs, which are sometimes booming or metallic, and are due to propagation of the heart tones; they are occasionally heard at some distance from the patient.

Cavities are due to the breaking down of caseous areas and are usually connected with the bronchi. Their evacuation into the bronchi is partly favorable, in that the infective material is evacuated; partly unfavorable, because it opens up avenues for infection by aspiration. They rarely heal by obliteration; they may leave fistulous cicatrices (Laennec's *cicatrices fistuleuses*); if they are held by adhesions the cavity remains, contracting cicatrization being impossible; there is always danger of their further extension by secondary infection, or of gangrene. The cavity has a well-marked limiting membrane, from which pus, nummular sputum, elastic fibers, and grayish granular detritus composed of masses of tubercle bacilli, are voided; the cavities are frequently multilocular, may heal on one side and extend on another, and sometimes occupy an entire lobe or even an entire lung. They may rupture into the pleura, pericardium, or bloodvessels; in one instance the author saw a cavity rupture into the mammary gland, resulting in diffuse subcutaneous emphysema.

Stages of Pulmonary Tuberculosis.—Tuberculosis usually develops for six to eighteen months before any symptoms appear.

1. **Phthisis Incipiens.**—In incipient phthisis the general and local symptoms appear as above described, with fever, anæmia, and rapid pulse. The modes of onset are various, for tuberculosis may simulate chlorosis, malaria, dyspepsia, laryngitis, or bronchitis, or may appear in other forms more directly suggestive of tuberculosis, such as hæmoptysis, pleurisy, or cervical adenitis. The physical findings are uncertain or absent.

2. **Phthisis Confirmata.**—The appearance of distinct physical findings indicate not incipient phthisis, but confirmed established phthisis.

3. **Phthisis Consummata.**—This is advanced consumption, with marked hectic fever, cavity formation, and emaciation.

Course.—The average case of "chronic ulcerative phthisis," with the symptoms and signs already described, stands midway between the following very acute and extremely chronic groups:

(*A*). **Acute, Galloping or Florid Consumption.**—This type probably always develops from some old focus, such as tuberculosis of the bronchial glands, or from a small cavity which by rupture disseminates the process; possibly the virulence of the germ is greater in these cases; pregnancy, diabetes, alcoholism, measles, whooping-cough, influenza, or trauma, may be the immediate cause.

1. ACUTE PNEUMONIC FORM.—The acute pneumonic form is acute caseous pneumonia. The terms are employed because of the great clinical resemblance to, and confusion with, acute lobar pneumonia; but it must be remembered that genuine lobar pneumonia does not caseate, and that this form is not synonymous with tuberculosis complicated by acute pneumococcic pneumonia, which, in this association, runs its usual course. Acute caseous pneumonia begins in an indi-

vidual in seemingly perfect health, or perhaps in one whose health is somewhat but not suggestively reduced. An initial rigor is fairly common, or the spitting of blood may be the first symptom. There may be cough, pain in the side, rapid breathing, and sudden fever, which are all apparently compatible with a diagnosis of genuine pneumonia. The fever, however, is remittent or intermittent, and more variable than in lobar pneumonia; dyspnœa and cyanosis are less frequent than a gradually increasing pallor; sweating is usually copious. In a few days dulness prevails over a lower lobe but much more often over an upper lobe or over an entire lung. The breathing, which is at first indistinct, may remain so (Traube), but quite often becomes bronchial and is associated with crepitant and coarse mucous rales. The sputum is often typically pneumonic, viscid and rusty, or again is of an olive or grass-green color (Traube). Tubercle bacilli are sometimes found, but in this case originate not from the fresh pseudopneumonic patches, but from the causal focus (the primary cavity). In some few cases the physical signs of the primary cavity in an upper lobe can be elicited. Albuminuria is uncommon; the diazo reaction is nearly always present. The physical findings change but little, though the crepitant rales decrease and are replaced by coarse mucous rales; the anticipated crisis does not occur, and most cases are still called unresolved pneumonia until the increasing anæmia, loss of strength or rapid pulse, suggests a tuberculous pneumonia. Of 13 cases reported by Fraenkel and Troje, 11 died within six weeks, and Traube observed a fatal issue in thirteen days. In some few cases considerable regression may occur and it is barely possible for connective tissue to develop, dragging out the course for three months or even for years. The findings at autopsy are most interesting; usually an old focus of tuberculosis is found, a ruptured cavity or gland; from it caseous material, containing bacilli and tuberculous toxins, is aspirated into many finer alveoli and bronchioles, where they set up a miliary caseous lobar pneumonia, depending on the size of the affected area only. The inflammation is seemingly due wholly to Koch's bacillus or its toxins, and is (*a*) partly fibrinous and desquamative, red and white polynuclear and mononuclear cells being poured out; proliferative changes are noted, as the epithelioid and giant cells which are seen in the typical tubercle; caseation ensues, as shown in the already fused and fusing yellowish-gray areas; (*b*) the most rapid exudation is serous, Laennec's "gelatinous or smooth pneumonia," which is probably due, as Fraenkel and Troje insist, to the toxins of the tubercle bacillus and not to mixed infection as Ortner and others maintain; this part of the inflammation may regress, and does not necessarily caseate. Cavities rarely form in this type.

2. ACUTE DISSEMINATED FORM.—Acute disseminated tuberculosis, sometimes characterized especially by hæmoptysis, may occur as a special acute type, or may complicate the ordinary form of phthisis. Following the rupture of a small centre of softening, which is usually located only at the postmortem, and following dissemination by aspira-

tion, there develop fever, repeated hæmoptysis, chills perhaps, rapid pulse, rapidly increasing dyspnœa, diffuse crepitant or bubbling rales, considerable delirium, or even a pronounced status typhosus. Hæmoptysis is absent in some cases. The sputum is absent or scanty, and the bacilli are sometimes absent throughout the course. Death may occur acutely in three to four weeks without a breaking down of the foci, or a remission may take place, after which there is breaking down of the indistinct foci which cause slight dulness and distant bronchial breathing. At autopsy, scattered, opaque, yellow, partly caseous peribronchial nodular foci are seen; in some cases the presence of tuberculosis is proven only by microscopic examination, for the foci may resemble the ordinary bronchopneumonic patches and may present no caseation. Secondary infection with streptococci is usually said to cause this form, but Prudden's experiments have demonstrated that the above lesions may result from the tubercle bacilli alone.

3. ACUTE DISSEMINATED ULCERATIVE FORM.—The acute disseminated ulcerative type is often observed in children between two and six years of age (Grancher), in pregnancy, alcoholism, diabetes, and influenza. The foci, which vary from the size of a pea to that of a walnut, are widely disseminated; they fuse, break down early, and develop cavities without walls, which are only ulcers or consist of necrotic caseous material. There are present clinically hectic or continuous fever, pronounced anæmia, exhausting sweats, rapid emaciation, and viscid sputum, which may be brownish but is more often purulent and contains many bacilli; diffuse fine and large rales and bronchial breathing may be heard. The course is galloping, and ends fatally in one to four months.

4. ACUTE MILIARY FORM.—Acute miliary tuberculosis (*q. v.*) develops in 3 per cent. of the cases of pulmonary phthisis.

(*B*). **Chronic Tuberculosis.**—1. CHRONIC ULCERATIVE FORM.—The ordinary chronic ulcerative phthisis may run a course which is long latent, except for anæmia, dyspepsia, or other undistinctive symptoms. Improvement may follow the detection of such positive lung findings as an apical catarrh, and the cough, fever, sweats, and anorexia, may subside; another relapse may occur after quiescence for years, or exacerbations may develop, with loss of weight and strength. Recovery may occur, even with pronounced physical signs. Cornet regards the breaking out after years of new tuberculous manifestations as reinfections rather than relapses.

2. PHTHISIS FIBROSA.—Phthisis fibrosa is the slow development of fibrous connective tissue excited by the tubercle bacillus. It may begin acutely or follow an insidious course from the onset. Its explanation is difficult; Auclair thinks the bacillus secretes two toxins, one with a necrosing action which leads to caseation, and another with an indurating action which leads to fibroid phthisis. There may be no cough, sputum, fever, emaciation, or anæmia. Bacilli may or may not be found in the sputum. Ulceration may result only from mixed

infection. Sometimes there is hæmoptysis. It may develop in an emphysematous chest (Sokolowski). If left-sided, the spleen rises higher and the retraction of the lung away from the heart makes its apex wider, the cardiac shock more diffuse, and the pulsation of the pulmonary trunk more visible. If right-sided, the liver rises, the right ventricle is apparently enlarged (from lung retraction) and is often actually hypertrophied and dilated from increased intrapulmonary tension. The heart may be drawn to the right or left by the inevitable pleural adhesions, bronchiectases are not uncommon, the sound lung is vicariously emphysematous, and the diseased lung is smaller, flatter, and depresses the shoulder, impairing expansion and often presenting bronchial breathing and large rales.

3. PLEURITIC FORM.—The pleuritic form (see PLEURISY) follows small latent foci in the lungs, lymph glands, and spine. Exudates not due to rheumatism, inflammation in contiguous organs, tumor, or nephritis, are always suggestive of tuberculous pleurisy. Pleurisy, in fact, more often precedes than succeeds pulmonary tuberculosis and repeated attacks are frequent, with moderate irregular or continuous fever, perhaps with rales in the apex of the affected side (not the rales frequently heard just above the upper level of the exudate), or with dulness in the contralateral apex. Bowditch, following up 90 cases of pleurisy, found that 33 per cent. developed pulmonary tuberculosis later in life.

Variations According to Age.—(*A*). **Children.**—Tubercle bacilli easily pass the bronchial mucosa and produce tuberculosis of the bronchial and mediastinal glands; the wide lymph-vessels and more active metabolism have already been noted. In children the process in the glands attacks secondarily the hilum of the lung and the lower lobes (80 to 98 per cent.). This invasion also explains the frequency with which the thoracic duct and bloodvessels are attacked and miliary dissemination results.

1. CHRONIC GENERALIZED TUBERCULOSIS.—The younger the child the less local and the more general is the tuberculosis. Measles, pertussis and influenza are very prone to arouse a latent tuberculosis in the bronchial glands. The course is generally less chronic than active, like that of tumors in children. Emaciation, anæmia, enlargement of the spleen and liver, moderate intumescence of such peripheral lymph-glands as the cervical, inguinal, and occipital, are observed. Fever may occur only toward the end of the process. Hectic fever is seen chiefly in children over three years. The sputum is swallowed, but washing the stomach recovers bacilli-laden sputum.

2. ACUTE MILIARY TUBERCULOSIS (*v. s.*).

3. LOCALIZED LUNG TUBERCULOSIS.—Localized lung tuberculosis may be (*a*) of the acute caseous type resembling pathologically bronchopneumonias of other causation, and the microscope may be necessary to detect its nature; hæmoptysis is rare, though Henoch has observed over a dozen cases under six years of age. (*b*) Chronic localized forms, like those of adults, are very infrequent under six or seven years of age.

In children, the physical findings are often absent; when present they are usually over the lower lobes, very seldom near the clavicles. In half the cases the wrong diagnosis is made (Henoch). Percussion is more difficult than in adults, because of the small chest and its thinner walls; for example a cracked-pot resonance can be obtained in a normal young chest by strong percussion. Children, too, breathe irregularly, and puerile respiration is physiological. Induration is rare.

(*B*). **The Aged.**—In the aged, hæmoptysis is rare, physical findings are less frequent than in adolescence, because the breathing is weaker, and emphysema is more common. Cough and fever are also less conspicuous.

Diagnosis of Pulmonary Tuberculosis.—Early diagnosis is most important. A positive family history is of some value, but it does not establish the diagnosis in a doubtful case, nor does its absence exclude tuberculosis. The *Bacillus tuberculosis* distinguishes pulmonary tuberculosis (*a*) from other catarrhs, such as acute and chronic bronchitis, or influenza; (*b*) from other consolidations, pneumonia, bronchopneumonia, actinomycosis, neoplasm, or syphilis; (*c*) from other vomicæ, such as the cavities of abscess, gangrene, actinomycosis, echinococcus, syphilis, or bronchiectasis; (*d*) from indurations other than tuberculous. *Cultures and inoculations* may be made in doubtful cases. Injection of Koch's *tuberculin* produces a local exudative reaction, and after four or five to ten hours, a systemic reaction, which is characterized by chill, fever elevation of even 5 degrees, headache, general pains, malaise, nausea, sometimes by erythema nodosum, an increase of sputum, rales or even a slight area of pulmonary dulness; if the first injection of 1 mg. of Koch's older tuberculin is negative, a second injection of 2 mg. may be given the following day. A reaction generally means that there is a tuberculous focus in the body, but not necessarily in the lungs, unless rales develop at the suspected spot after the injection. In some cases positive reactions have been obtained in chlorosis, syphilis, leprosy, cancer, and actinomycosis. Whatever the percentage of individuals with latent tuberculosis, it is large enough to cause many errors and render it of little clinical value; thus, a case with enlarged mediastinal and supraclavicular glands, without other physical findings, gave a positive tuberculin reaction, though the autopsy showed that they were carcinomatous from a clinically latent gastric carcinoma, and the reaction came from a single tuberculous mediastinal gland which was of no diagnostic significance. In veterinary medicine it is more valuable, leading to a correct diagnosis in more than 90 per cent. (Bang) or 97 per cent. (Voges). The author no longer uses it, for four cases of miliary tuberculosis have apparently resulted from its employment. Marmorek has lately devised a more rapid method, by which material (tissue or fluid) from the suspected case is injected into the peritoneum of a guinea pig, followed in twenty minutes by 0.3 c.c. of tuberculin; if the fluid is tuberculous, the reaction is obtained. The serum *agglutination test* is again reviewed by Arloing and Courmont (1904) in 100 cases; the bacilli (from homogeneous liquid cultures) produce macroscopic agglutination in a few hours; the test was positive in 88 per cent. of clearly tuberculous cases, in 73 per cent. of surgical tuberculosis, in 27 per cent. of apparently healthy persons, but is apt to fail in advanced consumptives.

Sticker recommended the use of *potassium iodide* in small doses (gr. iij); it congests the lungs at the seat of the lesion, possibly makes the auscultation findings clearer, and carries bacilli into the sputum; its use is not wholly free from danger. Neurasthenics may develop a phthiseophobia, just as they may develop a syphilophobia.

Pseudotuberculosis.—The Rhizomucor parasiticus (Lucet and Constantin), the ray fungus, streptothrix (Eppinger), and the aspergillus may cause pseudotuberculosis. The form caused by the Aspergillus fumigatus is described as a primary lung affection by Dieulafoy, Chantemesse, Widal, Rénon, Grancher, and Sergent. Many writers consider it secondary only. The fungus is composed of mycelia with clubbed ends. It stains with thionine and can be grown. The symptoms of streptothricosis are hæmoptysis (23 per cent.), fatigue, anorexia, dry cough, and, later, a greenish purulent sputum. The physical findings are those of bronchitis, less often those of slight consolidation or cirrhosis of the lung. Ashton and Wright (1905) collated 26 cases of streptothricosis pulmonum, of which most had symptoms resembling phthisis; over 50 per cent. were unmixed infections, and 76 per cent. died.

Prognosis of Pulmonary Tuberculosis.—Two issues are possible, recovery and death.

1. **Recovery.**—Autopsies on hospital cases show 25 per cent. (4 to 50 per cent.) of healed tuberculosis. Many of the recoveries are only relative, since Kurlow's inoculations from seemingly latent caseous foci into guinea-pigs were positive in 77 per cent.; such foci may be aroused by intercurrent disease, though it is the tendency of some writers to regard such recurrence as a fresh infection rather than the awakening of an old one. Watson observed a clinical course of fifty-four years, and Fagge one of thirty-five years. Recovery is effected by encapsulation, scar formation, and the deposit of lime salts. Liebermeister believes recovery is possible in 50 per cent. of incipient cases. The outlook depends on several factors: (*a*) the hereditary vital endowment of the patient; (*b*) his character, social situation, habits and intelligence; (*c*) whether the localization of the disease is single or multiple; (*d*) secondary or mixed infection; (*e*) digestion; (*f*) degree of toxin absorption (fever and emaciation); (*g*) youth, diabetes, pregnancy and lactation are unfavorable; and (*h*) complications (*v.i.*).

2. **Death.**—The clinical course is variable, even in chronic cases; it averages two years (Bayle, Laennec, Andral), over two and a half years (Pollock), three years (Cornet), and seven years ([Williams and Dettweiler], though the latter's cases did not include acute forms nor cases in children). The process of death is usually peaceful and

painless, though at times it comes with dyspnœa (stagnant secretion), delirium or convulsions. Mechanism of death: (*a*) heart failure is the common cause, associated with œdema, asthenia, anæmia, and marasmus. Other causes are far less frequent, as (*b*) profuse fatal hæmoptoë; (*c*) pneumothorax; (*d*) pulmonary embolism (clots or gas); (*e*) uræmia; (*f*) meningitis; (*g*) perforative peritonitis; (*h*) sudden death without adequate pathological findings. In cases of sudden death, Arnold, Brissaud, and Toupet, have found in the liver large numbers of bacilli, which probably caused an acute intoxication, since Mafucci's experiments have demonstrated that the injection even of dead bacilli causes rapid marasmus and death.

Complications.—Tuberculosis in other organs will be considered below.

1. Respiratory.—(*a*) Tracheitis and *bronchitis* occur in almost every case; and laryngitis in many cases results from the toxins. (*b*) *Dysphagia* usually results only from local involvement of the pharynx or larynx; swallowing into the larynx is usually the result of paralysis of the epiglottic depressors. *Hoarseness*, dysphonia, and aphonia, always suggest laryngeal tuberculosis, but may develop from such other causes as syphilis, erosions, or catarrh; from paralysis of the recurrent laryngeal nerve from pressure by lymph glands; from inflammation, on the right side by pleural scars, and on the left by large pleural effusions; or from thickening at the hilum. (*c*) Pulmonary *gangrene* may develop in the cavity wall. Lobar pneumonia is not infrequently a cause of death. (*d*) Dry *pleurisy* is an almost invariable complication, and is a conservative process, which lessens the probability of pneumothorax. Exudative pleurisy more often develops previously to lung phthisis than during its course. In the latter instance it is usually serofibrous, and is generally not extensive. Hemorrhagic pleurisy is more common in pleural than in pulmonary tuberculosis. Chyliform or adipose effusions may occur (see CHYLOTHORAX AND CHYLOUS ASCITES); purulent forms result from mixed infection, from cavities, or from pneumothorax. Some pleurisies appear to result from toxæmia rather than from direct bacterial invasion. Effusions by compression sometimes seem to exert a favorable effect on a lesion of the lung. Litten thought that their sudden absorption might occasion miliary tuberculosis. (*e*) *Pneumothorax* is observed in 5 per cent. of the cases (Powell, West), and results from rupture of superficial cavities which were forming rapidly without a limiting membrane. Perforation occurs most frequently between the second and fourth interspaces, and between the mammary and axillary lines. Its onset is spontaneous, or results from straining, or coughing. Its occurrence is usually marked by sudden symptoms, as great dyspnœa, orthopnœa, and cyanosis, and sudden physical signs, as lying on the side of rupture, sudden ectasia, immobility, dislocation of the heart, spleen, or liver, weak vocal fremitus, voice sounds and breathing, a tympanitic note, and when serum or pus forms, succussion. The onset is occasionally gradual and less urgent, so that pneumothorax may be an accidental finding. Death in a few hours may result, but life may be prolonged weeks or months. Infrequently pneumothorax (*q. v.*) may exert a beneficial effect on the lung trouble.

2. Circulatory.—(*a*) The *rapid pulse* first appears only after meals or exertion, but later becomes permanent. It is probably toxæmic, but may be due to mechanical pressure of large lymph-glands on the vagus, or to compression or inflammation of the vagus from mediastinitis, pleurisy or pericarditis. There is a constant and early *lowering* of the *blood-tension,* and the vasomotor instability causes the bright-red cheeks. The pulse may be soft and full throughout the course. Sometimes a capillary pulse or a centripetal venous pulse is observed. (*b*) A functional, systolic murmur over the pulmonary valves is very common. The *heart* is pale, fatty, flabby, and weak, and has been described as atrophied since the time of Laennec, but Potain considers that the small heart is merely accommodative to the malnutrition and anæmia. Cardiac hypertrophy is due to renal disease, pericardial or pleural adhesions, valvular disease, or emphysema. Hypertrophy and dilation of the right heart may follow pulmonary induration or massive pleural adhesions. Adhesive or effusive *pericarditis* may result by extension from pleurisy or caseated lymph-glands, but may be due to tuberculosis, pyogenic infection, or to toxæmia alone. Endocarditis tuberculosa is very infrequent; tubercle bacilli may be deposited in cardiac clots, or in endocarditic excrescences of non-tuberculous origin. They were first found by Kundrat and Heller, but Tripier's case of tuberculous endocarditis was the first clear one; according to Benda, this endocarditis is due to miliary deposits in the intima, and clinically escapes recognition in most cases, though it is sometimes suspected on account of a systolic murmur. (*c*) *Thrombo-phlebitis* occurs in about 1 per cent. of the cases.

3. Nervous and Muscular.—(*a*) There may be brain anæmia. In some instances the mental power may be normal; oftener there is a *slight mental weakness,* irritability, psychical lability, self-concentration, lack of endurance, and triviality. Sleep is often disturbed. Consumptives are usually *optimistic,* and they may plan for the future on the day of their death (*Spes phthisicorum*); occasionally melancholia or mania develops; fever, anæmia, toxæmia, marasmus, and vasomotor changes in the brain, such as œdema, may cause terminal aberration or paralysis. (*b*) *Neuralgia* and hyperæsthesia are not uncommon. Multiple *neuritis* not infrequently results from the toxæmia (Pitres and Vaillard); it may be latent or may cause only slight sensory disturbance, or it may result in amyotrophy; it may in part explain the œdema when it is not due to cardiac weakness or to thrombo-phlebitis. (*c*) The same toxins which reduce the body weight one-half or more, result in marked *muscular degeneration* and atrophy, which causes the flat chest, and possibly also the cardiac wasting. The muscular and adipose wasting is expressed in the terms synonymous with tuberculosis—consumption, *phthisis*, tabes. The muscles are dry, pale, thin, fatty; they show transverse rents, separation of the sarcolemma, poor markings, granular degeneration, some pigmentation, nuclear multiplication or hyperplasia of the connective tissue; myositis is described by Delorme and Reverdin. Their wasting and the nervous toxæmia explain the *asthenia* and incapacitation for work, a matter of national economical importance. The muscles are often irritable, show fibrillary contractions, and when tapped or pinched show local contraction or lumps, known as *myoidema,* which also results from emaciation in other such affections as typhoid or carcinoma.

4. **Digestive.**—(*a*) The tongue may be furred, or red and clean. Aphthæ are frequently distressing. Excessive *redness* of the *gums* is a frequent complication, but is also common in other affections. (*b*) *Stomach* disorder is present in 33 to 66 per cent. of cases; it may be the initial symptom of toxæmia, or appear only late in the process. The appetite may be unimpaired, and Cornet instances an advanced patient who complained that he could only eat four beefsteaks for dinner. *Anorexia* varies in degree and occurrence, being most marked toward evening, when the fever is higher, and most frequent in those with previous dyspepsia or poor appetite; it is sometimes nervous (anorexia nervosa) with normal gastric chemism; again it may be due to glands compressing the vagus (Peter, de Mussy). Brieger found the chemism normal in 50 per cent. in the incipient stage, in 33 per cent. in moderately severe, and in but 16 per cent. in severe advanced cases. Klemperer thinks that hyperacidity is usual, while Marfan considers it exceptional. After eating there may be a sense of fullness or even of pain. Nausea, gagging and *vomiting* may result from pharyngitis, coughing, or from forced feeding. In advanced cases, nausea and vomiting occur from over-eating, from toxæmic gastric catarrh, swallowing of sputum, weakness of the stomach musculature, and deficient secretion; anatomically, the stomach shows anæmia, catarrh, fatty degeneration of the glands (36 per cent.), interstitial hyperplasia, and sometimes amyloid infiltration (12 per cent.) or peptic ulcer.

(*c*) *Intestines.*—Constipation may be caused by gastric hyperacidity. Diarrhœa is usually late; it may be caused by the toxin in the blood or in swallowed sputum, by intestinal catarrh, toxæmic obliteration of the lymph- and bloodvessels in the gut, peptic or duodenal ulcers, tuberculous ulcers, amyloid degeneration or amyloid ulceration in the gut. Tuberculous *ulcers* may exist without symptoms or may cause colic, tenderness, pain, and diarrhœa; much depends on their location. According to Nothnagel, ulcers in the small gut, cæcum and ascending colon probably cause diarrhœa only when combined with catarrh or amyloid degeneration; ulcers in the lower colon and rectum almost constantly induce diarrhœa. It may, therefore, occur from causes other than ulceration, and ulcers may exist without it. Blood, pus, hæmatin crystals, and tubercle bacilli, may be present, but not always. Tubercle bacilli do not necessarily indicate intestinal tuberculosis, as they may pass through with the sputum swallowed. Anal fistula occurs in 1.3 to 5 per cent. of the cases. (*d*) The liver is fatty (in 10 to 15 per cent.); Jonescu (1902) collected 33 cases of cirrhosis cardiotuberculosa (induration from stasis, with tuberculous granulations and fatty degeneration). (*e*) Tuberculous *peritonitis* (*v. i.*) may occur by (i) lymphogenic infection—*e. g.*, from the pleura or pericardium, (ii) from the intestine, (iii) from the genito-urinary tract, or (iv) from the blood stream. It occurs in 5 to 15 per cent. of pulmonary tuberculosis. Its forms are diffuse exudative, diffuse adhesive, and tumor-like agglomerations.

5. Amyloid Degeneration.—This occurs mostly in chronic ulcerative phthisis. According to Blum, 80 per cent. of all amyloid degeneration is due to tuberculosis, and 68 per cent. to pulmonary tuberculosis; it is found in 8.8 per cent. of tuberculous autopsies. The spleen is affected in 93 per cent., the kidney in 77 per cent., the liver in 62 per cent., the intestines in 28 per cent., and the stomach in 12 per cent. of cases.

6. Genito-urinary—URINARY.—The loss of weight causes *phosphaturia* (calcium and magnesium), increase of nuclein and chlorides (0.5 to 1 or even 12 gm.). More proteid is excreted than is ingested. Albumose is sometimes present. *Albuminuria* usually indicates nephritis, amyloidosis, or local tuberculous disease. Acute and chronic parenchymatous nephritis is not infrequently due to long-standing toxæmia. *Amyloid* disease of the kidney produces albuminuria in but 50 per cent. of the cases; in amyloid of the kidney, nephritis is also present in 75 per cent., and the chronic parenchymatous type in 66 per cent. Bacilli are seldom found, unless there are secondary genito-urinary deposits. Ehrlich's *diazo reaction* is very frequently present, and is usually of unfavorable import; the converse is not true—*i. e.*, the prognosis is not necessarily good if the reaction is absent.

SEXUAL.—In some individuals, the sexual desire is more intense, particularly in women, though its increase is grossly exaggerated. The *menses often disappear early*, and pregnancy is less apt to occur. Pregnancy often temporarily betters the condition of the lungs, but a more rapid course is usual after delivery. Hypertrophy of the mammary glands, described by Bedor in 1836, occasionally develops, particularly in males, but is rather a simple chronic interstitial mastitis than an actual hypertrophy.

7. Skin.—Cyanosis is not common. *Pityriasis versicolor* occurs very frequently in the tuberculous; it is due to the tinea furfur (Eichstedt). Chloasma, desquamation, coarseness of the hair, herpes zoster, lichen scrofulosorum, and purpura, are sometimes observed. *Clubbing of the fingers* (the "Hippocratic fingers"), and toes, develops chiefly in chronic cases, is largely due to hypertrophy of the soft tissues (see ACROMEGALY) and is not pathognomonic of tuberculosis, for it occurs also in congenital cardiac disease, aneurysm, bronchiectasis, and emphysema. Emphysema of the skin is very uncommon; it follows interstitial emphysema of the lungs, and results from rupture of the alveoli, pneumothorax, or cavities. Œdema results from anæmia, cardiac weakness, phlebitis, or neuritis.

Treatment of pulmonary tuberculosis is considered later, after description of other localizations of the tubercle bacillus.

III. Tuberculosis of the Lymph Glands.

Glandular tuberculosis is most frequent in young children, 68 per cent. being found in the first ten years of life (Wohlgemuth). It also occurs fairly often in adolescence and sometimes even in the aged. It is found anatomically in 99 per cent. of tuberculous children (Sterner and Neureuther), or according to other statistics in 80 to 90 per cent.; it is found in 25 per cent. (Mueller) to 50 per cent. (Babes) of all autopsies on children. *Scrofula* is glandular tuberculosis, but it presents characteristics different from those of pulmonary infection, in explanation of which it is held by Arloing that the virulence of the bacilli is less in scrofulous glands; he found that inoculations made from infected glands produced local tuberculosis only in rabbits, while generalized tuberculosis resulted in the more susceptible guinea pig. Others explain it as an infection with very few bacilli. Neither of these explanations is wholly adequate, whence the assumption of a special glandular tendency to infection; it has been suggested that the glands invaded are poorly developed. The skin, mucous membranes, and lymph vessels, are physiologically more patent or more permeable in the very young, and an increase of this condition or its persistence in older children is regarded as an infantilism or embryoism of the glands, mucosæ, and skin, *i. e.*, a structural tendency toward infection. The accessory causes are the same as in pulmonary tuberculosis: (*a*) an hereditary tendency; (*b*) poor hygiene, dark, damp dwellings, inadequate nutrition and coarse food given to the very young; (*c*) infections, such as pertussis and measles, to a less extent scarlatina, diphtheria, variola, or syphilis.

General Characteristics.—(*a*) It usually runs an extremely chronic course. (*b*) Recurrence is very frequent. (*c*) Multiple lesions are usual. (*d*) Glandular involvement is frequently associated with affections of the skin (as eczema, scrofuloderma, or lupus), inflammations of the mucous membranes (as conjunctivitis, otitis, rhinitis, or bronchitis), and disease of the bones or joints (as coxitis, or Pott's disease). This association is frequently known as scrofula, of which some authors, as Cornet, distinguish two varieties—(i) the tuberculous and (ii) the pyogenic, caused by the staphylococcus or less often by the streptococcus. There is much dispute regarding this division, and it certainly seems incongruous to unnecessarily associate tuberculous lymphadenitis and arthritis with non-tuberculous forms.

General Anatomy.—(*a*) Few bacilli may cause but little inflammatory reaction and little cellular proliferation; regression is possible. (*b*) Conglomerate tubercles form, with the following issues: death of the bacillus in the central caseation, softening and external rupture, sometimes calcification or extension locally by the lymph stream or generally by the blood current. (*c*) Hyperplasia with epithelioid proliferation (Ziegler) may occur, without caseation or with very late caseation; this form results from infection with few bacilli. The lymphadenoid tissue is compressed by epithelioid spindle-shaped cells; the glands may reach the size of an egg, and their cut section show no gross evidence of tubercle. (*d*) Mixed infection causes rapid caseation, necrosis, softening, and early rupture; their cut section appears "like that of a raw potato but not so moist" (Virchow); the inflammation is often not limited to the glands, but causes a periadenitis, whereby the glands fuse, a phlegmon results, and generalization of the process throughout the body ensues. In the purely pyogenic (non-tuberculous) lymphadenitis, the glands may become homogeneous and dry (the hyperplastic lymphoma of Virchow), or productive inflammation may result in fibrous induration.

1. Cervical Tuberculous Adenitis.—Infection occurs through the nose, eye, scalp, mouth, pharynx, teeth, or ear; the primary atrial lesion may leave no mark; or the bacillus may pass through the mucous membranes without producing any lesion in them. Writers who describe scrofula as a special symptom-complex, distinguish four stages: (*a*) the initial skin or mucous membrane inflammation; (*b*) the lymphadenitis; (*c*) involvement of the joints and bones; and (*d*) various complications, as amyloid degeneration, or miliary tuberculosis.

The figures of Balman (first column) and Wohlgemuth (second column) show the following localizations:

	Per cent.	*Per cent.*
Cervical and occipital glands,	81	93
Axillary (from the mamma),	6	2.78
Inguinal (from the hip, ilium, testicle, and knee),	7	0.93
Cubital (from the fingers),	5	0.23
Popliteal (from the foot),	0.7	0.23
Pre- and postauricular,	——	2.9

The cervical glands thus make 96 per cent. of Wohlgemuth's series. In cervical scrofula, the glands at the angle of the jaw and beneath or anterior to the sternomastoid muscle are most often involved; in bilateral involvement, the glands of one side are usually larger. The glandular swelling is slow in onset, intermittent in progression, is usually unattended by pain or other symptoms, and is frequently an accidental finding. In some cases the first symptoms are acute, but the trouble subsides in two or three weeks. The intumescence is first confined to glands which are discrete and freely movable. Later the nodes may blend into large nodular masses, and periadenitis may fuse the glands and cause their adherence to the skin, which becomes œdematous, dough-like, and reddened. Softening and fluctuation are then frequent, with burrowing of the pus in various directions and external rupture through small fistulæ. The fistulæ widen, discharge cheesy and milky material, and form ulcers covered with granulation tissue and difficult to heal. Cicatrization leaves deep, deforming, depressed scars. Extension may occur to the deeper glands or to the subpectoral or axillary glands. Fever, anæmia, and other toxæmic manifestations, may develop, as in lung tuberculosis.

2. Bronchial Glands.—The bronchial glands are very frequently diseased. In autopsies on tuberculous children they are almost invariably affected (in 98 per cent. of Steiner's and Neureuther's and in 100 per cent. of Northrup's series); in adults they are affected in 40 to 80 per cent. In children tuberculosis of the bronchial glands is primary, the bacillus passing the patent intact bronchial mucosæ, and tuberculosis of the lung is secondary. In adults lung involvement is usually primary and bronchial adenitis is secondary. Small tuberculous foci cause neither glandular enlargement nor symptoms. Biedert, in 1,346 autopsies on tuberculous children, found the lungs diseased in 80 per cent., the bronchial glands in 78 per cent., the intestines in 32 per cent., and the mesenteric glands alone in 40 per cent.

SYMPTOMS.—In most cases no symptoms develop; in others, primary or secondary pulmonary lesions overshadow the symptoms of bronchial involvement; in others, miliary tuberculosis develops, promoted by the close contact of the bronchial glands with, and easy rupture into, the pulmonary vessels; in still others, symptoms result from the pressure of the glands on the nerves, vessels, or air tubes in the mediastinum; in very few cases are there actual physical findings.

Pressure Symptoms.—Pressure on the vagus, known to Wrisberg, causes the rapid heart which occurs so frequently in tuberculous children; paroxysmal asthma, dyspnœa, or spasmodic cough which sometimes closely resembles that of whooping-cough; lessening of the sensitivity of the bronchi, which favors infection; and it finally leads to cardiac dilatation and death. Pressure on the recurrent laryngeal nerve is marked by hoarseness, aphonia, dyspnœa, or laryngospasm. The air passages, trachea, or bronchi, may be compressed. This may be marked by dyspnœa, impaired expansion of one or both sides of the chest, and holding the head backward. The pupils may be unequal from pressure on the sympathetic nerve. Other pressure symptoms are: hypertrophy of the heart or the paradoxical pulse, from pressure on the aorta; œdema and cyanosis of the face and arms, from compression of the superior vena cava; cough, rales, or hæmoptysis, from stenosis of the pulmonary veins; a murmur over the innominate vein on throwing the head backward; dysphagia, from œsophageal compression.

SIGNS.—Bronchial breathing is occasionally heard between the second and fourth dorsal vertebræ, more clearly behind than in front; more on the right than on the left side; and more markedly than the high-pitched breathing heard physiologically over this region. If the bronchus is compressed there is neither bronchial breathing nor increased vocal fremitus. An irregularly outlined dulness is elicited on exceptional occasions either between the clavicle and third rib, on either side of the sternum, or behind between the third and fifth dorsal vertebræ. It is due to enlarged glands near a bronchus, or sometimes to a distended obstructed vein. Sometimes a tympanitic note in the upper lobes results from relaxation of the lung. Both the symptoms and signs are extremely variable, appearing suddenly and remitting unaccountably.

ISSUES.—(*a*) Tuberculosis of the lung (in children); (*b*) miliary tuberculosis; (*c*) tuberculous pleurisy or pericarditis; (*d*) pressure by calcification or fibrosis; (*e*) adhesion to and rupture into the air passages, causing sudden suffocation; (*f*) fatal hemorrhage into the air tubes, which is less frequent, as the vessels yield before pressure, but which may result from rupture simultaneously into a bronchus and a vessel; (*g*) rupture into the œsophagus or traction upon it, causing an œsophageal diverticulum.

DIAGNOSIS.—As Henoch puts it, "A diagnosis can rarely be made (from the physical signs), but if made is nearly always correct," as the bronchial glands are nearly always enlarged in infantile tuberculosis.

3. Mesenteric Glands.—The bacilli pass the patent intact intestinal mucosa and settle in the mesenteric glands, precisely as they pass the bronchial mucosa and lodge in the bronchial glands. Carrière found that but 1 per cent. of his cases were primary; of the secondary cases, the primary focus was in the peritoneum in 40 per cent., in the lungs in 30 per cent., in the intestines in 20 per cent., in other glands and bones each in 5 per cent. There are few characteristic symptoms. The glands may be palpable as thick nodular masses through a thin abdominal wall, but are often obscured by the abdominal distention. The general emaciation (*tabes mesaraica*) contrasts strikingly with the tumid belly. Digestive disturbance is most frequent, and there is usually a diarrhœa with thin, stinking stools. Compression is less frequent in the more distensible abdomen than in the less yielding thorax, though the veins of the abdomen may stand out and œdema of the legs may appear. The glands sometimes heal by induration and calcification, or, by softening, rupture into the intestine or peritoneum (plastic or exudative peritonitis). Amyloid degeneration may result from cases of long standing.

4. Generalized Lymphadenitis.—This may closely resemble Hodgkins's disease, as was first pointed out by Richard Paltauf and his co-worker, Sternberg, though the latter goes too far in declaring that most cases of pseudoleukæmia are glandular tuberculosis. In two personally observed instances of supposedly clear pseudoleukæmia, the clinical and gross pathological findings were typical of that disease, and only the epithelioid hyperplasia and the bacilli tuberculosis stamped them as tuberculous. This form is rather common in the negro.

The *treatment* of tuberculous lymphadenitis is that of tuberculosis in general (*v. i.*), and is surgical if the process is accessible and localized. In 175 cases of cervical adenitis which were operated on, 58 per cent. were cured after three years, 8 per cent. had recurrence, 11 per cent. developed pulmonary tuberculosis, and 23 per cent. died. In another series of about 700 cases which were treated medically, 28 per cent. developed tuberculosis in some other organ (Demme). The syrup of the iodide of iron and cod-liver oil are very largely used. Inunctions of green soap, recommended by Richter (1846) and Kappesser (1878) are considered beneficial by Hoffa; ʒss-j should be thoroughly rubbed into the back two or three times a week.

IV. Tuberculosis of the Serous Membranes.

These membranes may be involved simultaneously or separately. Multiple serous involvement is known as multiple serositis or polyorrhomenitis. Multiple serositis may be acute, subchronic, or chronic; it may be serous, fibrinous, or purulent; it may occur with or without lymphadenitis or visceral tuberculosis.

(*A*). **Pleura.**—Tuberculous pleurisy has been touched upon as a complication of phthisis, and must be separately considered under PLEURISY (*q. v.*). It may develop as an apparently primary infection, it may be clearly secondary to unmistakable phthisis, or it may appear as a terminal infection. Miliary tubercles in the pleura may occur without coincident pleurisy. Pleurisy develops in some form in 66 to 100 per cent. of pulmonary phthisis. It may be acute, subacute, or chronic; it is most often serous or serofibrinous; it may be serohemorrhagic, purulent, less often chyliform or adipose. Pleurisy may result from direct bacillary invasion from the bronchial glands, lung, spine, or peritoneum, or possibly from simple toxic action without the presence of bacilli. In some cases the pleura proliferates and forms a thick, massive, adhesive, obliterative membrane. Sometimes the interstitial tissue of the lung is invaded, resulting in pulmonary cirrhosis (pleurogenous cirrhosis). The symptoms are those of fibrinous, effusive or adhesive pleurisy (*q. v.*).

(*B*). **Pericardium.**—Miliary tubercles may in rare cases cause tuberculous pericarditis; these accidental miliary deposits must be sharply distinguished from tuberculous pericarditis wherein inflammation predominates. It is less common than pleural and peritoneal involvement, but is not a rare pathological finding. It is found in 2 to 3 per cent. of pulmonary tuberculosis. Tubercles may be detected beneath the fibrin deposits or adhesions, as they are in pleurisy of the same type. Without association with some other tuberculous lesion (especially that of the pleura) it is rarely separable clinically from the ordinary forms of pericarditis. Like these forms it may be symptomatically latent, it may form an unessential part of a generalized tuberculosis, or it may present the ordinary symptoms of pericardial effusion or concretion (*q. v.*). Only eight primary cases are recorded (Scagliosi, 1904).

(*C*). **Peritoneum.**—Tuberculosis of the peritoneum was known to Bayle, Laennec, Bichat, and others, a century ago, but became especially important when Louis, in 1825, declared that nearly all chronic peritonitis was tuberculous. Though some chronic peritonitides are due to trauma, alcoholism, nephritis, portal and cavastasis, most of them are tuberculous.

Frequency.—It complicates 9 to 13 per cent. of pulmonary tuberculosis and according to Grawitz and Brunn, it was found in 2 per cent. of 13,422 autopsies. According to Cummins it constitutes one-quarter of all kinds of peritonitis.

Age.—It is almost equally frequent in the second, third, fourth, and fifth decades, ranging close to 20 per cent. in each; 66 per cent. of all cases occur between ten and forty years of age; in the very young and the very aged it is infrequent.

Sex.—Ninety per cent. (Nothnagel), or 78 per cent. according to König, occurs in women. More cases are found in autopsies on males, though more women are operated on. In America it is more common among the negroes than among the whites. It is very seldom (0.7 per cent.) primary, but is secondary to (*a*) lung or pleural tuberculosis (in 80 per cent., Leube); (*b*) intestinal ulcers or mesenteric and retroperitoneal adenitis, especially in children. Tubercle bacilli may pass the intestinal wall without localizing in it and may reach the peritoneum. It is thought by some that intestinal lesions are the most frequent cause; (*c*) tuberculosis of the Fallopian tubes, which some consider causative of a third, or half, of the cases; tubal tuberculosis, however, is usually secondary to some small focus in the bronchial glands. Infection by contiguity and by the lymph-stream is said to be far more common than hæmatogenous infection, although the writer believes that tuberculous peritonitis is hæmatogenous.

Tuberculous peritonitis is a frequent complication of liver cirrhosis, as pointed out by Rokitansky, Foerster, and Weigert, though it seems strange that tuberculosis should develop in any stasis. It also complicates ovarian tumors. In some instances it follows trauma, which probably disseminates infection from a preëxisting quiescent focus. Occasionally it is found in a hernial sac (tuberculosis herniosa), of which 62 cases are reported (1903).

Types.—A clinical description is impossible, for the disease may be acute, subacute or chronic, latent or stormy, circumscribed or diffuse; it may be marked by adhesions, by free or encapsulated ascites, or by large tumor-like masses; it may be obscured by coincident miliary tuberculosis, liver cirrhosis, exudative pleurisy, or ulcerative phthisis, or may appear as a seemingly primary clinical fact. Though the forms are similar and atypical cases are common, certain types may be described. The unessential forms of miliary tubercle, and the small and usually non-inflammatory tubercles seen over intestinal ulcers or in chronic phthisis, are not taken into clinical consideration.

1. The *acute form* usually begins with severe symptoms, which include fever, rapid pulse, abdominal tenderness and pain, free fluid in the peritoneal sac, which shifts with change of position, considerable meteorism from paresis of the gut, vomiting, sometimes diarrhœa or at other times constipation. This type may resemble typhoid when the intoxication is more marked than the local signs. On the other hand, cases occur which simulate internal strangulation and ileus, which at times actually results from intestinal adhesions or incar-

ceration beneath bands. The writer has seen three cases precipitated by delivery and for this reason resembling a puerperal infection. Indicanuria, so frequent in other acute peritonitides, is usually absent. The ascitic fluid is usually lemon-colored, serous or slightly flocculent, and generally of a specific gravity of over 1,014; red blood-cells sometimes impart to it a hemorrhagic hue; the leukocytes are usually mononuclear. In rare instances the acute peritonitis is suppurative, and is due to perforation of a tuberculous ulcer in the gut. In some cases the acute type is a terminal infection. Though it is commonly diffuse, there is sometimes acute local pain or circumscribed effusion, which may occur, for instance, over an intestinal ulcer. Plastic adhesions occasionally tend to modify the freedom with which this ascitic form shifts with alteration of posture. Acute tuberculous peritonitis comes more often under the care of the physician than of the gynæcologist or surgeon.

2. *Subacute or chronic forms* present more or less diffuse peritoneal involvement. (*a*) The *fibrino-plastic form*, causing adhesions, may result from the acute form or may develop slowly and insidiously; symptoms and signs are usually lacking, or when present are not distinctive. (*b*) The *fibrino-caseous form* is characterized by the development of tubercles, which by fusion often form large pigmented solid masses. There are adhesions, and thickening of the intestine, mesentery, and omentum. Between the recesses formed by the inter-intestinal adhesions, or by the growth of the gut to the liver or spleen, pockets of exudate frequently occur, containing blood-stained serum, cold abscesses, or cheesy material; the intestine is thickened and contracted, the mesentery deformed, and the omentum shrunken. (*c*) The *effusive form* is marked by serous, serofibrinous, serohemorrhagic, and less often by puriform or adipose exudate, which is more or less encapsulated.

All these types and subtypes vary so much that the *individual symptoms* will be considered separately.

Individual Symptoms—1. Adhesions and Retraction.—The infiltrated *omentum* gradually *indurates and retracts* so that it is found above the navel (Bamberger) or along either costal arch as a fibrocaseous tumor-like mass; it may often be palpated, even when there is much exudate, and becomes clearer after paracentesis; it may be mistaken for carcinoma of the stomach, enlarged liver, or distended gall-bladder, especially when in chronic forms there is no temperature; it may also occur in simple or carcinomatous chronic peritonitis, but is quite suggestive of tuberculosis. The *walls of the gut* are frequently so *thickened* that intestinal stenosis and longitudinal shortening result; the gut may measure but one-half or one-third of its original length; it may be thickened, adherent, surrounded by exudate, and may also resemble a tumor. Adhesions may continue to retract until the entire peritoneal sac is deformed (Kleb's *peritonitis deformans*); this is a species of spontaneous recovery but is attended by danger of intestinal stenosis. The mesenteric thickening and retraction (*mesenteritis retrahens*) affects the character of the physical findings and the tympany and the dulness; the mesentery may measure one-half to one inch (1 to 2 cm.) in thickness.

2. Ascites; Exudate.—The abdomen is often moderately enlarged, which is due to serous exudation and meteorism; in some cases inspection reveals no objective change. Large exudates may cause some distention of the abdominal veins, as in ascites (*q. v.*), because the cava is compressed. When the abdomen protrudes, its centre is not usually flattened nor are its sides bulging as in the ordinary ascites of liver or cardiac disease (Bouilly), but it is often prominent in the median line, as in ovarian cyst. Löhlein states that most of his cases were sent to him with a diagnosis of ovarian tumor or cyst. In cases without adhesions the dulness shifts easily with change of posture, so that tympany exists over the supernatant gut and dulness is found over the settling fluid. The physical signs, however, differ from those of a common ascites in that the tympany is often on the right side; Thomeyer pointed out that the *mesenteritis retrahens* pulls over the small gut to the right side, and that its chief cause is tuberculous (less often carcinomatous or simple) peritonitis; though this disposition of the tympany is common, nevertheless, a tuberculous mass may develop anywhere, as for instance, in the right iliac region, in which case it dislocates the tympanitic intestines to the left, or in the median line or in the pelvis. In general the abdominal distention is asymmetrical rather than symmetrical; it is generally less extensive than, for instance, is liver cirrhosis, with which it may co-exist. In very rare cases the fluid may break through, into the bowel or externally through the navel. Dulness does not always indicate the presence of fluid but may result from close adhesions, from thickening of the gut, from caseous masses or enlarged glands.

3. Meteorism.—This may result from sudden paresis of the gut in the acute type, or from chronic fibrous obstruction in cases of long standing. Tuberculous tumors may also impair peristalsis and thus cause gaseous accumulations. Meteorism is one of the stigmata of tuberculosis of the mesentery glands (*tabes or phthisis mesaraica*) and then is often due to coincident peritonitis; in both conditions, but most often in children (Berggrün and Katz), the stools may appear uncolored and fatty.

4. Palpation.—A *doughy sensation* is often experienced on palpation, which is usually referred to peritoneal adhesions or constricted intestine, but in some instances is due to early infiltration of the preperitoneal (subserous) tissues.

5. Friction Sounds.—In some cases the *Beatty-Bright friction-rub* can be elicited.

6. Pelvic Effusion.—This is particularly common in women, who also suffer from painful and disturbed menstruation.

7. Temperature.—Temperature frequently occurs, but is irregular in character; it is sometimes continuous, even typhoidal, and sometimes remittent, or hectic; it is most frequent in acute types or in acute exacerbations of chronic cases. In some chronic cases it is absent or subnormal. The disease therefore cannot be excluded by the absence of temperature. Fever, with pain and tenderness, usually, but not always, distinguishes tuberculous encapsulation from simple ascites.

8. **Pain and Tenderness.**—These are generally conspicuous, though moderate in degree and by no means constant.

9. **Marasmus**—Emaciation is common and has been especially emphasized by Vierordt; König, however, remarks upon the frequency with which cases of tuberculous peritonitis present a good color and robust appearance.

10. **Gastro-intestinal Disturbances.**—Digestive disorders are fairly frequent; the usual constipation is occasioned by impaired peristalsis; in some cases diarrhœa, amounting to 3 or 4 movements daily, is possibly caused by toxæmia.

11. **Other Symptoms.**—The spleen may be enlarged, but is difficult to find in the majority of cases. In rare instances there are icterus, pylethrombosis, and other complications due to special localizations of the process.

Diagnosis.—The diagnosis is usually made correctly, though sometimes there are no just grounds for even suspecting the presence of the disease. Considerable importance should be attached to tuberculous lesions elsewhere, as in the lungs, pleuræ, glands, or Fallopian tubes. Multiple serositis is extremely suggestive. If another tuberculous lesion cannot be established, tuberculous peritonitis, which causes nearly all chronic peritonitides, is very probably present; malignancy and traumatism must be considered. Tuberculin injections may be given but do not prove that the lesion is peritoneal or tuberculous. Inoculation may be made with fluid withdrawn by paracentesis (see PLEURISY). Exploratory laparotomy is justifiable, particularly as simple abdominal section is the foremost therapeutic measure.

Course.—The onset is usually insidious with marked but not extreme pain, tenderness, anorexia, vomiting, colic, fever, and constipation or diarrhœa. The course, independent of all therapeutic measures, shows spontaneous remissions, as Vierordt and Nothnagel have particularly insisted.

Treatment.—Simple rest in bed, good hygiene, and full feeding, very often arrest the process. The tumors, exudate, and adhesions, may recede and no symptom reappear for months or even years. Spontaneous recovery was first described by Bamberger but was ignored until relatively recent times. In 1884 König proved what Spencer Wells had observed twenty years earlier, that healing may follow laparotomy (Czerny, Hegar, Chrobak, Bumm, Israel). Tubercles may heal entirely in six or seven weeks. Then Lindner, Phillips, Valenta, Roersch, Hinterberger, and others, showed that recovery was frequent (in 25 per cent. of the cases) without either medication or laparotomy. Experimental researches have shown that the tubercles heal by simple degeneration, or by vascularization and development of connective tissue.

Operation gives various results, recovery occurring in 33 per cent. (Frees), 55 per cent. (Chrobak), 64 per cent. (König), 91 per cent. (Dohrn) and 94 per cent. (Mazzoni). Larger numbers of cases give equally high percentages; 70 per cent., Roersch, 358 cases; 85 per cent., Margarucci, 250 cases; von Krenecki, 266 cases, 72 per cent. recoveries in ascitic, 62 per cent. in adhesive, and 75 per cent. in encysted forms; 75 per cent. in 405 cases, Adossides. If the primary focus is accessible, as in tuberculous tubes, it should be removed, thereby raising the percentage of recoveries from 71 to 97 per cent. (Mayo). Simple paracentesis is much inferior to laparotomy. Lavage is superfluous. It is thought that the access of air or sunlight, or manipulation or lavage, is the potent curative factor in operative treatment. Veit advances the plausible theory that the withdrawal of fluid allows the access of fresh serum with increased antitoxic properties. Operation, in the author's opinion, is clearly of great value; but it may be mentioned that some conservative writers believe that the tubercles which heal after operation were already in process of healing spontaneously.

V. Tuberculosis of the Brain and Meninges.

Involvement may result from (*a*) the miliary tubercle, (*b*) the solitary, or, as Virchow prefers to call it, the conglomerate tubercle, (*c*) the tuberculous abscess, and (*d*) meningitis, acute or chronic. The miliary form has already been described; the solitary or conglomerate tubercle produces symptoms identical with those of brain tumor (*q. v.*), and tuberculous meningitis will be described with other meningitides (*v.* MENINGES).

Primary cerebral tuberculosis is extremely rare. Warfringe, Fraenkel, Alvarez and Duerck have described primary meningitis, but it is extremely probable that small or microscopic foci elsewhere escaped detection. Fraenkel found tuberculous meningitis in 4.7 per cent. of adults dying with pulmonary phthisis.

VI. Tuberculosis of the Alimentary Tract.

1. Lips.—The lips are very seldom affected; lupus or tuberculous ulcers may invade them. The latter are very painful and may be confused with cancer or chancre.

2. Gums.—The gums are seldom involved, though tuberculosis has followed caries or extraction of the teeth.

3. Tongue.—This is involved in 1 to 2 per cent. of cases. Tuberculous tumors may develop in its substance. They are sometimes mistaken for gumma or cancer, and may break down into cold abscesses. Tuberculous ulcers develop on the dorsum or edges; they have rough caseous bases with uneven or undermined edges; they are almost never primary; they are differentiated from syphilitic ulcers by the inefficiency of mercury and iodides, from cancer by the absence of glands in the neck, and from both by the presence of bacilli and the results of inoculation.

4. **Pharynx.**—Pharyngeal involvement was known to Morgagni but was especially studied by Ricord (1865). Primary disease is most infrequent, and involvement secondary to pulmonary or laryngeal phthisis is more common, but still is infrequent; Heller saw but 4 instances in 8,000 throat cases; Kidd found 6 cases in 500 tuberculous autopsies. The process begins largely in the lymphoid structures, which are infected by tuberculous sputum or food, or it extends by the lymph-vessels from the larynx. Three-fifths of the cases (Martineaux) are associated with tuberculous disease of the intestine, and 15 per cent. (Barth) with oral tuberculosis. It is promoted by syphilis, by the use of alcohol and tobacco, by trauma, and by preëxisting ulceration. The cervical glands may be affected in acute, but rarely in chronic, cases. The ulcerations are usually superficial; when propagated from the larynx, the most distressing dysphagia results. *Retropharyngeal abscess* (Read, Lindenbaum, Northrup) usually results from suppuration of the lymph glands and from spinal or osseous disease.

5. **Palate.**—The palate is said to be affected in 1 per cent. of cases of phthisis; in swallowing or coughing, tubercle bacilli from the sputum come in close or even forcible contact with the palate. Lublinski observed three instances among 16,000 throat cases, and Kidd saw 4 cases in 500 tuberculous autopsies.

6. **Tonsils.**—The tonsils are diseased in 4 per cent. of cases of pulmonary tuberculosis (Warthin); their folds and crypts contain bacilli; the process may stop at the tonsils, for they act as barriers to the infection or a tuberculous cervical adenitis may develop. Their infection may be primary, and Dieulafoy holds that tonsillar infection may be followed by a descending tuberculous adenitis, which in turn infects the lungs. Some maintain that the tonsils are almost invariably infected by the sputum in phthisis of the lungs; Schlenker finds them tuberculous in 65 per cent., Krueckmann in 48 per cent., and Dmochowski in 100 per cent. of his cases of pulmonary phthisis. The lesions are miliary more often than caseous.

7. **Salivary Glands.**—These are fairly, but not absolutely, immune.

8. **Œsophagus.**—The œsophagus is also relatively immune and is infected less frequently even than the throat. No clear primary case is recorded, and Cone could collect only 48 secondary cases. Œsophageal trauma, cancer, or ulceration, tends to localize the bacillus. Infection by contiguity, from the peribronchial glands, is more common than infection by tuberculous sputum; these glands may occasion œsophageal perforation, hemorrhage, diverticula, or stenosis. Extension from the larynx or pharynx along the lymph tracts in the submucosa, or from the spine or lung cavities, is possible. Miliary deposits have no clinical importance.

9. Stomach.—The stomach almost never shows primary ulceration. There are only four cases on record in which the smallest claim for primary involvement can be made. Secondary disease in pulmonary tuberculosis is estimated at four-tenths of one per cent. to 2 per cent. Letorey, in 1895, collected 21 cases of tuberculous ulcer, but they are more frequent than statistics show. Ricard and Chevrier assert that there are on record sixteen cases of tuberculous stenosis of the pylorus. Klebs's idea that gastric localization was promoted by round ulcer is not generally accepted. The HCl is inimical to gastric tubercle. Tuberculous ulceration of the stomach occurs from the sputum, from invasion by contiguity (glands, peritoneum, or colon), or in few cases from hæmatogenous infection.

10. Intestinal Tuberculosis.—This was first observed by Bayle, (1810). (*a*) It is usually *secondary* to lung tuberculosis with cavity formation, and results from infective sputum; its frequency is 30 per cent. (Eichhorst), 34 to 38 per cent. (Leroux and Mueller, in children), 51 per cent. (Heinze), or 90 per cent. (Weigert, Orth, Warthin). Tubercle bacilli may be swallowed without causing intestinal tuberculosis. Of all kinds of intestinal ulceration, tuberculous ulcers are the most frequent. Infection in some cases may occur from the peritoneum or mesenteric glands, but adhesions often render the determination of the primary focus impossible. Hæmatogenous infection is barely possible. (*b*) *Primary* intestinal tuberculosis is much more rare, occurring in but 0.3 per cent. (Eisenhardt), and in only 0.1 per cent. of the cases in adults. The intestinal mucosa in children is like the bronchial, as it often allows of the passage of tubercle bacilli through it to the intestinal glands without disease of the intestine itself; this is due to the loose infantile mucosa and the large lymph vessels. Kingston, in 339 autopsies on children with tuberculosis, found its atrium was respiratory in 64 per cent., intestinal in 19 per cent., and uncertain in 17 per cent. Infection may occur by infected milk or meat. Koch takes an extreme view in denying infection by milk, and Behring takes the opposite extreme in affirming that it is the chief cause of tuberculosis.

The *ulcers* are most frequent in (i) the *ileo-cæcal* region and next in the colon. (ii) They always originate in the *lymphadenoid follicles* or Peyer's patches and never in the mucous membrane; they are promoted by erosions or obstipation. Small tubercles fuse into large ones, which caseate and ulcerate; in exceptional cases the caseous nodes do not rupture but cause the peritoneum and mucosa to bulge out. (iii) The ulcers are *irregular* in shape, and (iv) their disposition is *ring-like; i.e.*, they are located transversely around the gut, because extension occurs by the lymph vessels (lymphangitis tuberculosa). Occasionally they lie in the long axis of the gut and are ovoid, corresponding, though often incompletely, to Peyer's patches. (v) They are single or multiple, and sometimes cover nearly all the surface of both the large and small intestines. (vi) Their bases are rough or caseous and their edges infiltrated; the infiltration also invades the contiguous mucosa and submucosa. (vii) Localized adhesive peritonitis is a common issue when the process reaches the serosa. An early stage of this is seen in the serous and subserous miliary tubercles and in the bead-like tuberculous lymphangitis; perforation occurs in 5 to 10 per cent. of the cases, but is resisted by the muscular coat and by adhesions; it results in pericæcal abscess and in suppurative peritonitis. *Stenosis* of the intestine, single or less often multiple (129 cases collected by Arbuson, 1904), may also result; recovery is very rare. König in two years saw 5 cases recover, and Eisenhardt saw 10 complete and 26 incomplete instances in 567 tuberculous ulcerations. Hepatic suppuration or steatosis, may result.

Symptoms.—The symptoms are those of any form of intestinal ulceration (*q. v.*); they commence usually with signs of catarrhal enteritis, diarrhœa, pains, and fever. They may for a while resemble those of typhoid. The only pathognomonic finding is the tubercle bacillus, and it is possible that its presence may result only from swallowed sputum. Rosenblatt's method of detecting the *bacilli* in the fæces is to give laudanum until the stools become hard, and then examine the mucus on their surface; the bacilli are usually found on the first examination, for the hard fæces apparently scrape the bacilli from the ulcers. *Pus* is often found and hemorrhage occasionally results. Girode called attention to the black stools, which are usually ominous, as they indicate malnutrition; the picture of dysentery is rarely observed. Fatal hemorrhage is infrequent, though cases have been recorded by Vallin, Hanot, Osler, Chandèze and others. An *appendicitic form* may be noted, with quick or insidious onset and with some induration; recurrence is frequent and fistulæ occasionally develop. In *typhlitis tuberculosa* there is great thickening in the cæcal region, with a hard, slightly movable or totally adherent, vertically disposed, tender tumor. The onset is usually insidious, pain is recurrent, symptoms of increasing intestinal stenosis are present and constipation alternates with diarrhœa. Hemorrhage is infrequent, fever is usually absent and emaciation is sometimes pronounced. Duguet described this condition in 1869, and, since it so fully resembles cancer or sarcoma, it has attracted the especial attention of the surgeons, Bassini (1887), Bouilly (1889), and Billroth (1891). Campiche (1906) collected 379 cases of tuberculosis of the appendix and cæcum; about 25 per cent. of the operated cases died from the operation; about 35 per cent. were well for months or years; and about 40 per cent. ultimately died of tuberculosis. It sometimes co-exists with cancer. The process usually begins in the mucosa and is marked by ulceration, villosities in the mucosa, great local infiltration and hypertrophy of the bowel, and destruction of the ileocæcal valve. The process may begin, as shown by Conrath, in the serosa, with which tuberculous glands may come in contact. Patients may die of acute obstruction, or only after a chronic course of a decade or more; recovery by fibrosis is possible. Benoit and Dieulafoy regard the process as primary in 80 per cent. of subjects.

In the *rectum,* ulcers, either anal or rectal, lupus, tuberculosis cutis verrucosa, and periproctal abscess, are sometimes seen. *Fistula in ano* occurs in 1 to 5 per cent. of cases of phthisis, and rarely develops in other conditions; the general idea that its excision excites pulmonary disease is a confusion of cause with effect.

11. Liver.—The liver is involved less in adults than in children (38 per cent. Leroux), but at all ages it is of greater pathological than clinical interest. (i) Large tubercles may develop in the liver, sometimes with peritonitis and perihepatitis. (ii) Miliary tubercles are of no clinical significance. (iii) Hanot maintains that there is a primary cirrhosis of the liver, associated with tubercles and fatty degeneration; at the most it is extremely rare; it is said to be associated sometimes with tuberculous peritonitis and perihepatitis (*v. s.* TUBERCULOSIS OF THE LUNGS: COMPLICATIONS).

12. Spleen.—The spleen is involved in 43 to 47 per cent. of tuberculous children (Leroux and Mueller). Bayer (1904) collected 28 cases of primary tuberculosis of the spleen; 19 were not recognized clinically; 9 were operated on, of which 2 died and 7 recovered. (See DISEASES OF SPLEEN: SPLENOMEGALY WITH CYANOSIS.)

VII. Tuberculosis of the Genito-urinary Tract.

Genito-urinary tuberculosis was described by Morgagni and by Bayle in the beginning of the last century, but for the first time fully by Lichtheim, in the cadaver, and by Rosenstein, Babes, and Smith, clinically. It is most frequent in the period of greatest sexual activity (twentieth to fortieth year), and 72 per cent. occur in males, in whom the two tracts have more continuity and community of surface.

Modes of Infection.—1. Hæmatogenic Infection.—This is the most frequent variety. It occurs in 5 per cent. of cases of chronic pulmonary tuberculosis. The majority (76 per cent.) of cases are secondary to some lung or peribronchial glandular focus, which very often has produced no symptoms, so that most seemingly primary foci are really secondary. Jani found tubercle bacilli in perfectly sound testes and prostate glands, but they were clearly an invasion of the blood occurring in the death agony.

2. Urogenic Infection.—The general opinion was previously that a secondary infection mounted from the lower genital to the higher uropoietic structures. It may be conveyed by infected fingers, instruments, or catheters. Infection by sexual intercourse is possible but has been grossly overrated. It may follow syphilis and gonorrhœa.

3. Infection from the Peritoneum and Contiguous Structures.—The *Fallopian tubes* are diseased in a third to a half of the cases of tuberculous peritonitis. Jani found tubercle bacilli in the tubes in women dying of phthisis. The bladder, seminal vesicles, and vasa deferentia, may be invaded *per contiguitatem* from peritoneal, vertebral, rectal, and other foci. In many cases it is impossible to determine where the process began, for extension may have occurred so rapidly and diffusely.

(*A*). **Renal Tuberculosis.**—The kidney in *miliary* tuberculosis is involved in 90 per cent. (Frerichs), but is marked by no distinguishing symptoms. *Caseous nodes* develop in the cortex, infection reaching it most often (in 90 per cent.) from the blood stream (desquamative caseous nephritis); they often break down into cavities, and may form cold abscesses or undergo secondary pyogenic infection, or the process may remain localized in one pole of the kidney. Tubercles also develop in the apices of the pyramids and pelvis of the kidney, and form caseous infiltration and ulcers; involvement in this form was at one time considered the result of an ascending infection, though Caspar and Walker oppose this view; some maintain that infection from below may pass through the bladder (without causing lesions in it) and reach the kidneys, just as the bacilli may pass the lungs or intestine and cause bronchial or mesenteric adenopathies. Israel and others, on the contrary, believe that the process often (41 per cent. of cases) descends from the kidney to the bladder. Wherever the process begins, upward and downward extension in the kidney itself often occurs, so that the entire organ may be more or less diseased. When the process is well developed there is a tuberculous pyonephrosis. The entire kidney may become a large sac of caseous material, detritus, and lime salts; this is called *dégénérescence massive* by Tuffier. The kidney is often adherent to adjacent structures; its capsule is thickened, its removal strips off particles of renal substance, and it is sometimes perforated, causing tuberculous paranephritis. The surface of the kidney may be smooth and normal, lumpy from caseous deposits, or softened in many places from diffuse ulceration. In exceptional cases spontaneous healing is possible. Whether one or both kidneys are diseased depends on the stage of the process, and is a most important matter when surgical interference is considered. Israel, in his celebrated observations, stated that he found one kidney involved usually without any other genito-urinary disease. Pallet and Albarran observed unilateral disease in 80 to 85 per cent. of their cases, while postmortem figures show unilateral renal disease in but 48 per cent.; naturally, in autopsy figures, the extent of the disease is greater than in clinical figures. Compensatory hypertrophy of the sound kidney is frequent.

Symptoms.—These may be absent if the renal pelvis remains sound. Otherwise the symptoms are (1) urinary, (2) local, and (3) general.

1. URINARY.—The urinary symptoms are chiefly those of pyelitis (*q. v.*). The urine is acid, though alkalinity develops in complicating tuberculous or non-tuberculous cystitis, which may be determined by the cystoscope. Urination is often difficult (dysuria) or frequent (polliakuria), so that the existence of cystitis is at first suspected; it is a safe rule always to consider the possibility of renal phthisis in every cystitis the origin of which is not entirely clear and the treatment of which is unsuccessful. Bladder symptoms are reflex or are due to compli-

cating cystitis, simple or tuberculous. The urine is sometimes increased; Guyon describes an early polyuria in which the urine is clear, and a later polyuria in which it is turbid. *Hæmaturia* (*q. v.*) is frequent; it may be severe or slight and its occurrence is more often early than late; its frequent recurrence is so marked in some cases as to justify the term "hemorrhagic type"; it may last one and a half months (Routier) to four and a half years (Tuffier). *Pyuria* generally occurs when the pelvis is invaded, but is not observed when cortical foci do not communicate with the pelvis nor when the ureter is occluded; the pus cells correspond to the amount of albumin. Epithelial cells, sometimes caseous matter, elastic fibers, and necrotic renal tissue, are found. The sediment is often a crumbly, rapidly settling detritus; *tubercle bacilli* are often found, but must not be confused with the smegma bacillus (*v.* GENERAL BACTERIOLOGY); sometimes bacilli in the urine are found in pulmonary tuberculosis without any disease of the kidneys. Casts are very infrequent. In unilateral renal disease with occlusion of the corresponding ureter, the urine is naturally normal. Urinary segregation or ureteral catherization determines which kidney is involved.

2. LOCAL.—Local symptoms are lumbar pain and tumor. *Lumbar pain* sometimes results from capsular tension; it is severe, colicky and is reflected to the groins and testes, which may be retracted as they are in renal colic; this is probably due to the passage of caseous particles or blood clots; differentiation from calculous disease (*q. v.*) may be difficult in such cases until tubercle bacilli are found. *Tumor*, in tuberculous pyonephrosis or hydronephrosis, is a small, tender enlargement, though seldom of the size attained in calculous or other obstructive disease.

3. GENERAL.—General symptoms are hectic fever, malnutrition, tuberculosis in the lungs, and foci in the testes. The heart is almost never hypertrophied. In 60 per cent. of the cases operated on, the diagnosis was found correct. The course is chronic, with remissions; two or three, or even five, years is the usual duration; recovery from circumscribed lesions is possible; death results from tuberculous generalization, less often from sepsis, perinephritis, or amyloid degeneration, and rarely from nephritis or uræmia. The *treatment* is surgical. In Krönlein's cases, the operative mortality of nephrectomy was 5.8 per cent. and the ultimate mortality was 17.6 per cent.; 70 per cent. was cured. Caspar claims that, since the introduction of ureteral catheterization and functional renal tests, the mortality has fallen to 7.7 per cent. This is denied by Israel. In Albarran's series the operative mortality was 3 per cent.

(*B*). **Ureter and Bladder.**—The ureter is thickened, infiltrated, caseous, ulcerated, and often stenosed; it is involved in 80 per cent. of cases of renal tuberculosis (Palet) and may cause various adhesions, such as to the peritoneum. Secondary involvement from the kidney may be limited to the ureter at its vesical ending or may spread more widely over the mucosa of the bladder.

Bladder disease is rarely primary; secondary disease results *per contiguitatem* or from hæmatogenous deposits, as can be seen by means of the cystoscope. Israel (*v. s.*) noted tuberculous cystitis in 41 per cent. of cases of renal tuberculosis, and Vignernon in 50 per cent. Cystitis of long standing should always excite suspicion of tuberculosis, even when it has gonorrhœal or other antecedents. Medical measures are only palliative. Surgical intervention is indicated.

(*C*). **Male Genitalia.**—The epididymis is more often involved than the testis, except in youth, when the epididymis and tunica albuginea are more frequently affected; in the first years of life testicular disease results from generalized tuberculosis; about one-half of Jullien's cases occurred under two years of age. Some hold that these cases are congenital, though the weight of evidence is against this view. Sometimes no caseation results, as is the case in the hyperplastic lymphadenitis, and even microscopic confusion with sarcoma is possible unless the bacilli are especially looked for. Testicular tuberculosis is most often confused with syphilis (*q. v.*), though in the latter there is less pain and fever and more nodular enlargement. The seminal vesicles, vasa deferentia and urethra may be invaded and a rectal examination should never be neglected. Recently the prostate, "the cross road of the urinary and genital tracts," has attracted especial attention; its primary involvement (Lancereaux) is disputed by some, but the gland is affected in 50 to 80 per cent. of the cases of genital tuberculosis in the male. The lungs are invaded in primary genital tuberculosis in 70 per cent. and the urinary tract in 56 per cent. *Treatment* is surgical. Medical measures are usually unavailing.

(*D*). **Female Genitalia.**—The female genitalia become tuberculous in 1.5 to 2.5 per cent. of the cases of pulmonary tuberculosis. Infection is secondary in over 80 per cent. Tuberculous tubes are found in 1.5 per cent. of abdominal operations; according to W. Williams, 8 per cent. of the tubes removed for inflammatory lesions were found tuberculous. Probably nearly all cases are due to some small focus in the bronchial glands. The tubes show nodules, infiltration, caseation and ulceration; bilateral involvement is usual, and its true nature may be apparent only under the microscope; tuberculous salpingitis may simulate tuberculous peritonitis; tubal disease may be found in very young children. The uterus ranks next and is, as a rule, involved secondarily; systematic microscopic examination would doubtless show higher percentages than those usually given. Ovarian phthisis is almost always secondary. Tuberculosis of the vagina, cervix, vulva, and female urethra, are very exceptional.

Mammæ.—The mammæ are occasionally diseased; Schley (1903) collected 65 cases of which 12 were primary. Carcinoma may be closely simulated, for tuberculous lesions occur at the same age, often cause nipple retraction and tumor formation, or involve the axillary glands (66 per cent. of the cases). When cold abscesses, fistulæ, or ulcers form, the diagnosis is naturally more definite. A chronic interstitial non-tuberculous mastitis was described by Bedor.

VIII. Tuberculosis of the Upper Respiratory Tract.

(*A*). **Nose.**—The nose is seldom affected, because its irritation excites lachrymation, sneezing, and increased secretion, which is inimical to the development of tubercle bacilli. Nasal localization was first described by Willigk (1853). In the rare primary form there are very few bacilli and there is an exuberant non-caseous growth, which may somewhat resemble sarcoma; the absence of caseation is not unique because it is sometimes seen in lymphadenopathies and in the Fallopian tubes; there is little pain and the chief symptom is a chronic coryza. In the secondary form the bacilli are abundant; multiple caseation and ulceration occur as in other localizations; suppressed coughing, with the mouth closed, is thought to force germs into the nose. Nasal infection may occur by picking the nose, and is promoted by foreign bodies, wounds and ulcers, and by stagnation of the secretion by various obstructive processes. Infection may spread by the lymph vessels, especially in children, to the cervical glands or even to the meninges. Lupus (in 33 per cent.) affects the nose (Bender).

(*B*). **Nasopharynx.**—Its primary involvement is infrequent. Secondary disease usually develops late in phthisis and occurs in 20 per cent.; it is promoted by coughing with the lips closed. It may extend by contiguity from the pharynx. The third tonsil may be affected (Lermoyez); adenoids are tuberculous in 20 per cent. (Dieulafoy) or in 27 per cent. (Moure and Brindel).

(*C*). **Ear.**—Tuberculous *otitis media* occurs chiefly in late phthisis (according to Fraenkel in 32 per cent.); otitis may occur from mixed infection. It is promoted by measles and scarlatina, is rarely primary, and often causes cervical lymphadenitis.

(*D*). **Larynx.**—Laryngeal phthisis is very rarely primary, though some well established cases are recorded. Louis, in 1825, held that it was secondary to lung disease resulting from infection by sputum, which view still holds; hæmatogenous and lymphogenous infection is rare. Laryngeal tuberculosis develops in 20 to 30 per cent. of cases of pulmonary tuberculosis in adults; it develops in only 3 per cent. of pulmonary phthisis in children (Parrot). It complicates 9 per cent. of lupus cases. Promoting factors are inflammation, fissure, and syphilis. In 700 cases Krieg observed unilateral involvement in 275 (39 per cent.), of which 92 per cent. occurred on the same side as the lung lesion; Kreig attributed this to lymphatic extension; Cornet refers it to paresis of the vocal cord on the affected side, (which is due to compression or neuritis of the recurrent laryngeal nerve), and to stagnation of the sputum in various recesses on the paralyzed and anæsthetic side. Unilateral disease usually becomes bilateral from contact.

Symptoms.—The *cough* varies with the time of onset of laryngeal symptoms; in those rare and unfavorable cases in which the trouble begins with laryngeal symptoms, the cough is often somewhat dry, brassy, or metallic; it may come on in the form of acute laryngitis. In cases clearly following lung disease, the ordinary bronchitic hack frequently becomes brassy; in the last stages it becomes incomplete, ineffectual, or like an eructation (Trousseau), as the vocal cords cannot come in apposition. Tickling in the larynx is common. *Pain* and *hoarseness* are followed by *dysphonia* (or, later, *aphonia*), *dyspnœa*, and the most painful and distressing *dysphagia*.

Forms.—(*a*) The most common form is the ulcerative; ulcers are found in the interarytenoid region, vocal cords, posterior wall of the larynx, arytenoids, and epiglottis; they are not deep but are broad; their edges are ragged or "nibbled," and, on their rather pale and sometimes caseous bases, papillary excrescences are fairly frequent. Particles may be inhaled into the lungs, causing tuberculous or suppurative foci. They are to be distinguished from syphilitic ulcers (*q. v.*), which are more often single and are redder, have more "cut out" edges, frequently show cicatrization, involve oftener the parts nearer the pharynx (epiglottis and posterior surface of the arytenoids), and less often attack the vocal cords; show no tubercle bacilli, do not show local reaction on tuberculin injection and do respond to mercury and iodides. Ninety per cent. of all laryngeal necrosis is tuberculous. Perichondritis may occasion sudden œdema glottidis, urgent dyspnœa, and early death unless timely tracheotomy is performed. (*b*) In other cases the submucous tissues are infiltrated and the mucosa is thickened or sometimes hardened (pachydermous form); the true cords are less involved than other parts; stenosis rarely occurs, and if ulceration develops it is inconspicuous. (*c*) Caseous tumors are not frequent; they may precede the ulcerative form or occur independently of it. (*d*) The vegetative type occurs with ulceration, and small excrescences develop on the floor of the ulcers, or without ulceration, in which case they appear on the otherwise intact mucosa. (*e*) The miliary form is of no essential importance, as the deposits can rarely be seen and their symptoms are obscured by the pulmonary or cerebral manifestations.

Treatment.—Treatment is unsatisfactory, as pronounced lung changes already exist and dysphagia occasions rapid emaciation and disinclination to eat. Besides general measures (*v. i.*), local measures may be employed, but as a rule they do not essentially modify the process. Insufflations of boric acid, iodoform, or iodol, and painting with 10 per cent. menthol, 10 per cent. carbolic acid or 20 per cent. lactic acid solution (gradually increased in strength to 80 per cent.), may relieve the local pain; gr. ¼ of morphine hypodermically and 10 per cent. solution of cocaine locally, allow the patient to eat, for a time at least, but finally fail to anæsthetize the widespread ulceration. The patient may be able to draw up milk and water by a tube, when the head is turned over the edge of the bed. Caustics, such as silver nitrate, do more harm than good.

IX. Tuberculosis of the Heart and Vessels.

(*a*) Pericardium: (*v.* SEROUS MEMBRANES). (*b*) Myocardium: miliary tubercles are occasionally seen; tuberculous fibrous myocarditis is quite rare; caseous nodes are very uncommon and occur chiefly in the young; Anders in 1902 collected but 72 cases. (*c*) Endocardium: Weichselbaum, Birch-Hirschfeld, Klebs, Kotlar, and Chiari, have found tubercle bacilli in cardiac thrombi; Heller, Kundrat, Tripier, Londe, Petit, and Courmont, found them in endocarditic vegetations; Hanot holds that toxins cause the excrescences in which the bacilli are later deposited; Norris found endocarditis in only 1.4 per cent. of over 11,000 phthisical necropsies; tuberculous endocarditis is almost always vegetative and very rarely ulcerative or caseous; secondary infection is the cause of some cases of endocarditis which complicate tuberculosis. (*d*) Involvement of the vessels has been described under miliary tuberculosis. Primary disease of the vessels seems highly improbable, though some few cases are reported; 18 cases of tuberculosis of the aorta are mentioned in the literature collected by Simmitsky.

X. Tuberculosis of the Bones and Joints.

This topic properly belongs to surgery, but its relations to glandular, pulmonary, and other lesions demand some consideration. Koch was the first to demonstrate the almost invariably tuberculous nature of caries, fungus joint lesions, and tumor albus, and Damsch first determined this relation by inoculations. Schuchart, Krause, Demme and Renke proved that spina ventosa was usually tuberculosis. König and Orth found that about 80 per cent. of bone and joint tuberculosis was clearly secondary to pulmonary, glandular, genito-urinary, and other tuberculosis. Probably *all cases are secondary* to tuberculosis elsewhere. Infection is *hæmatogenic* in the majority of cases, far less often lymphogenic. *Trauma* has long been considered an important etiological factor, and experimentation shows that it may determine localization in animals already infected with the Bacillus tuberculosis. This factor has probably been overestimated, and Sprengel even maintains that the joints oftenest involved are those least subject to traumatism. Thirty-three per cent. of the cases occur in the first decade of life, and 50 per cent. in the first two decades. Tuberculosis in children involves the bones and joints in 22 per cent. of cases (Müller).

Localization.—Watson Cheyne, from his own and other cases, estimates that the knee is affected in 16.5 per cent. of cases, the hip in 14.6 per cent., tarsus and ankle in 14.4 per cent., elbow in 6.3 per cent., skull and face in 5.5 per cent., sternum, clavicle and ribs in 5.2 per cent., pelvic bones in 3.5 per cent., femur, fibula and tibia in 3.5 per cent., spine in 2.3 per cent., shoulder in 1.5 per cent., and scapula, ulna and radius in 1 per cent. Jaffe finds involvement of the spine in

20 per cent., foot in 21 per cent., hip in 13 per cent., knee in 10 per cent., hand in 9 per cent., and elbow in 4 per cent.

1. **Bones.**—Miliary tuberculosis affecting the bones produces no symptoms. The spongy ends (epiphyses) of the long bones are the particular seat of tuberculous osteomyelitis and periostitis, whence infection easily reaches the joints; the diaphyses and flat bones are seldom involved. Involvement of the short bones of the fingers and toes, and less often of the ulna, may cause bulging,—the *spina ventosa,* which is seen oftenest at about five years of age. In the bone-marrow there develops a grayish-red granuloma which dissolves the bone (*caries sicca*); the trabeculæ necrose and are discharged through fistulæ as sequesters or "bone sand," in a caseous, pus-like fluid. Even large foci may heal by granulating osteitis, in the same manner as tuberculous adenitis may heal. Infection may remain unobserved or semi-quiescent until aroused by such infections as pertussis or measles, or by trauma. *Cold abscesses* in the bone are lined by granulation tissue. There may be nocturnal pain, varying in intensity and sometimes radiating, stiffness, local tenderness, and fever. Bone lesions are not incompatible with seemingly perfect nutrition and otherwise robust health.

2. **Joints.**—Involvement may occur alone or follow contiguous osseous disease. Very often there is synovial inflammation and proliferation; far less frequently there are isolated tubercles in a non-inflamed synovial sac. Serous or serofibrinous synovitis (or the rice bodies, *corpora oryzoidea*) may result from toxins in a neighboring focus in the bone, without the actual presence of tubercle bacilli in the joint, exactly as toxic pleurisy or pericarditis may result from contiguous pulmonary or glandular foci. The *symptoms,* or onset, may be acute or insidious. Pain, especially at night, contractures, and fever, may be noted. In the later stages of joint disease, granulation tissue forms with large caseous deposits, hydrops, empyema articulorum, or the tumor albus (so named because of the thickening of the connective tissue, and the œdema and glistening appearance of the soft, periarticular structures). The joint is sometimes quite disorganized. Seventy-five per cent. recover under such conservative treatment as immobilization, iodoform or formalin injections, soap inunctions, cod-liver oil, and sea air.

Within a few years Poncet first, and then Leriche, Patel, Trebeneau, Bezancon, and Griffon, have called attention to a tuberculous pseudo-rheumatism, in which the incipient arthritis closely resembles genuine rheumatism (*rheumatisme tuberculeuse ankylosante*). It is thought by these French writers that the exuded leukocytes are strictly of the lymphocyte type; inoculations have proven positive in a number of cases, but toxæmia is the most probable cause (Mohr). It is most frequent in children. Lejars found one case of primary tuberculosis of the muscles.

XI. Tuberculosis of the Skin.

1. Lupus.—Lupus is the most frequent form. In 66 per cent. of the cases it is located on the nose, lips, and angle of the eye. It is often associated with tuberculosis elsewhere; Besnier found it with pulmonary tuberculosis in 21 per cent. and Leloir in 38 per cent.; Sachs and Bender found other tuberculosis in 62 per cent. and Block in 79 per cent. It is typical granulation tissue with true tuberculous tumors, which contain few bacilli because of the low temperature of the skin, and it often advances on one border while it cicatrizes on another. Its various forms, such as lupus maculosus, exulcerans, or hypertrophicans, need not be described in detail.

2. Scrofuloderma.—Scrofuloderma, the subcutaneous granuloma of Neisser, appears as movable, painless, subcutaneous nodes, which later become cold abscesses (the so-called scrofulous gumma, *gomme scrofuleuse* of Despres and Besnier); they discharge a milky fluid and leave a granulating ulcer, which is more sharply marked than that of lupus.

3. Lichen Scrofulosorum.—Lichen scrofulosorum is often but not always tuberculous; the eruption originates from the follicles, is constituted of thick, oily, epidermal cells which can be scratched off without causing bleeding, and is usually distributed in sharply marked groups of isolated yellow or yellow-brown nodules as large as a pinhead.

4. Tuberculosis Cutis Verrucosa.—Tuberculosis cutis verrucosa of Riehl and Pautauf is observed on the backs of the hands and fingers and in the interdigital folds, chiefly in butchers, cooks, and horsemen.

5. Postmortem Tubercles.—Postmortem tubercles were described by Verneuil, Verchère, Pick, and Pfeiffer. Laennec referred his case of pulmonary phthisis, though probably incorrectly, to a postmortem tubercle. Gerber suffered from tuberculosis of the axillary glands following a tubercle. They are observed in butchers, veterinary surgeons, and pathologists, especially in those beyond middle life.

6. Tuberculous Ulcers.—Tuberculous ulcers may develop from cuts by broken sputum cups; from bites of tuberculous subjects, ants, or flies; from burns, tattooing, skin grafting, or leeches; and from circumcision when the wound is sucked by tuberculous rabbis; lupus has followed vaccination.

Scrofula is tuberculosis; however, in descriptions dealing with scrofula as a separate clinical picture, affections of the mucous membranes are often associated with the skin lesions above described, with adenopathies (*q. v.*), and with bone or joint disease. Among these may be mentioned chronic ozæna, rhinitis, adenoids, friable carious teeth, otitis media, blepharitis, conjunctivitis or keratitis, particularly of the phlyctænular type, tracheobronchitis, intestinal catarrh, and leukorrhœa.

Treatment of Tuberculosis.

(*A*). **Prophylaxis.**—1. **Antibacillary.**—Antibacillary prevention concerns the etiological factors in the dissemination of the bacillus and of the elements of contagion. Cleanliness of the skin; disinfection of contaminated fæces, urine, or pus; confiscation of diseased meat; and regulation of dairies and inspection of cows are important; but the *destruction of infective sputum* is the prime indication. It is well to regard all sputum as dangerous, and to teach the public and patients this doctrine. Tuberculous, as well as pneumonic, grippal, and other sputa must be destroyed. An appeal must be made to the conscience or selfishness of tuberculous patients, setting forth the risks of autoreinfection. Patients should carry small spit cups, of which many varieties are made, and the sputum should be destroyed by burning or boiling before it dries. Spittoons, unæsthetic as they are, should be placed conveniently in the halls and wards of hospitals, sanitoria, factories, cars, and stations, either on the floor, or, better, somewhat elevated on brackets. The patient should sleep alone, and kissing must be interdicted. The bedclothes, linen, and eating utensils, should be carefully steamed or boiled. Rooms should be cleaned with moist cloths and should be swept with the windows and doors open. The danger of swallowing sputum must be clearly set forth, and the mouth should be washed out with some antiseptic solution before eating, though a few bacilli with the food are not very dangerous, as the gastric juice probably destroys them. The more minute details are as impossible to describe as those of consistent surgical asepsis, though they are equally logical and necessary. In Germany the reduction of mortality by 33 per cent. is clearly due to an awakened public intelligence.

2. **Governmental.**—Governmental prophylaxis, somewhat less promising in republican than in autocratic states, is important. It comprises: education of the public by publication of the dangers and preventive measures in tuberculosis, as in the recent excellent and plain pamphlets of the Illinois State Board; the establishment of sanitoria for the poor, among whom the mortality is four times, and the morbidity many times, greater than in the rich; the building of separate hospitals, or at least wards, for the hopelessly diseased; and the regulation of the air capacity of dwelling rooms in tenements, factories, or stores. Aside from great suffering and sorrow of thousands in every large community, there is an enormous economical waste in work and wages, an enormous cost in hospital care, and a great loss in live stock,—over 62,000 cattle and 56,000 hogs were condemned in Prussia alone in one year. The comparative sociological damage is apparent, as the ravages of tuberculosis involve the working years of life (15 to 60), while 94 per cent. of the deaths from diphtheria occur under ten years.

3. **Individual.**—Individual prophylaxis includes the increasing of physiological resistance, the maintenance of general health, and the

aiding of sound development in children. Weakly children should be brought up in the open air, judiciously fed, watched during acute infections, kept but few hours in school, sent into the country during vacation, taught moderation, and, later, should be informed as to the danger of alcoholism, sexual excesses, and infections.

(*B*). **Hygienic Treatment.**—The three great factors are *fresh air, proper food,* and *rest.* Bodington (1839), in England, treated cases by fresh air, but to Brehmer (1850), in Germany, is due the credit of soundly establishing the fresh-air treatment and the curability of phthisis. A few of his views are untenable, but many of them are the basis of the modern methods practised by McCormack, Driver, Detweiler, Trudeau, Knopf, and others. Patients are treated in sanitoria, and those at Nordrach, and Saranac, and many others, have given such excellent results that institutions of this class have increased; they number 54 in Germany, 30 in France, and are growing in number in other countries. Results depend on (*a*) the extent of the disease; (*b*) the condition of organs other than the lungs, as the pleura, intestines, larynx, and heart; (*c*) the social and financial status of the case. Detweiler states that 30 per cent. of his cases recovered and 40 per cent. improved; Trudeau brought about recovery in 31 per cent. of all, and in 66 per cent. of incipient, cases; Turban considers that 88 per cent. recover; Rumpf followed 97 per cent. of his cases: 70 per cent. of incipient cases were able to work—"the best test of a cure"; 55 per cent. of those in the second stage, and 23 per cent. of those in the third, recovered. One of the chief values of sanitorium treatment is that the patient learns the lesson of living properly, which too often in general practice is not sufficiently impressed upon him.

1. **Fresh Air.**—This is the chief hygienic factor in treatment, at home, in sanitoria, or in change of climate. (*a*) *Treatment at home,* for financial reasons, is especially important, as but 5 or 10 per cent. of tuberculous people are able to leave home. The patient should be carefully clothed and should recline in the sun with the windows of his room open, or should lie on a veranda. He should be outdoors for one-quarter to one-half the sunny hours, according to the season of year. At night the windows should be kept widely open. Rain, snow, dampness, and extreme cold, are no contra-indications, nor are fever, cough, or hæmoptysis; but wind, dust, and sudden variations in temperature, are to be avoided. Very thick clothing, which is so frequently observed among the poor, is unhygienic. Acute caseous and advanced types should generally be kept at home. (*b*) *Sanitorium treatment* offers the advantage of strict discipline, systematic living, and the constant presence of a physician who regulates the details of every-day life, gives explicit directions, and cheers the patient. Sanitoria should be located near, but well outside of, every large centre of population. (*c*) Change of *climate* was once considered absolutely indispensible; now it is said "to be not without influence." No climate is specific. *High altitudes* offer the advantage of purer air, stimulation of breathing, increase of vital lung capacity, increase of

the chest dimensions, growth of the muscles of inspiration, increase of metabolism, and stimulation of the appetite and blood-making organs. Colorado, Arizona, and New Mexico (four to seven thousand feet elevation), are excellent for cure and for opportunities for entering some permanent occupation after recovery; because of the emphysema which may be induced, a return to the sea level should be avoided. St. Moritz, Les Avants, and Davos (5,200 feet elevation) offer a dry, clear, cold, still atmosphere, where patients may reside in summer and winter; it resembles the climate of Colorado, Montana and northern Wyoming. There are also the Peruvian Andes and numerous other altitudes, for details of which see *Solly's Medical Climatology.* High altitudes are best adapted to suspected cases, to those with limited or incipient lesions, to those with slight cavity formation and little emaciation, or to more advanced but slowly progressing, or torpid, cases. Slight fever or slight hæmoptysis are not contra-indications to such climates. *Moderate altitudes* (2,000 to 2,500 feet) probably best serve the majority of cases, as Asheville (2,250 feet), the Adirondacks (2,000 feet), Aikin (2,250 feet), Summerville, Thomasville, etc. The climate of Minnesota, Nebraska and Dakota (1,000 feet elevation), is cool and dry; dryness is usually more desired than lack of variability. Moderate elevations do not induce emphysema, whence return to the sea level is easier. The *sea level* is the best location for the tuberculous aged, for quiescent cases with great cicatrization or extensive damage, for those with diabetes, albuminuria, nephritis, scrofula, bone disease, marked anæmia, repeated hæmoptysis, emphysema, or rapid softening, for those with incurable forms, for thin, nervous subjects, and for those with exophthalmic goitre. Sea voyages are always somewhat experimental, and Robin holds that but 8 per cent. of cases are benefited by them; those with incipient forms, or those with genito-urinary, glandular, or osseous disease, or with open wounds, are often helped. Moist warm climates (at the sea level) are especially adapted to laryngeal disease,—as the Bermudas, Florida, the Madeira or Canary Islands; patients with dyspepsia and diarrhœa do well in Corsica, Palermo, and Capri. Warm, dry climates, as southern California, Egypt, Algiers, or the Riviera, are good for catarrhal types. Climate alone, without proper rest, diet, and reasonable hygiene, rarely benefits.

2. **Nutrition.**—Practice differs; most physicians recommend 5 or 6 meals daily, but some get better results by giving but three meals, whereby the stomach is allowed some rest. Feeding varies with individuals as to the amount and as to the kind of food. A liberal mixed diet of meat, fatty meats, and fish, should be given; well-cooked leguminous vegetables (which are advantageously given in soups), butter, oil on salads, eggs, milk, cream, bread, pastries, and cakes, are the chief articles of diet. Anorexia is a common obstacle to full feeding. If it is of the nervous type, patients must be compelled to eat, but long hours in the fresh air usually stimulate the appetite. With habitually light eaters caution is necessary; beef juice, milk and

koumyss, should be given; the amount should be gradually increased and other foods should be added. Faradization of the epigastrium, cold compresses over the stomach, wine, vermouth, the fluid extract of condurango ♏ xv–xxx (Immermann), or strychnia gr. $\frac{1}{30}$ in solution, half an hour before meals, are valuable appetizers. Raw beef (zomotherapy), which is especially recommended by Richet and Hericourt, has lately come into use; fresh lean meat is cut, soaked for two hours in one-fifth its weight of water, is then pressed to extract the blood and fluid, and administered, 1 to 2 pounds for moderately sick cases to twice that amount for severe cases; it must be freshly prepared each time, as it soon becomes tainted. Raw eggs are often given between meals, beginning with 1 and increasing to 4, three times daily. Fever does not contra-indicate full- or overfeeding. Milk should be given, up to 1 or 1½ quarts daily; more than this amount promotes gastric atony; it is given to advantage with bread, crackers, or toast, in order to divide the curds which form; it may be peptonized, disguised with cocoa or cognac, or given by the rectum (*v.* GASTRIC ULCER); cream and butter are excellent substitutes for cod-liver oil, and buttermilk is indicated when the stomach is weak. Fats and carbohydrates are necessary to offset nitrogenous waste; the dark form of cod-liver oil (*v. i.*), lipanin (94 parts olive oil and 6 parts oleic acid), malt, honey, cocoa, nutrose, eucasin, and somatose, are valuable foods. In all forms of overfeeding (Debove's *suralimentation*) the tolerance of the stomach must be considered, for this is more important than the use of drugs (creosote, cough syrups, or cod-liver oil). Debove's method consists of gastric lavage followed by the introduction through the tube of 3 ounces of powdered meat, a quart of milk, and one or more eggs. Alcohol is at the present time administered less often and in smaller amounts than formerly, but is frequently indicated symptomatically (*v. i.*); small amounts of red wine or cognac and whiskey in egg-nogs are very valuable as a food and tonic. Tobacco should be interdicted; in special cases a cigar or two daily may be allowed, but the smoke must never be inhaled.

3. **Rest.**—Rest is one of the essentials of Detweiler's therapy. Exercise destroys tissue, often induces anæmia, and irritates the heart; it is distinctly contra-indicated by fever, emaciation, hæmoptysis, rapid pulse, nausea, and coughing. It has been well said that "too many cases of phthisis *walk* into their graves." Late in the treatment graduated exercise may be taken. Pulmonary gymnastics and pneumatic differentiation, as that by compressed air, are clearly injurious. Patients should lie in the bright sunshine and fresh air, and active mental occupation should be carefully avoided. Hardening and exercise should be obtained by quick, dry rubs with a large coarse towel, after which rapid partial, and then general, wet rubs may be instituted; cold rubs and hydrotherapy are to be shunned when there is any tendency to hæmoptysis.

(*C*). **Specifics.**—As yet there are no specifics for tuberculosis. Drugs are only of symptomatic value. Parenchymatous injections into the lungs, of carbolic acid, creosote, iodine, and iodoform, have been suggested by the surgical results attained in lesions of the bones and joints; but they are not without danger. Koch's original tuberculin, his later tuberculin TR, Maragliano's serum, and Behring's efforts to immunize cattle, have given no certain results.

(*D*). **Expectant or Symptomatic Treatment.**—**1. Fever.**—Rest, quiet, digestible food, and fresh air, or change of climate, are indicated in great preference to drugs. Brehmer administered strong wine at the time the fever began to rise; arsenic is recommended by Pollack and Powell, but no remedy in safe or harmless doses controls fever. Quinine disturbs the digestion. Sponging is fairly efficacious.

2. Cough.—A slight cough serves to remove secretion, but, if excessive, it begets coughing by congesting the lungs and tubes; the dangers of excessive coughing are exhaustion, depression, insomnia, aspiration, and vomiting, which necessitate interference. Fresh air, and avoidance of dirt, dust, and smoke, are most beneficial. Rest in general, and especially after eating, should be enforced. The cough should be suppressed, for it is certainly somewhat under the control of the will; the comment on the absence of coughing, especially at the table, is universal among observers of sanitorium methods; slow breathing, with the head slightly thrown back, moist compresses, or the sipping of warm water (with 10 grains of salt), often aid in its suppression. *Creosote* was discovered by Richenbach, in 1832, and was used by Addison, Sonnenberg, and Jaccoud; it is not specific, but often increases the appetite, modifies abundant secretion from the bronchi (by which the drug is in part eliminated), and relieves the cough. Administration in the fluid form is better than in capsules, which are often undigested. Beginning with one drop (well diluted) after meals, the dose may be increased to ten times that amount; but full dosage often irritates the stomach and kidneys and necessitates constant supervision of the urine; it should not be given in febrile cases nor in hæmoptysis.

Creosote may be administered in milk, in water with the compound tincture of gentian (℥iij), or in sherry wine.

℞

Creasoti	ʒiss.
Glycerini	℥ss.
Tr. gentianæ co.	q.s. ad ℥iv.

M. et S.—One teaspoonful after meals.

Creosote may be given in pill form as follows:

℞

Creasoti	ʒiss.
Mucilag. acaciæ	ʒiij.
Pulv. althææ	q.s.

M. et ft. pil. no. 100.
S.—One after meals.

Guaiacol carbonate (gr. v, *p. c.*) is often better tolerated by the stomach than creosote. *Cod-liver oil*, first used by Hughes-Bennett, is usually given in doses so large as to disturb gastric and intestinal digestion; the first dose should be 10 drops and the amount should be gradually increased. It is more valuable in tuberculosis of the glands and bones than in the other types. Eructations can be avoided by administering it one or two hours after meals, when the food begins to leave the stomach. The oil is readily absorbed, because of the bile which it contains, and is possibly somewhat alterative; but it is not very superior to butter or cream, and is really more a food than a medicine. Cod-liver oil may be floated on whiskey or milk, and gulped down. Its taste may be partly disguised by a "chaser" of whiskey, or by floating it in beer or porter. Syrup of orange disguises its taste fairly well. It is seldom given when there is fever or indigestion. It is prepared in large capsules, which obviate its unpleasant taste. *Opiates* may be used, such as paregoric; codeine gr. ¼ with aq. laurocerasi and glycerine āā ♏xv and syr. tolutani ʒj q.i.d.; dionin gr. ¼ q.i.d.; heroin gr. 1/20 q.i.d.; even morphine sulph. gr. ¼ with ac. hydrocyanici dil. ♏ii. Other remedies are myrtol gr. ii. A.M. and P.M.; terpin hydrate gr. ii to v, t.i.d.; ol. terebinthinæ, ♏ij and for unproductive coughs, spts. chloroformi or spts. ætheris co. ʒss in whiskey, p.r.n. (Various formulæ are given under BRONCHITIS.)

℞

Tr. belladonnæ	ʒss.
Spr. chloroformi	℥iss.
Mucilag. acaciæ	ʒj.
Aquæ	q.s. ad ℥iij.

M. et S.—One teaspoonful after meals, or as indicated.

Expectorants are to be avoided because of their nauseating tendencies. Tr. benzoin co., and creosote, may be vaporized. Menthol and guaiacol āā may be taken internally in olive oil. If the cough is nasopharyngeal, the mucosa should be painted with iodine, potas. iodide, and glycerine, in proportions of 1, 10, and 100; if laryngeal, 20 per cent. menthol should be applied locally (*v.* LARYNGEAL TUBERCULOSIS); if due to enlarged bronchial glands, inunctions of green soap and potas. iodide internally, should be given.

3. Sweats.—The room should be cool at night, the windows open, the bed not too heavily covered, and the body protected by a flannel night-gown. A glass of cold milk, with ʒij of cognac, given at bedtime, is often beneficial. Morphine on retiring is indicated when the sweats are associated with cough and insufficient oxygenation. Atropine gr. 1/100 to 1/60, and camphoric acid gr. xv to xxx, at bedtime are the most reliable remedies, and are superior to aromatic sulphuric acid ♏x to xx, agaricin gr. 1/16 to 1/12, tr. nucis vomicæ ♏xxx, picrotoxin gr. 1/60 and muscarin ♏v of a 1 per cent. solution.

℞

Quininæ sulph.	ʒss.
Calcii hypophosphitis	ʒj.
Extr. nucis vomicæ	gr. v.

M. et ft. pilulæ 20.
S.—One after meals.

4. **Hæmoptysis.**—Exertion, loud talking or singing, and excesses in alcohol, venery, coffee, or tobacco, must be shunned in all cases. It is well to warn patients of the possibility of hæmoptysis, in order to prevent undue excitement, if it appears. Effusive pleurisy should not be tapped in a subject who has recently bled from the lungs, as thoracocentesis has produced fatal hæmoptysis. The chief indication is absolute rest, physical and mental; the patient should be kept in the semirecumbent posture; silence on his part should be enforced; massage and stimulants are contra-indicated; no physical examination should be made except by auscultation, as percussion is obviously dangerous; and a hypodermic injection of morphine should be given to lessen cough, to quiet the heart, to allay the excitement attending most hemorrhages, and also possibly for its slight hæmostatic effect. As the tonus of the pulmonary vessels is beyond regulation, ergotin, tannic acid, and other hæmostatics, are useless, though digitalis and fresh suprarenal extract gr. ii to v sometimes seem efficacious. Salt ʒj is a popular remedy and is supposed to act by stimulating the vagus. An ice-bag should be placed over the heart and one over the lung, if rales are heard. Deep breathing seems to lessen bleeding. Ol. terebinthinæ ♏ij to iij is considered the best drug by Fraentzel. In severe or obstinately recurrent hemorrhage, bandaging of the extremities (Hippocrates), to lessen the return flow of blood, injections of gelatin, induction of pneumothorax by gas injection or puncture (Murphy, Cayley), or even emetics to stimulate the vagus (Fraenkel) may possibly be indicated. Rest and the administration of morphine, however, are usually effective.

5. **Pain.**—Pain may be pleuritic, and should be treated by adhesive straps or by painting with iodine; if muscular, it should be treated by codeine to relieve coughing, by liniments, massage, fomentations or phenacetin.

6. **Dyspnœa.**—This is not common. If due to stagnant secretion, moderate movement or change of posture aids in its evacuation; if asthmatic, iodide gr. iij after meals is indicated but it must be given with care lest it congest the lungs; if dyspnœa results from compression of the vagus by bronchial glands, iodide and green-soap inunctions should be given; if cardiac, alcohol or strychnia is indicated; if caused by pleuritis or pneumothorax, these affections should be treated.

7. **Cardiac Weakness.**—To relieve cardiac weakness, rest, an ice-bag over the precordia, cognac, champagne, camphor, or strychnia, are superior to digitalis or strophanthus.

8. **Insomnia.**—To relieve insomnia the patient should have sunlight, and also fresh air night and day; a light, early evening meal, a glass of beer, porter, or some whiskey on retiring, the bromides, codeine, morphine, and galvanization of the neck and head, are beneficial.

9. **Gastro-intestinal Symptoms.**—Nervous anorexia is relieved by fresh air, forced feeding, electricity, and guaiacol (*v.s.*). Dyspeptic

symptoms necessitate a scant diet for a few days, feeding by rectum, and poulticing of the epigastrium. In gastric atony, frequent meals of small volume, limitation of fluids, and strychnia, are indicated. For gas formation or pain the following are indicated: carbolic acid gr. j; resorcin gr. iij; fluidextract of gelsemium ♏ij; bismuth; extr. belladonna or spts. chloroformi ♏xx. Pyrosis may be corrected by sod. bicarb. and magnesia usta āā gr. xv to xxx. Hyperacidity should be treated by milk diet, belladonna and Carlsbad water or other alkalies; anacidity is relieved by easily digested foods, as somatose or eucasin; and subacidity by hydrochloric acid. *Vomiting* is caused by irritation of the pharynx (Lugol's solution locally, or gargles [1 to 10] of pot. bromide), the cough, overfeeding, or overmedication; small doses of carbolic acid (*v.* TYPHOID) are most successful. *Constipation* (*q. v.*) is treated by the usual measures. Diarrhœa may be dyspeptic, catarrhal, toxæmic, ulcerative, or amyloid; the following is indicated: Tannic acid gr. v; opium gr. j; lead acetate gr. ij; camphor gr. ij; tannigen gr. vij to xv, or bismuth ʒj (see TYPHOID).

10. Anæmia.—This is benefited by fresh air, change to a high altitude, full diet, iron in small doses, and arsenic.

11. Sexual Symptoms.—Intercourse must be forbidden in incipient as well as in advanced cases. Two or three years after recovery marriage may be sometimes allowed; it is generally less injurious to men than to women. Tuberculous parents may rear healthy children, but tuberculosis and scrofula are rather common in such families. In 66 per cent. of pregnant tuberculous women the prognosis is bad, and laryngeal tuberculosis is almost invariably fatal within a short time. In severe cases abortion may be contemplated; opinions vary as to its indication, for some contend that the mother runs the greater risk and others maintain that abortion has no appreciable effect on the tuberculous process; Kaminer found that in 70 per cent. of the cases abortion does not alter the clinical course.

12. Treatment of Other Complications.—The treatment of laryngeal, pleural, peritoneal, intestinal, glandular, and meningeal complications (*v. s.*) is considered elsewhere.

LEPROSY.

Definition.—A chronic incurable contagious disease, caused by the Bacillus lepræ and characterized clinically by nodules, which belong to the infective granulomata (Lepra tuberosa), and are observed externally on the skin and nasal and oral mucosæ, and internally in many organs; or by a perineuritis leprosa, which causes sensory, motor, and other symptoms (Lepra maculoanæsthesia). The nodular and anæsthetic forms usually co-exist in some measure, though in varying degree.

Distribution.—The home of leprosy is Egypt, where it existed 2,400 years B.C. (Brugsch) or even 4,620 B.C. (Engel-Bey). The leprosy of Leviticus probably includes other diseases. It prevailed in India 700, and in China 400, years B.C. Just before the Christian era, Pompey's soldiers brought leprosy to Italy, whence it spread over Europe, and the Crusaders at the end of the thirteenth century further disseminated the disease. In Europe there were 20,000 asylums, but some cases were probably syphilis; the disease has steadily decreased since the sixteenth century. It is estimated that at present there are 300,000 cases in the world. In Europe cases are found in Sweden, Norway, Finland, Russia, Dalmatia, Bosnia, Montenegro, Germany, France, Italy, Spain, Portugal, Turkey, Greece, and in some Mediterranean islands; Arning estimates that there are 3,000 cases in the west half of Europe. In Asia numerous cases are found in India (5 cases to each 10,000 of population), China, Malay Archipelago, Formosa, Corea, Persia, Turkestan, Syria, and Arabia. In Africa it prevails in Egypt along the Nile; in Tripoli, Tunis, Algiers, Mozambique, Zanzibar, Madigascar, and in the Canaries and Azores. In Japan, Australia, Oceanica, and Sandwich Islands, it is also found. In America foci exist in Canada (New Brunswick, Nova Scotia, and British Columbia), in the United States (California, Louisiana, Minnesota, North Dakota, Florida); in Mexico, West Indies, Honduras, Columbia (5 per cent. of the population), Brazil and Paraguay.

Bacteriology.—The *Bacillus lepræ* was found by Hansen (1871) and was fully described by Neisser (1879). It is a fine, narrow rod, measuring ½ to ¾ of a red cell, and closely resembles the tubercle bacillus (*q. v.*) morphologically and in its staining properties; both bacilli contain fat. Inoculation is the surest differential criterion between the two, for the lepra bacillus is not inoculable. When in the internal viscera it may exceed the dimensions which it attains in the skin and mucosæ five or six times (Cornil). It is motile and cannot be cultivated. It is found in the typical nodes and infiltrations of the skin and superior mucosæ, in the fresh maculæ and in recent nerve disease, in the sweat, in the sebaceous and hair-glands, and it is free in their lumen. It also exists in the nerves and Pacinian bodies of the skin; in the brain, cord, eye, lungs, liver, spleen, intestine, and kidney; in the salivary, pancreatic, adrenal, and lymphatic glands; in the larynx, testis, epididymis, tubes, ovaries, and bloodvessels; in the blood during fever (metastatic distribution), and in the nasal, dermal, fæcal, lachrymal, bronchial, vaginal, urethral, and mammary secretions and excretions. It is not known why the bacillus is found so much more frequently and persistently in the nodular and mixed types than in the nervous forms; Stricker found it in the nasal secretion in 96 per cent. of the nodular, 96 per cent. of the mixed, and in 66 per cent. of the nervous, type. It has never been identified outside of the human body or of its secretions and excretions. It corresponds to but one of Koch's criteria; *i.e.*, it is always present in leprosy; it cannot, however, be cultivated, and the value of successful inoculations on criminals under sentence of death has been justly criticized, for these cases developed in a leprous community. That *leprosy is* *contagious* can no longer be doubted, as shown by its increase where no restriction is enforced, by its decrease on segregation, by its development in two-thirds of the (colored) attendants in lazarettos, and by other factors. It is less communicable than syphilis or tuberculosis and is promoted only by long and intimate contact with lepers. The nodular is far more dangerous than the anæsthetic type. Infection may be direct or indirect (by infected linen, utensils, etc.). It occurs chiefly though the skin and nose, and possibly also through the genitalia. Vaccination has probably never caused it. Extension occurs more by the lymphatics than by the bloodvessels. Hereditary transmission is improbable; a predisposition toward leprosy may possibly be inherited, but not the disease, and only 4 per cent. of children of leprous parents acquire it. More cases occur in colored than in white races, more in men than in women, and more in the poorer than in the upper and middle classes. It most frequently occurs between the ages of fifteen and thirty, and rarely before the third year.

The *incubation* is long, at least three to five, and sometimes ten, twenty, or even thirty-two, years.

Symptoms.—The chief clinical groups do not include the visceral involvement which is seen in every type; the main types are nearly always somewhat blended.

I. Nodular Type (*Lepra tuberosa seu tuberculosa*).—The prodromal toxæmic symptoms indicate an infection which has already been established, but has escaped recognition; they are depression, coldness, formication, sweats, pains, vertigo, digestive disturbance, temperature, and sometimes nasal irritation. After months, or years, *maculæ* appear on the face and extremities, which may itch or may be hyperæsthetic; they may disappear, leave pigmentation, or metamorphose into *nodules*, which are present in 75 per cent. of all cases. The nodule, or leproma, is a granuloma and is very similar to the tubercle or gumma; the nodes are highly vascular and are formed of a scanty fibrillar reticulum, in which lie emigrated leukocytes, small round epithelioid cells, and the larger multinucleated lepra cells of Virchow, which contain close clumps of bacilli. The nodes by fusion form *infiltrations* which are gelatinous, glistening, and yellow-red. The nodes vary in size from that of a millet seed, to that of a hazelnut or walnut; they lie in the skin, are dark red, and later become yellow-brown; they are glistening, vascular, slightly desquamating, and at first soft but later become harder. *Sensation* is always *disturbed* in their vicinity. They are most conspicuous on the face and hands, but also appear on the extensor surface of the knee, elbow, and fingers, and very seldom on the soles of the feet, the palms of the hands, the scalp, or penis. When they develop on the face, the forehead, nose, cheeks, chin, lips, and ears, are thickened, the nose becomes flatter, the eyebrows, lashes and the beard are shed. The eyes are involved in over 90 per cent. of the cases; the expression is leonine (*leontiasis*), or, as the ancients called it, faun-like (*satyriasis*). The nodes usually grow and extend slowly, and the lymphatic vessels and glands, of the neck and groin especially, become enlarged and tender. Sometimes the progression is interruptedly acute, with intermittent fever and an eruption resembling erysipelas. They may shrink, but more often *necrose,* soften, and ulcerate. At the same time or later, nodules which are prone to early ulceration and infiltration, occlude the *nose,* roughen the voice (*raucedo leprosa*), distort the larynx and throat, impair mastication, swallowing, or respiration, and even provoke suffocation. Cicatrization of the throat or face causes added *deformity.*

II. Nervous Type (*Lepra nervorum, anæsthetica, maculo-anæsthetica, seu mutilans*).—The most common prodromes are fever and such nervous symptoms as rheumatic pains, neuralgia, paræsthesia, hyperæsthesia, and vasomotor cerebral congestion. *Maculæ* develop on the face, neck, trunk, or limbs; they are pale or dark-red, flat or slightly raised, growing peripherally and paling centrally, and are glistening or present a powdered aspect. Histologically they are identical with the nodules, but contain fewer bacilli, are flatter, contain no large cells, and tend more toward fibrous change. Their distribution is asymmetrical, like that of the underlying nerve lesions; they are often oversensitive, sometimes pigmented (*lepra nigra*), or leukoderma-like (*lepra alba*). Early vesicles sometimes develop. If the skin is powdered with dry methyl violet and if pilocarpine is then given, the sound tissue sweats and stains, while the maculæ remain uncolored because they do not sweat.

Sensory disturbances are due to *lesions* in the *nerve trunks,* which are the seat of a perineuritis and interstitial neuritis which slowly compress the nerve fibers. The connective tissue development and the presence of the bacilli differentiates this from the parenchymatous neuritides. Unlike the ordinary neuritis, the leprous neuritis affects the *sensory more than* the *motor filaments.* There is an irregular dissociated anæsthesia in which the perception of pain and temperature is usually more dulled than other forms of sensation, and this occurs mostly in the peripheral branches in the arms and legs. Sensation may be perverted or retarded, and anæsthesia may come on suddenly, which is considered rather characteristic. Pain may be slight, or agonizing as in a case of ten years duration which was personally observed. The *nerve trunks are palpable* and are tender in many cases; the ulnar nerve is most markedly enlarged and in the case cited was thicker than a lead pencil, but the auricular nerve is more often felt (in 90 per cent., Baelz). Motor weakness and atrophy are less common and are rarely extreme; the eyelids, muscles of the mouth, and sometimes the masticatory and ocular muscles, and also the hands and feet, are paretic. The ulnar and peroneal nerves are sometimes paralyzed, causing the "claw-hand" and "claw-foot." Facial paralysis occurs in 9 per cent. of cases. The tendon reflexes are often increased, though sometimes normal or decreased; there is often fibrillation and increased myotatic irritability. Trophic changes are less common and include œdema, glossy skin, "*mal perforant*" (26 per cent.), loss of the hair or nails, dry gangrene, bone resorption, rheumatoid swellings, ankylosis, and pigmentation. Great *trophic* changes mark the *lepra mutilans.*

III. Mixed Type.—This is a combination of the nodose and anæsthetic types. *In all developed forms the internal tissues are invaded:* the liver, spleen, and lymph glands, are enlarged; the lungs show peribronchial foci, cavities, or induration; and the intestine is infiltrated. In both sexes the sexual functions are decreased and later are entirely lost. Infantilism results in cases developing before puberty. Cachexia develops, the patient becomes very sensitive to cold, the temperature, except for intermittent rises, becomes depressed, heart weakness develops, and a degree of apathy is noted. Abortive or long localized forms are described.

Course and Prognosis.—The course is slow and progressive, the outcome invariably fatal. The nodular type covers eight to twelve years, the anæsthetic form fifteen to twenty years, but either variety may endure six or seven decades. According to Hillis, 38 per cent. of cases die of leprosy (exhaustion, diarrhœa, cardiac weakness), 22 per cent. of chronic nephritis, 17 per cent. of lung disease, which is more often leprous than tuberculous, and 10 per cent. of diarrhœa (leprous infiltration of the gut). The remaining 13 per cent. die of erysipelas, sepsis, amyloidosis, and intercurrent diseases, for lepers possess a decidedly reduced resistance.

Diagnosis.—The diagnosis is based on the history of residence and of exposure (which is often suppressed by the patient), on the macules or leukoderma (which are found in 90 per cent.), on the anæsthesia, amyotrophy, and mutilation, and on the finding of the lepra bacillus (necessary for exactness), for which the nasal secretion, nodes, and macules, should be examined (*v.* SYRINGOMYELIA).

Treatment.—**I. Prophylaxis.**—All suspicious cases should be registered and all clear cases should be sequestrated.

II. Therapy.—Drugs are useless, though a few are helpful. (*a*) Gurjun oil ʒss to ʒij in water or lime water; (*b*) chaulmoogra oil, in milk, beginning with a few minims and increasing to ʒj–iij; (*c*) salves of 5 to 10 per cent. pyrogallol, 10 per cent. chrysarobin, 10 per cent. ichthyol; (*d*) Calamette's antivenin, 20 to 30 c.c., which was suggested by cases which improved after snake bite; (*e*) arsenic, salicylic acid and corrosive sublimate; these are the most extensively used remedies. Symptomatic treatment of pain, ulcers, etc., is also indicated.

INFECTIONS OF UNCERTAIN ORIGIN.

(A). FEBRICULA.

Definition.—A fever of short duration, due to a number of causes. If only of twenty-four hours duration it is called *ephemeral fever;* when it lasts a few days, it is called *febricula;* when attended by herpes, *febris herpetica;* or when accompanied by gastric disorder, *febris gastrica.*

Febricula is no morbid entity, and its varied *causes* are not exactly known. It embraces (*a*) benign or abortive forms of various acute infections, such as rheumatism, typhoid, scarlatina, tonsillitis, and pneumonia; and (*b*) febrile dyspepsias, especially those in children. Gastric fever is a term which was formerly applied to febrile gastritis, which is possibly produced by toxins in the food. Sewer-gas is popularly held as the cause of fugitive fever with gastro-intestinal disturbance, but Abbott and others have justly contested its etiological significance. Exposure to cold is also a possible factor.

Symptoms.—The three common symptoms are (*a*) chill or chilliness; (*b*) sudden rise of temperature; (*c*) a critical resolution, more often prolonged than acute. The onset is abrupt, usually without prodromes. The fever rises quickly to 101°–103°, or higher in the young, with the usual signs of toxæmia (headache, coated tongue, anorexia, and febrile urine). Herpes is quite frequent. Bronchitis, acute splenic tumor, albuminuria, and, in children, cerebral toxæmia, are sometimes present. The term ephemeral is rather inappropriate, as the average duration is three days, with a slow crisis.

Diagnosis and Treatment.—The diagnosis is solely by exclusion, and the treatment is expectant. Rest in bed is advisable as a precaution against some longer and insidious malady, such as typhoid; the bowels should be unloaded, the diet restricted, and a drop or two of tincture of aconite and spts. ætheris nitrosi ʒss should be given every three or four hours.

(B). GLANDULAR FEVER.

Definition.—An acute infection observed in children and characterized by pharyngeal injection, a short high fever, and a painful intumescence of the cervical lymph glands. It was described as *Drüsen fieber* by Pfeiffer, in 1889. Its *etiology* is unknown but it is evidently an infection, as suggested by the clinical signs and by its frequent occurrence in epidemics. It is usually seen in children between five and eight years of age, and during the colder months. West observed an epidemic of nearly 100 cases in Bellaire, Ohio.

Symptoms.—The symptoms begin without prodromes, with fever of 100° to 103°, pain in the head and neck, redness of the throat, some dysphagia, often pain in the limbs, nausea, vomiting, and abdominal pain. On the second or third day the cervical lymph glands become swollen and tender, but seldom cause redness of the skin. The glands back of the sternomastoid are most involved. The inguinal and axillary glands are involved in 75 per cent. of cases, and the mesenteric in about 40 per cent. The glands may reach the size of an egg. After ten to twenty days the swelling subsides, having lasted longer than the fever. In some instances a persistent dry cough suggests involvement of the bronchial glands. The spleen is enlarged in 60 per cent. of cases, and the liver in 90 per cent. The fever lasts but a few days. Complications are rare. Suppuration in the glands is exceptional. Acute nephritis and otitis media are occasionally observed.

Prognosis and Treatment.—The prognosis is good, though a relapse may occur. The absence of a rash excludes the adenopathies of varicella, scarlatina, and measles, and the generalized glandular swelling excludes the local swelling due to simple pharyngitis. The treatment is expectant.

(C). MILIARY FEVER.

Definition.—An acute infection, probably microbic, marked by fever, profuse sweating, a miliary eruption, nervous symptoms, palpitation, and precordial anxiety. It first appeared in England after the Battle of Bosworth (1486) and broke out several times in the following century. It was known as the "English sweats" (*sudor Anglicus*), and its mortality reached even 50 to 80 per cent. In 1529 it caused 3,000 deaths in Dantzig. There have been about 200 epidemics between the epidemic in Picardy, in 1718, and that in Poitou, in 1887. It is apparently highly contagious (Brouardel). Epidemics have also occurred in Italy, Switzerland, Germany, and Austria, with a death-rate of 5 to 25 per cent. Fortunately they last only a week or month.

Symptoms and Treatment.—In mild outbreaks the chief symptoms are fever, profuse periodic sweating, and miliary eruptions. The eruption may be erythematous or morbilliform (miliaria rubra), or there may be no redness of the skin (miliaria alba s. crystallina); the spleen may be enlarged. In severe cases there are marked cerebral toxæmia, rapid pulse, precordial distress, palpitation, extreme prostration, and hemorrhages into the skin; and death results in a few hours or days. Its average duration is one to two weeks. Desquamation is frequent and convalescence rather tardy. The treatment is supportive and expectant.

(D). WEIL'S DISEASE.—(ACUTE FEBRILE JAUNDICE.)

Definition.—An acute infection, characterized by fever, jaundice, herpes, enlarged spleen and liver, sore throat, muscle pains, and albuminuria. It was especially described by Weil (1886), whose name it most often bears, though it was previously mentioned by Mathieu, Bouchard, Lancereaux, and Coynes. Nicolaysen describes 63 epidemics of icterus in Norway and Sweden in the last fourteen years; 3 to 28 per cent. of the inhabitants in certain communities acquired the disease.

Etiology.—Its etiology is unknown, whence our difficulty in stating whether it is a morbid entity or whether it includes several diseases. It occurs largely in men between twenty-five and forty, especially in butchers and alcoholics; it also occurs in the warmer seasons and sometimes in epidemics. The symptoms mentioned above, those described below, and the autopsy findings of granulo-fatty changes and ecchymoses, mark it as an infection. It is either directly microbic or toxæmic. Bordoni, Uffreduzzi and later Jaeger found the Proteus fluorescens, and Freund a diplococcus, but the *intravitam* findings are usually negative. It is not typhoid, recurrent fever, sepsis, malaria, or phosphorus poisoning, with which it has been sometimes identified. Some cases have occurred in this country (Lanphear, Musser, Guiteras); the author has seen eight cases in Chicago.

Symptoms.—The symptoms begin suddenly without prodromata, with fever, chill or chilliness, headache, vertigo, insomnia, and severe *pains* in the limbs and extremities. The *fever* may reach 104°, 105° or 107°; it afterward shows remissions, and falls either by crisis or by lysis after five to eight days. The nervous symptoms may include delirium, sopor, or even coma. *Jaundice* develops on about the third day, is probably due to catarrhal radicular angiocholitis, and may be slight or extreme; bile usually appears in the urine, and the stools are often acholic, though the author has twice seen deep jaundice without obstruction of the bile-duct. *Muscular pains* are very common and intense, especially in the calves, which are usually also tender. Articular pains are not uncommon. *Herpes* is very common. The *rapid pulse* is very marked compared with the usual slow pulse of simple catarrhal icterus. In one case the author observed blood-stained bronchitic sputum. Digestive symptoms are common, such as furred tongue, nausea, vomiting, epigastric pain, abdominal distention, and diarrhœa, with acholic or bile-stained stools. The *liver* is enlarged and tender in more than 50 per cent. of the cases, the *spleen* in over 66 per cent., and the *lymph-nodes* are frequently affected. The urine is albuminous in over 50 per cent.; acute nephritis is frequent; hæmaturia occurs occasionally, and uræmia exceptionally; the urea and the amount of urine are decreased during, and increased after, the seizure (Chauffard's nitrogenous and polyuric crises); glycosuria developed in one fatal case.

Diagnosis.—Weil's disease must be differentiated from recurrent fever by its spirillum, and from sepsis and typhoid,—in regard to the latter of which it may be noted that icterus may produce a weak Widal reaction. Differentiation is more difficult from suppurative angiocholitis (*q. v.*) and acute yellow atrophy (*q. v.*).

Prognosis.—This is usually good and convalescence is generally established in two weeks; in one epidemic, 11 per cent. of the cases died. Recurrence may be anticipated if the splenic tumor has not subsided (Mathieu); renal or hepatic insufficiency, pregnancy, hæmolysis with epistaxis and hæmatemesis, render the prognosis less favorable.

Treatment.—The treatment is directed to (*a*) the intoxication, for which calomel and salines are indicated; cold water enemata (Krull and Chauffard) increase the renal flow, and, with it, the excretion of toxins; (*b*) the jaundice, for which salines should be given to lessen duodenal catarrh; (*c*) the albuminuria, for which a milk diet is indicated; (*d*) muscular pains, for which heat, and massage with chloroform liniment, are most beneficial.

(E). MILK SICKNESS.

Definition.—A disease acquired by man from the milk or meat of cattle suffering from the "trembles." Cows with the disease, after being driven too rapidly, suffer from anorexia, injection of the eyes, vertigo, trembling, and convulsions ending in death. Young horses and sheep may also acquire the disease. Milk sickness occurs chiefly in newly settled countries, as in the Southwestern states, and it is still seen in North Carolina. Its origin and pathology are unknown.

Symptoms.—In man the nervous symptoms are frequently conspicuous, as restlessness, convulsions, or coma, which appear after a few days of malaise. The tongue is swollen and heavily coated, the breath fœtid, the stomach painful and irritable, the fever high and sometimes typhoidal. The issue is either fatal, in acute forms, within a few days, or recovery may result after weeks. Flesh, butter, cheese and milk, from infected animals may, even when boiled, cause death when given to dogs.

(F). MOUNTAIN FEVER.

(*a*) Some European cases are due to the anchylostoma and are associated with anæmia. (*b*) Many cases are identical with typhoid (Woodruff, Smart, Hoff, and Raymond). (*c*) Wilson and Chowning (1902) think that the disease is caused by a red-corpuscle protozoön like that of Texas fever and malaria, though not pigmented; Stiles and Craig could not confirm their findings. Cases develop in the early spring, and are marked by chill, fever of one to two weeks, rapid pulse, epistaxis, albuminuria, rapid breathing, bronchitis, splenic tumor, rapid anæmia, moderate leukocytosis, slight icterus, muscular pains, and widely diffused macular rash,—which develops on the third day, disappears on pressure at first, but afterwards persists on pressure. The liver and spleen are enlarged. Quinine may be beneficial in some cases. The death rate may be 70 per cent. (*d*) In high altitudes slight fever, associated with headache, dyspnœa, thirst, and malaise, is known as mountain sickness.

(G). FOOT AND MOUTH DISEASE.

Definition.—Epidemic stomatitis, Aphthous fever, or Aphthæ epizooticæ is one of the zoönoses and its nature is unknown. Loeffler and Frosch have shown that the virus is ultramicroscopic, as it passes in full virulence through porcelain filters. The disease, first described by von Valentini, in 1695, and known, since the middle of the fifteenth century, chiefly affects cattle, sheep, and pigs, but sometimes also goats, horses, dogs, and even fowls. *In cattle* there is fever, swelling of the mouth, salivation, and the formation of small, clear vesicles on the tongue, gums, lips, and udders. These vesicles soon become opaque, and bursting, form small ulcers; emaciation is rapid, and the milk becomes yellowish and mucoid. Recovery is usual in one to two weeks. The disease spreads very rapidly. It is observed in Hungary, Germany, France, Switzerland, England, and occasionally in this country.

Etiology and Symptoms.—*In man* infection occurs from diseased milk (as proven on himself by Hertwig), butter, and cheese, but not by the meat of the diseased animal. Open wounds may be directly infected. Cattle have been inoculated from man. The incubation is three to five days. There are fever, salivation, aphthous ulcers, angina, nausea, vomiting, and vesicles on the skin—which have caused confusion with vaccinia. Hemorrhages occur in severe epidemics. Ebstein has suggested that Foot and Mouth Disease may be kindred with the ordinary aphthous stomatitis of nurslings, or pregnant women. Children are more endangered than adults. The death-rate is 2.3 per cent. (Hulin) to 8 per cent. (Siegel). Recovery usually occurs in two to three weeks.

Treatment.—Prophylaxis concerns the boiling of suspected milk, the segregation of diseased animals or human subjects, and vaccination, as discovered by Loeffler. A dram of 5 per cent. solution of potas. chlorate, t. i. d., should be given, and the aphthæ should be touched with borax or silver.

SECTION II.

DISEASES OF THE CIRCULATION.

DISEASES OF THE PERICARDIUM.

(A). PERICARDITIS.

Definition.—An inflammation of the pericardium, usually secondary to infectious diseases, to inflammation in contiguous tissues, to other diseases of the pericardium, or to dyscrasiæ; it is fibrinous or plastic, exudative, or adhesive.

Etiology.—The disease is rarely primary, and final analysis usually demonstrates that the so-called primary cases are tuberculous, rheumatic, or septic. Cold, exposure, and trauma, probably only reduce the physiological resistance of the tissues to bacterial invasion. Pericarditis is usually mycotic, the organisms of suppuration, pneumonia and tuberculosis, being most frequently found; it seems, however, that toxins alone may excite inflammation.

Pericarditis is almost invariably *secondary* (*a*) to *infectious diseases*, of which rheumatism (14, 37, 66, per cent.) is foremost, and it is most frequent when many joints are involved in rapid succession. Pericarditis may be the only manifestation of rheumatism. Many "primary"cases are probably rheumatic. It occurs with recurrent rheumatism, with endocarditis, and sometimes with chorea. It usually develops rather early, within half a week to two weeks, but may precede arthritis, especially in children. It is commonly serofibrinous, most exceptionally purulent.

It is common in pneumonia, septicopyæmia, puerperal fever, ulcerative endocarditis, navel infection in babes, osteomyelitis, scarlatina, and more rarely in other exanthemata, and in tuberculosis (pulmonary, multiple serous inflammations, pleuritis, peritonitis). In children, scarlatina and rheumatism are most important.

(*b*) It is secondary, *by contiguity*, to adjacent affected organs; to pleurisy, pneumonia, and aneurysm; to disease of the ribs, sternum, bronchial glands, spine, œsophagus, stomach (ulcer and cancer), spleen, liver (abscess), peritoneum, myocardium, and endocardium; and to cervical cellulitis (diphtheria, mediastinitis). Trauma of these parts, from without or within, such as penetration of foreign bodies, may be included under this class.

(*c*) It is secondary to *certain diseases of the pericardium*, as tuberculosis, carcinoma, and other processes. In 30 per cent. of Matter's "cryptogenetic" cases, tuberculosis was found at autopsy.

(*d*) It is secondary to *cachexiæ or dyscrasiæ*, gout, diabetes, carcinoma, to nephritis (3 to 10 per cent. of cases, especially those of contracted kidney), to alcoholism, blood diseases, and scurvy; and often, as a terminal infection, to reduced physiological resistance, in which case it may easily escape recognition clinically. This class is usually found in adults. Though encountered at any age from fœtal life to old age, it is most frequent between the fifteenth and thirtieth years.

Fibrinous (Plastic) Pericarditis.

A strict division into dry (plastic) and exudative (effusive) pericarditis is impossible, because the forms are very often combined. Dry (plastic, fibrinous) pericarditis is *circumscribed* over the base and large vessels, or is *diffuse*, and consists pathologically of vascular injection, punctate ecchymoses, loss of the smooth, glistening, mirror-like appearance of the serous membrane, and exudation of plastic lymph in small particles or irregular lamellæ, which may appear smeared as with butter, spongy, shaggy, or hairy (*cor villosum*). Some fluid is usually found in the meshes of fibrin. Stripping off of the fibrin may reveal tubercles. The subjacent myocardium often suffers infiltration and degeneration in the more intense or protracted cases.

Symptoms.—Symptoms are lacking in most cases; pain is inconstant, and, when present, is by no means characteristic.

Physical Findings.—Inspection and percussion are usually negative. The writer has seen a strong apex beat where the autopsy revealed a fibrin deposit which was an inch in thickness. Palpation may disclose a friction fremitus, usually over the sternum, which may be obtained as frequently as is friction on *auscultation* (*described under Pericarditis with Effusion, to which refer also for diagnosis, prognosis, and treatment*).

Pericarditis with Effusion.

This form has been called the second stage, the fibrinous or dry form being the first stage. The fluid may be *serous* with much or little cellular or fibrinous admixture (sero-fibrinous); it may be *hemorrhagic*, this type being observed in the aged, in the dyscrasiæ (such as those having scurvy or cancer), in cases of tuberculosis, in those having purpura, hemorrhagic exanthemata, or Bright's disease; the exudation may be tinged with fresh or with darker altered blood; it may be *purulent*, and is then usually sero-fibrinous rather than purely purulent, and is often associated with pyæmia or contiguous suppuration; it may be *putrid*, when caused by pyæmia, carcinoma of the œsophagus or stomach, or by lung cavities. (Sixty-seven per cent. of pericarditis cases are sero-fibrinous, 19 per cent. are hemorrhagic, and 14 per cent. are purulent [Breitung].) The pathological sequences of pericarditis are fatty and cloudy degeneration, inflammatory infiltration, or actual exulceration of the heart muscle; rupture of a purulent exudation externally into the tissues of the chest, or possibly into neighboring organs or cavities; inflammatory extension outside of the pericardium (*pericarditis externa*) and thence to other tissues, such as the mediastinum or pleura; formation of polypi, which, becoming detached, may, in rare cases, produce foreign bodies in the pericardium; complete resorption of the exudate and *restitutio ad integrum*, which is a rather uncommon issue; the formation of hard plaques, *maculæ tendineæ*, which are usually not inflammatory in origin; organization of connective tissue, either as local adhesions, which are mostly observed near the fixed base of the heart, because the heart's movements probably detach apical adhesions, or as general obliteration of the sac (*concretio pericardii*); and, finally, desiccation of the exudate, leaving a cheesy mass which may become tuberculous or may calcify. Coincident endocarditis is less a sequence than an associate or cause of pericarditis.

Symptoms.—All symptoms are frequently absent (Latham, Graves, Stokes), and no symptom is diagnostic. Leudet found total latency of symptoms in 55 per cent. of his cases. Daily examination of the heart in those diseases which are most likely to cause pericarditis is the only certain method of avoiding diagnostic errors. *Fever* may exist from the disease, or from the causal affection, but is irregular (Wunderlich). It is often absent, especially in secondary pericarditis, and the temperature may be subnormal, even in purulent effusions. High fever is infrequent even in rheumatic cases.

Dyspnœa, or thoracic oppression occur. Oppolzer observed cyanosis without dyspnœa when the pulmonary circuit remained intact. Dyspnœa is due largely to heart compression by the exudate or extension of inflammation to the myocardium, and is less frequently due to vagus irritation, phrenic-nerve inflammation, venous, pulmonary, and arterial compression. It is remarkable that patients may sometimes walk into the hospital with enormous pericardial effusions. The respirations are increased.

Pain is variable, is often absent (in secondary pericarditis), and is said to be more common in small effusions. Severe pain oftener indicates pleurisy than pericarditis (Bouillaud). Epigastric pain may precede pain elsewhere (Guéneau de Mussy). Anginal pain (Andral), radiating to the shoulder, arm, and neck, is rare. Precordial tenderness is at times present, and palpitation is common.

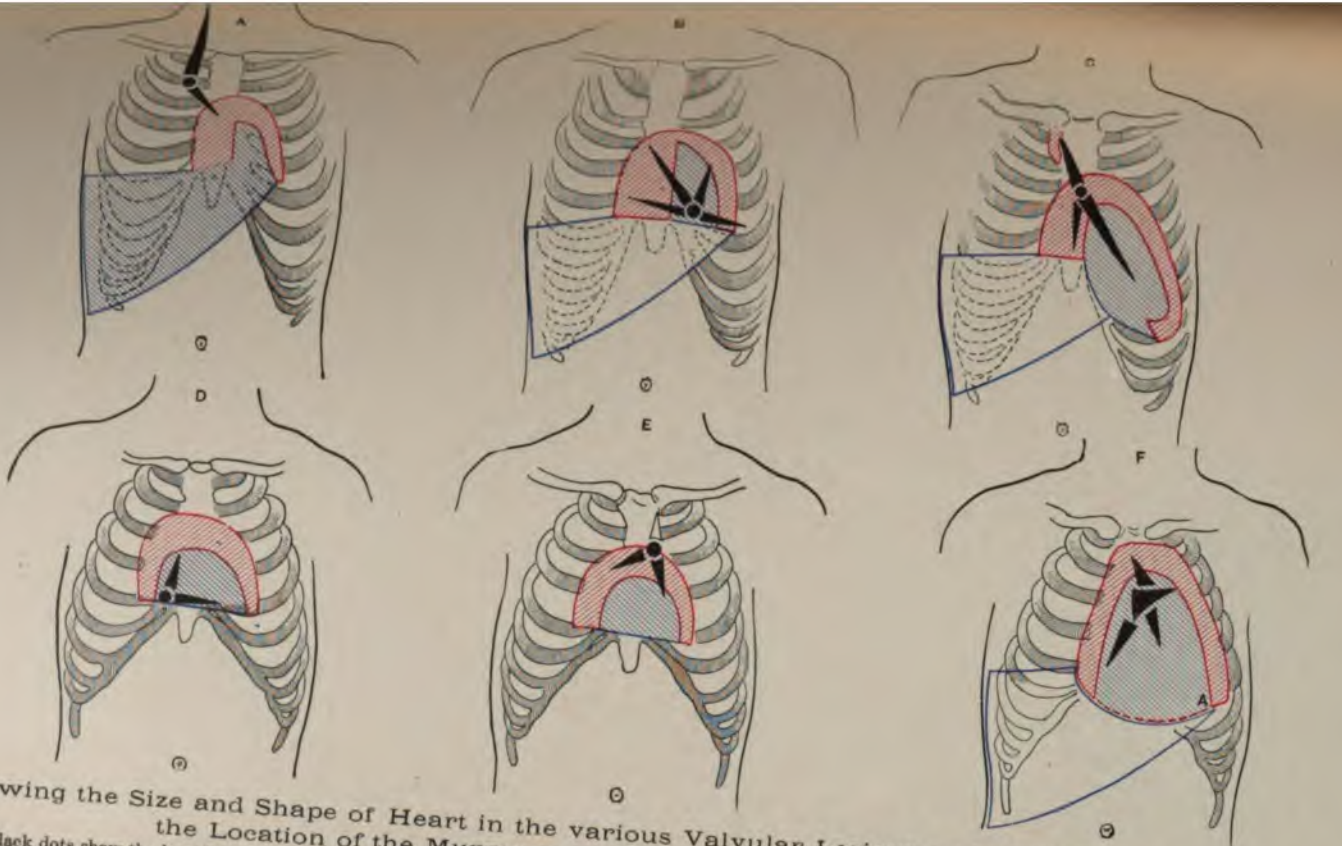
Cerebral Symptoms.—Delirium, anxiety, restlessness, insomnia, mania, choreiform movements, convulsions, psychoses (Burrows and Flint), or even coma, may be due to pericarditis, or to complicating uræmia or endocarditis. The symptoms may be suggestive of gastritis, while pericarditis is found on examination. Vomiting, singultus, dysphagia, recurrent nerve paralysis, and tracheal cough, are symptoms of pericardial pressure.

Physical Findings.—The definite diagnosis depends on the physical findings. These are obtained as follows:

1. **Inspection.**—Precordial prominence, *voussure,* first noted by Louis, may occur when the chest is plastic, as in women and children, and when the intercostal muscles are paretic from inflammatory œdema. The left chest often moves less than the right, since the left lung is usually compressed. An epigastric tumor-like bulging of the luxated liver was noted by Auenbrugger and Corvisart. The apex-beat is somewhat lower when exudation depresses the diaphragm; it is more horizontal, more to the left, and the *dulness transcends the apex to the left.* The greater the effusion, the weaker the apex becomes, and *disappearance of the previously distinct apex beat is all-important.* There may be only an indefinite, diffuse cardiac impulse. On bending forward the apex beat or cardiac impulse usually reappears. The interspaces may bulge. Collateral œdema of the chest wall (Rendue) is most rare. Undulation, described by Senac, is not due to the exudate, but to the heart itself. Graves and Stokes described tumor-like extrusion of the compressed lung above the clavicle. According to Ewart, the upper edge of the first rib can be felt as far as its sternal attachment, being separated from the clavicle ("first rib sign"). The veins of the thoracic wall are large. During convalescence the *apex may retract* during the systole. The decubitus is usually dorsal, or left-sided, rarely right-sided, for this compresses the sound lung; the head and shoulders are usually elevated, and the attitude is fixed and rarely changed. The knee-hand position was noted in Zehetmayer's case.

2. **Palpation.**—Palpation confirms the location of the apex. The *friction rub* is often felt. In Fuller's cases of palpable rub, there was always external pericarditis which favored conduction of the pericardial friction. *Disappearance of an apex beat previously observed, and the presence of a strong radial pulse, is very important,* because mere absence of the apex is observed in many conditions. *Epigastric tenderness* on either side of the ensiform, or tenderness between the attachments of the sterno-mastoid, is due to inflammation of the phrenic nerve (Guéneau de Mussy and Peter). Fluctuation is practically never felt.

3. **Percussion.**—Three to five ounces of fluid are usually necessary to produce distinct physical signs, though Fraentzel has detected the presence of one and one-half ounces. There are two complementary recesses in the pericardium: one over the basal vessels, where the fluid may accumulate, in which case it is early revealed by a triangular dul-



Showing the Size and Shape of Heart in the various Valvular Lesions and in Effusive Pericarditis, the Location of the Murmurs and the Direction of their Propagation.

The black dots show the location of the murmurs and the black wedges show the direction of propagation, growing thinner as the murmurs grow fainter. Over the heart, the red area designates the relative and the blue area the absolute cardiac dulness. A, aortic stenosis; B, mitral lesions; C, aortic regurgitation (note dulness over aorta); D, tricuspid lesions; E, pulmonary lesions (dark wedges designating pulmonary stenosis and insufficiency, and the light wedge, pulmonary stenosis); F, effusive pericarditis (pyriform dulness; wedges showing where friction is most often heard; A, apex, well within the area of dulness).

ness, whose apex is directed downward (Skoda, von Dusch, Oppolzer), which may compress the large veins or arteries; and the other in the fifth right intercostal space (Rotch, Ebstein), where the fluid exudate replaces the relative liver dulness, and is most easily recognized by palpatory percussion. Extensive exudates produce an outline usually designated as triangular, with the blunted apex upward, or as pyriform with the base directed downward. The dulness may even reach the interclavicular notch, the right nipple, or the left axilla, and may impinge on Traube's semilunar space, extend beyond the left lobe of the liver, compress the lungs, and markedly depress the diaphragm and liver. The dulness is, at times, trapezoidal (see Plate VIII, Fig. F.) rather than triangular. Very massive exudates may be mistaken for pericarditis plus pleurisy with effusion. Though the outline is roughly triangular, the right border is more vertical than the oblique left border, which is beyond the apex beat, if the latter remains visible (Skoda). The apex beat is dislocated abnormally when the patient lies on his side, because the pericardial sac is larger than normal, and permits greater cardiac movement. German writers hold that the heart falls back in the fluid, but the French think it comes forward. The dulness varies with fluctuations in the effusion and with resorption. When the patient is in the erect posture, the dulness may be one-third to one-half as broad again as when he is in the dorsal position; it also is not so high (Gerhardt). This is not invariable, because the lung borders must be normal and the exudate freely movable to allow free shifting of the fluid. Sahli also found increase in dulness when the subject was in the sitting posture in case of an enlarged heart, and in valvular disease; this he explains as caused by depression of the diaphragm, or by the emptying of the blood from the auricles. He thinks the lateral more important than the upper border. Sitting is usually unnecessary and often dangerous to the patient. Change of the dulness, especially when the patient lies on the right side (Rosenbach), is important, for this is uncommon physiologically, whereas the shifting of dulness in the left decubitus is of minor value.

Dulness may persist from cardiac dilatation, or from pulmonary retraction. The effusion may be enormous (10 quarts, Kyber; or 8 pounds, Corvisart), and may lead to a diagnosis of pleurisy, or pleurisy plus pericarditis. As the normal pericardial sac holds only two-thirds to three-quarters of a quart (650 to 800 c.c.), the pericardium must be greatly relaxed by the inflammation, to allow such large effusions. The pericardial dulness may not be typical when the effusion is largely covered by emphysematous lungs, or when the borders of the lung are adherent and cannot be pushed back by the effusion.

4. **Auscultation.**—Auscultation reveals the chief and most reliable diagnostic sign, the pericardial friction due to attrition of the inflamed pericardial surfaces. (*a*) It is usually a "*to-and-fro*" rubbing which is not exactly synchronous with either the systole or diastole, which character is most clear when the heart is slow. It is less often single, and occurs mostly late in the systole; it may be triple, *i.e.*, presystolic,

systolic, and diastolic. It is often absent in fibrinous, hemorrhagic, or purulent inflammation. In contradistinction to pleurisy, where the effusion of fluid abolishes the primary friction, it may be present, even with great exudation (Stokes), especially at the base, since here the heart may displace the fluid and touch the parietal pericardium. A certain consistence of the fibrin seems necessary to friction, for the latter is absent in very soft fibrinous deposits. (*b*) In *quality*, it may be harsh, soft like a gentle and interrupted scratching of the ear with the finger, whistling, creaking, crunching, musical, or even metallic (resonance from the stomach or intestines). (*c*) In *location*, it is most often heard over the base or the tricuspid region, where it is especially significant. It may also be heard over the apex. Sears describes a case in which the friction was heard most clearly at the angle of the left scapula. (*d*) It is very *superficial*, and close to the ear, not deep as in endocardial murmurs. Superficiality of the friction sound is absent when partial adhesions exist anteriorly, and the fluid accumulates behind the heart. (*e*) Its *propagation* is not wide (*il nait et meurt sur place*, Jaccoud), though it has been heard at a distance of nine feet. In children it may be heard over the whole chest, and the author has once seen the same in an adult. Coincident hydrothorax (Graves) has been thought to favor its propagation, and cardiac hypertrophy, by bringing the heart closer to the chest wall, propagates the friction more widely (Stokes). The murmur usually ceases abruptly when the stethoscope has been removed a short distance. It does not follow the same lines of propagation as it does in valvular heart disease. Propagation along the sternum is not uncommon. (*f*) *Inspiration* usually increases the murmur (Traube), and this is due to the closer apposition of the pericardial leaves, caused by the movable wedge of expanding lung. (Sometimes it is more marked during expiration [Lewinski, Paul], particularly when adhesions exist between the heart and lung [Riegel].) (*g*) *Moderate pressure* usually increases the friction, especially in plastic chests, though pressure may also accentuate valvular murmurs, and much pressure may inhibit the cardiac action (Friederich). Change of posture (Corrigan, Stokes), *e. g.*, leaning forward, often makes the murmur clearer. (*h*) *The murmur* is variable, now present, now absent, now systolic, or again systolic and diastolic, varying in acoustic properties, or changing with change of posture; it is often short lived, lasting sometimes but two to six hours. It is frequently stated that, as exudation increases, the friction disappears, but returns again during absorption. "It increases upward during exudation, and downward during absorption" (Sibson). Nevertheless, friction often persists when there are large exudations, if, indeed, its persistence is not invariable (Walshe). Balfour claims that friction, once heard, is never effaced by even the greatest effusion. It is not dependent on the intensity of the inflammation, but rather upon the accidental quality of the effused lymph and its location. Disappearance of the rub is explained by (i) regression; (ii) exudation; (iii) adhesions; (iv) weak heart.

As a rule, the heart tones gradually become more or less weakened and distant.

The *x*-rays outline the pericardial effusion.

Secondary Physical Signs.—There may be evidences of cardiac incompetence, which is due to the mechanical hindrance, by the fluid, of the diastole, especially of the auricles; it is also due to myocardial inflammation or degeneration, or to coincident cardiac disease. The signs of cardiac incompetence are hepatic and renal stasis, ascites, œdema, cyanosis, dyspnœa, cerebral anæmia (syncope), distention and pulsation in the jugular veins, and œdema of the lungs. The *pulse* at first undergoes irritative acceleration, but a lasting frequency indicates myocardial change. It may, in exceptional cases, be normal, or even slow (to 36), as a result of compression. There is no characteristic pulse; dicrotism is frequent; there may be the pulsus paradoxus (weakening or intermitting with each inspiration [Traube]). A very strong or water-hammer pulse may suggest underlying valvular disease. Irregularity is occasional, as is the pulsus differens and difference in the pupils. *Lung compression* is frequent. Bamberger observed a small area of compression, the size of a dollar, at the angle of the left scapula, where bronchial breathing, dulness (or tympany), and increased fremitus, are noticed; the compression clears up when the patient assumes the knee-chest posture (Pins). Pleural puncture excludes the possibility of fluid in doubtful cases, and the absence of crepitant rales excludes pneumonia. Bamberger's sign occurred in cases of hæmopericardium (Allbutt). There may be tympany (or dulness) under the left clavicle and to the left (and sometimes to the right) of the heart in cases where there is a fair-sized effusion. Ewart describes tubular breathing near the right mamma.

Thrombosis of the innominate veins may result from compression. Walshe observed tracheal compression. The second pulmonic sound may be accentuated, which Warthin regards as an early sign. The second tone may be split (Skoda). Metallic heart tones may be due to an adjacent cavity, to pneumothorax, or to the stomach and intestines. A systolic non-valvular murmur over the aorta has been explained by pressure on, or inflammatory relaxation of, the vessels, thus interfering with their normal vibration. Organic valvular or accidental murmurs may occur.

Diagnosis.—Differentiation rests upon (1) the *friction*, and (2) the *dulness*, or on both, with constant attention to (3) the *apex beat*. Subdivision into types, such as acute or chronic, fibrinous, serofibrinous, seropurulent, purulent, ichorous, or hemorrhagic, is inadvisable, as puncture alone reveals the character of the exudate. Fibrin formation and serous exudation usually coëxist (Latham).

1. The Pericardial Rub.—*The pericardial rub* is practically *pathognomonic* of pericarditis (Von Dusch). (It has resulted on rare occasions in cholera from tissue dryness, in tubercles, cancer, soldier's spots, hemorrhage, hypertrophied heart, and inflammation of the diaphragm or of the peritoneum, in which cases the heart beats against the inflamed surface.)

(*a*) In distinguishing it from *endocardial murmurs*, the acoustic quality, superficial character, lack of rhythmic precision, variability, slight propagation, palpatory difference in the rub, the basal location, and increase of the pericardial friction by pressure or inspiration are most decisive. Sometimes pericardial friction obscures a concomitant or preëxisting endocarditic bruit. The distinction is sometimes difficult, between an aortic to-and-fro murmur and pericardial friction. Valsalva's experiment—*i. e.*, a long inspiration with closed glottis followed by expiration with tense abdominal muscles—intensifies pericardial friction and decreases endocardial murmurs. Bearing in mind the great frequency with which pericarditis is a complication (or coördinate phenomenon) of endocarditis, this special point necessitates reserve in the prognosis, because the valvular disease remains after the pericarditis subsides.

(*b*) It must be differentiated from *pleuropericardial friction* (pseudo- or extrapericardial) originating in the inflamed pleura, where friction depends not only on respiration, but also on the heart's action. The usual distinction lies (i) in the close connection of the pericardial rub with the cardiac activity, which persists during expiration, and (ii) in the location of the pleuropericardial friction in the sinus mediastinocostalis (along the lingual lobe), in its intimate relation to respiration and in its usual cessation, either at extreme inspiration or expiration (Stokes, Addison). This method of differentiation is not infallible, for inspiration also augments true pericardial friction, which is, however, increased by bending forward. Valsalva's test (*v. s.*) is also useful. Pericardial friction toward the base or sternum is less often confused with pleural friction. Rosenbach finds differentiation most difficult when the basal vessels come in contact with a tuberculous cavity or pneumonia.

(*c*) Differentiation from *precordial emphysema* is possible on account of the crackling sounds of interstitial emphysema (air) in the anterior mediastinum. The sounds resemble rales, but are closely associated with the heart's action and are often metallic. The condition of the heart tones is decisive.

(*d*) Differentiation from *pericardial splashing* (see PNEUMOPERICARDIUM).

(*e*) *Crepitant rales* are easily differentiated.

2. The Dulness of Pericardial Exudation,—typically pyriform or triangular,—is modified by pleural or pericardial adhesions, and by pulmonary emphysema. The *relative cardiac dulness* (which may be markedly increased) should be considered rather than the *absolute* dulness (which is often little altered).

(*a*) *Hydropericardium* and effusive pericarditis may be confused, because stasis with hydrops pericardii may intervene in pericarditis, the friction may be absent or disappear, and fever may be lacking (chronic pericarditis). Hydropericardium shows its etiological factors. These are local pericardial changes or a general cause for hydrops, and are differentiated by previous anasarca or hydrothorax, which almost always develops first, by lower dulness, by resorption under digitalis and purges, by absence of friction, and by paracentesis, which reveals the usual differences between exudates and transudates in their opacity, specific gravity, and percentage of albumin (see PLEURISY).

(*b*) *Hæmopericardium*, resulting from trauma and rupture of a coronary artery, or from ventricular or aneurysmal rupture, is usually sudden, is attended by syncope, and blood is removed by puncture.

(*c*) *Pneumopericardium* (*q. v.*). (*d*) From *cardiac dilatation* the differentiation may be most difficult, especially when there is dilatation plus hydrops pericardii, in which case drastics, digitalis, friction, the history and evolution only, may decide. In dilatation the dulness is rarely triangular but rather is increased laterally, the shock-like impulse of the heart is more marked and often contrasts sharply with the weak radial pulse, compression of the lung is much more uncommon, and the heart tones are more clear and "snappy." The very distinctive relation between the apex and the outer dulness to the left is rarely fallible (*v. s.*). If the pulse is strong and the apex weak, pericarditis is present (with, of course, the typical dulness); but compression or weakness of the heart in pericarditis necessitates a weak, rapid pulse. In both affections the heart's dulness is increased by leaning forward; but this may clearly demonstrate the apex within the dulness of the exudate. When the patient lies on his right side the apex in pericardial effusion becomes visible (Rosenbach), which does not obtain in cases of enlarged heart. In children the diagnosis of pericardial effusion is often made incorrectly; the dulness is equally often a dilatation. (*e*) When there is *retraction of the left lung* away from the heart, the diaphragm is high, the apex and outer cardiac dulness coincide, the spleen is high, the pulmonary artery is exposed, and the proper signs of pulmonary contraction are elicited (Leube). (*f*) In cases of *mediastinal tumor* the dulness is irregular; there is no variation of dulness on postural change; the signs of increased conduction and bronchial breathing are noted with the absence of the cardinal signs of pericarditis. (*g*) From *aneurysm* the differentiation is usually possible, as well as from *marginal infiltration of the lung;* callous pleura, encapsulated pleurisy, superabundant fat (Walshe); and anterior mediastinitis (usually with friction, but with more vertically elongated dulness).

Diagnosis of the Character of the Exudate.—The fluid is serofibrinous in rheumatism; ichorous in perforation from hollow viscera; hemorrhagic in scurvy, carcinoma, tuberculosis, hemorrhagic exanthemata, and alcoholism; and purulent in sepsis and empyema. Paracentesis is the only certain test. Tuberculous pericarditis, even in clearly tuberculous cases, can be diagnosticated positively only by detection of tubercle bacilli in the aspirated exudate; a chronic pericarditis is probably tuberculous. In children 30 per cent. of pericarditides are suppurative and 17 per cent. are tuberculous (Baginsky).

Course.—The clinical course is variable; exudation may follow the friction in a short time or only after many days. The exudate may be rapidly absorbed in a few days or may endure for weeks or months. The average duration is one or two weeks. In convalescence the heart may long remain irritable on exertion.

Prognosis.—The prognosis depends largely upon the physiological resistance, the causal disease, and the character and quantity of the fluid; it is usually good in rheumatic and serofibrinous cases. Septic, purulent, putrid, or hemorrhagic types, are more serious. The pneumococcic form is fatal in 50 to 75 per cent. Pericarditis is most dangerous in the very young or aged. Death may be rapid in hemorrhagic cases; it usually is due to cardiac exhaustion, especially in cases of long-standing, and results from myocarditis rather than from simple pressure. The prognosis is more favorable in men than in women and children.

Treatment.—1. The *hygienic treatment* should be managed as it is in any acute infection with cardiac involvement.

2. *Absolute rest* in bed is indicated, in order to spare the heart, even during convalescence. The patient should never be allowed to sit up or leave the bed, even to urinate or defecate, for severe or fatal syncope may follow the effort.

3. *Laxatives* should be given, for constipation *per se* may increase the heart action, and difficult bowel movements, with the attendant straining, weaken or paralyze the heart. Sod. phosphate ʒss. to ij, is an excellent laxative.

4. The *diet* should be light or fluid, and tea and coffee, which excite the heart, should be avoided. Alcohol may be given when a septic process prevails or when the patient is accustomed to its regular use.

5. *Local antiphlogistic measures* should be employed. An ample ice-bag upon the precordia quiets and regulates the heart-rate, and relieves palpitation and pain. The author believes that it is often a better cardiant and sedative than drugs. Leeches may be applied after drawing some blood by small scratches over the heart; their action is regulated by applying salt, which causes them to release their hold. A Spanish-fly blister, as large as the hand, often not only relieves pain but stimulates resorption. It must be dressed carefully with weak carbolic salve and gauze. Cupping may be of service.

6. The *cause* should be treated. Therapy of the underlying disease is indicated,—such as the use of salicylates, in the rheumatic form.

7. *Heart stimulation* is indicated when the pulse is irregular, fast, or small, for this is due to early cardiac irritation or to later mechanical compression of the heart and lungs by the exudate (see TREATMENT OF VALVULAR DISEASES). Alcohol should frequently be given (see TYPHOID FEVER and PNEUMONIA for its action and indications). Strychnia, coffee, and camphor, operate more quickly than digitalis; they can also be used to follow up its action. Cardiac depressants, such as aconite, are to be carefully avoided.

8. *Treatment of the fever* depends on the basic disease. Hydrotherapy is preferable to the use of antipyretics.

9. *Compression symptoms*, such as dyspnœa, insomnia, and vomiting, when mild in degree or not of long standing, are met by the judicious use of morphine hypodermatically. Tardy resorption of large exudates or sudden or severe symptoms therefrom necessitate *tapping of the pericardial sac*, under the general precautions observed in pleural aspiration. The puncture is made in the fifth left (or right) interspace about an inch from the sternum, where the sac is not covered by the pleura. Care and gentleness in passing the trocar enable us to feel the giving way of the parietal pericardium and to avoid cardiac trauma. Removal of but a small quantity frequently provokes absorption by relaxation of the vessels in the tense pericardium, although results are sometimes disappointing. Sudden or extreme cardiac embarrassment may necessitate free venesection.

10. *Special indications* in various forms of the disease are encountered. Ergot is indicated in the hemorrhagic type, alcohol in drinkers, and care of the kidneys in nephritis.

11. *Delayed absorption* is treated by potassium iodide gr. x to xx, by diuretin 60 to 90 gr. *per diem*, and by small doses of calomel followed by salines. Hot packs, alcohol sweats, and pilocarpine, should be most cautiously employed or entirely avoided, for with their use symptoms of collapse occur quite frequently.

12. *Early pericardiotomy* is indicated in suppurative or putrid forms when the causal disease permits it. About one-half of the cases recover.

Adhesive Pericarditis.

(Pericardial Concretion [Synechia]; Cicatricial [Callous] Mediastino-Pericarditis.)

Though pericarditis may resolve completely, local adhesions within the sac or more extensive bands to the chest wall, pleura, spine or other structures, are frequent. They develop in 41 per cent. of cases of pericarditis (Breitung) and are found in 6 per cent. of autopsies (Leudet). In one group of cases, complete obliteration of the sac may occur with absolute clinical latency; the heart may beat itself loose from fresh or lax adhesions, especially when they are located about the apex. In a second group, there are undistinctive evidences of cardiac insufficiency. In a third series, distinctive physical signs exist. Adhesions most frequently follow serofibrinous pericarditis. A clear history or the actual observation of a previous pericarditis is one of the strongest points in the diagnosis.

Symptoms.—1. Cardiac insufficiency is caused by pressure on the coronary arteries, by myocardial fibrosis and degeneration, but more often by dilatation or atrophy. Though severe heart symptoms in advanced life without valvular disease are indicative of myocardial degeneration, in the young they suggest pericardial adhesions (Wilks). Evident failure of the right ventricle without valvular, renal, or pulmonary disease, or with sudden break in compensation in the usual valvular lesions, directs attention to possible pericardial adhesions. Weiss and Pick have called attention to an isolated ascites which is not found in simple cardiac incompetency, but is usually associated with large liver and somtimes with fibrous peritonitis; it is probably due to the extension of inflammation along the vessels from the pericardium to the peritoneum. Sudden death is sometimes due to pericardial synechiæ (Aran).

2. **Systolic retraction of the interspaces at the apex** may occur even with slight basal adhesions which hinder the systolic descent of the heart, while diffuse adhesions other than basal often produce no symptoms. It is especially significant when the normal systolic protrusion has been gradually replaced after pericarditis by systolic retraction, for retraction of the apex also occurs in other conditions. Systolic retraction is merely an expression of impaired locomotion of the heart and of lack of space at the apex. It is best seen on deep inspiration and disappears when the heart grows weak. Retraction of the apex can be seen, and can also be felt.

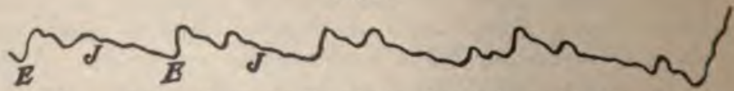
3. **Retraction** (not merely of the apex but) of the *sternum, epigastrium* (Oppolzer), *diaphragm*, and *lower chest wall*, may be observed when adhesions also exist outside of the pericardium in the mediastinum (Skoda); it is indicative of indurative mediastinitis, which attaches the heart anteriorly to the chest wall and posteriorly to the spine, and necessitates retraction unless cardiac weakness develops. Broadbent's sign is a systolic retraction of the eleventh and twelfth ribs posteriorly and on the left side.

4. **The diastolic recoil** (Friedreich), due to rebound of the retracted diaphragm and chest wall, is recognized by palpation and by auscultation as a dull vibration which may cause the second tone to appear doubled.

5. **Diastolic collapse of the cervical veins** (Skoda) is of great diagnostic value, and is due to their aspiration by the heart; it is sometimes dicrotic and may produce diastolic pallor of the face. The cervical veins often swell with the systole.

6. **The pulsus paradoxus** (a pulse intermitting with inspiration) strongly suggests cicatricial mediastinopericarditis, particularly when observed with the signs previously mentioned. It is observed to a moderate extent physiologically and is sometimes seen in other conditions which lessen the entrance of air into the lungs or render the heart sensitive, especially in myocarditis (Riegel). It is due in mediastino-

Fig. 21.



Pulsus paradoxus (Kussmaul); E, beginning of expiration, and J, of inspiration.

pericarditis to inspiratory stretching of the great vessels by well-attached mediastinal adhesions, which shuts off the blood supply to the extremities. It is a mediastinal symptom, and occurs (*a*) when the arterial system is poorly filled, and hence is more important when the heart tones are normal or stronger than normal, and (*b*) when the return venous flow is retarded. Schreiber holds it pathognomonic when (i) it appears without forced inspiration, (ii) when the radial and all other pulses are completely suspended by inspiration, (iii) when the heart's action is regular and (iv) when it is combined with inspiratory swelling of the cervical veins (*v. i.*). Struempell observed slowing of the pulse during inspiration. This was explained by him as irritation of the vagus by the mediastinal connective tissue.

7. **Inspiratory swelling of the neck veins** (Kussmaul), which normally collapse during inspiration, is due to the same mechanism as the pulsus paradoxus. Inspiration stretches the mediastinal bands, which constrict the large venous trunks and interrupt the return venous flow. Cyanosis may occur with each inspiration.

8. **Other signs** are fixation of the heart by adhesions, which gives no change of dulness by change of posture (von Dusch); absence of respiratory excursion over the heart; decrease of Traube's semilunar space by pleural adhesions (Jaccoud); decreased movement of the left half of the epigastrium (diaphragmatic adhesion to heart); expiratory weakening of the apex by pleural adhesions (Tuczek-Riegel); weak right heart with no accentuation of second pulmonic tone; loud, even musical, murmurs, especially in the aged; systolic emptying of the veins of the thorax due to systolic dilatation of the internal mammary veins (Broadbent); a rumbling presystolic murmur (Fischer, Hale White) at the apex, especially in children, without signs of mitral stenosis; precordial creaking (Perez) when the patient moves the arm; and other mediastinal symptoms, *e.g.*, left recurrent laryngeal paralysis, venous thrombosis in arm, etc.

Diagnosis.—In the diagnosis, two groups of symptoms are observed: first, the myocardial (dilatation, hypertrophy, relative tricuspid insufficiency); and second, the mediastinal (pulsus paradoxus, etc.) (See Pick's PERICARDITIC PSEUDOCIRRHOSIS, under DIAGNOSIS OF HEPATIC CIRRHOSIS.)

Prognosis.—The heart must suffer to a varying extent when its movement is impaired, whence the prognosis depends upon the condition of the myocardium.

Treatment.—The treatment is identical with that of valvular disease (*q. v.*) or of myocardial insufficiency in general. Brauer advocates surgical solution of the adhesions (cardiolysis); about a dozen operations are reported. The heart muscle must, however, be in good condition to warrant surgical interference.

PNEUMOPERICARDIUM.

The presence of gas or air in the pericardium is very infrequent; James (1904) could collect but 38 cases.

Etiology.—It results (*a*) from trauma, such as perforating wounds; (*b*) from perforative processes from cavities or viscera containing air or gas, such as pulmonary cavities; from gangrene, pneumothorax, œsophageal ulcers or neoplasms; gastric ulcer, liver abscesses, or caseated bronchial glands. There is almost always fluid, which may be ichorous, hemorrhagic, purulent (pyopneumopericardium), or more rarely serous (hydropneumopericardium). (*c*) Exceptional instances of spontaneous gas development without solution of continuity have been observed, due to the Bacillus aërogenes encapsulatus.

Diagnosis.—On *inspection,* there is precordial bulging and absence of the apex beat, though this may reappear upon leaning forward. On *palpation,* the vocal fremitus about the heart, and the apex beat are gone, friction and occasionally splashing, churning, gurgling sounds may be felt. On *percussion,* when the patient is sitting, a tympanitic or even metallic note is obtained above or indeed over the entire heart, especially when percussion is made with the plessimeter and the hard part of the percussion hammer or the finger-nail. Fluid is usually detected when the patient is in the sitting or lateral decubitus.

The tympanitic note heard above, and the fluid below, is present only when no adhesions are present and varies during the systole and diastole; it becomes higher in the erect posture and varies with the amount of exudation. The change in note on change of posture and the condition of the apex depend upon hydrostatic laws, as is the case in pneumothorax. The lung is compressed. On *auscultation,* the heart tones vary with the fluid, being loud if there is no great effusion, and usually metallic or musical, like a sound from a zither. The same is true of the quality of the friction (for pericarditis is almost invariably present). The tones, the metallic tinkling or splashing, the falling-drop sound, the mill-wheel churning and succussion, may be heard at a distance. They may keep the patient awake and may be heard in an adjacent room, as in cases of Stokes, Laennec, and Graves.

Differentiation is required from cavities or encapsulated pyopneumothorax near the heart. Consideration of the heart dulness, even though it be dislocated, and of the apex beat, renders the diagnosis easy. Confusion with distention of the stomach, which imparts a metallic note to the heart tones, is similarly excluded.

Prognosis.—Death occurs in 70 per cent. of the cases and usually results within two weeks, from the causal disease and from cardiac insufficiency. Recovery is most probable in the traumatic cases.

Treatment.—Treatment is the same as in pericarditis. Puncture to void the gas, and operation for putrid or purulent processes, are indicated.

HYDROPERICARDIUM (HYDROPS PERICARDII.)

Hydropericardium is always secondary to (*a*) *circulatory disturbances*, such as heart or pulmonary lesions, or local obstruction in the pericardial veins; the latter is infrequent; or (*b*) *cachectic conditions* producing blood alteration and transudation, such as Bright's disease, or carcinoma.

Symptoms.—They are much the same as in pericarditis, except that friction is absent. The heart region is sometimes prominent, the apex beat is lost, the heart tones are distant, the fluid changes with change of posture, the dulness is clearly triangular or trapezoidal since no adhesions exist. In most autopsies a small amount of fluid is found which transudes during the death agony or after death. In hydropericardium, the fluid is yellow-green, and is sometimes slightly tinged with blood. Cholestearin crystals are sometimes found. Evidences of inflammation are absent. The fluid is alkaline, contains some albumin, sugar, and urea, and is of low specific gravity. Chyle-like effusions (chylopericardium) are very rarely seen.

Diagnosis.—The signs of fluid are the same as they are in pericarditis (*q.v.* for differentiation), but there is no temperature, tenderness, nor friction murmur. Dilatation of the heart causes the most frequent diagnostic confusion and is differentiated as it is in pericarditis. The etiological diagnosis is most important, such as finding a cardiac lesion in the first and renal lesion in the second group.

Prognosis and Treatment.—The prognosis and treatment depend wholly on the cause. Cardiac stimulants, blisters over the heart, laxatives, etc., are indicated.

HÆMOPERICARDIUM.

Blood tinges the exudate in certain forms of pericarditis. A large quantity of blood may escape into the sac in trauma of the heart or of the sac, rupture of the heart (*q. v.*) or of aneurysms (*q. v.*).

The *symptoms* and *physical signs* relate to (*a*) the *acute anœmia*, with pallor, syncope, and rapid pulse, which usually ends, in a few hours or possibly a few days, in death; (*b*) signs of fluid in the pericardial sac. The *prognosis* is favorable only in the traumatic cases. The *treatment* is expectant; the heart should be stimulated and salt solution should be infused.

DISEASES OF THE ENDOCARDIUM.

ENDOCARDITIS.

Definition.—Inflammation of the endocardium. Its clinical importance attaches to the fact that the valves of the heart are involved. Two main varieties are distinguished, the *acute* and the *chronic*.

I. Acute Endocarditis.

This is practically always a *secondary, general* disease, and is *mycotic* in origin (excepting atheromatous endocarditis). Two main clinical varieties are described: (*a*) Malignant or ulcerative endocarditis, and (*b*) simple or verrucose endocarditis. They differ only in degree. Litten divides endocarditis into (*a*) the *benign* forms produced by rheumatism, chorea, gonorrhœa, scarlatina, measles, variola, typhoid, diphtheria, pneumonia, tuberculosis, grippe, and traumatism; and (*b*) *malignant*, (i) non-suppurative, caused by rheumatism, chorea, or gonorrhœa, and (ii) suppurative or septicopyæmic.

(A). MALIGNANT ENDOCARDITIS.

This variety is also known as ulcerative or septic.

Etiology.—The heart lesion is either (1) *secondary* to some clearly causal infection or disease, as furuncles, dysentery, typhoid, empyema, abscess or gangrene of the lungs, pneumonia, tuberculous foci with mixed infections, hepatic abscess, suppuration of the gall bladder, pylephlebitis, pyonephrosis, cystitis, gonorrhœa, calculi, diseases of the prostate, puerperal or other infections of the female genitalia, middle-ear disease, and to osteomyelitis or such specific infections as scarlatina or more rarely typhoid, rheumatism, diphtheria, smallpox, or malaria; or (2) *cryptogenetic*, when the causal infection is not apparent. Care in examination and at autopsy usually reveals an obscure cause and thus decreases the number of these cases.

The disease is more common in women than in men, even excluding the cases resulting from puerperal infections; it is most frequent between the years of twenty and forty. Its frequency is 2 per one thousand patients. Seventy-five per cent. of cases develop upon an old valvular lesion by which the physiological resistance is lessened, or in a hypoplastic aorta. The literature contains 11 cases of ulcerative endocarditis involving a patent ductus Botalli (Hart, 1904).

Pathology.—Malignant endocarditis most often involves the *left heart*, because it is far more frequently the seat of previous lesions and its arterial blood favors the growth of microörganisms, although the right heart is involved more frequently than it is in other types of endocarditis. Lenhartz found the left heart affected in 86 per cent., the right in 12 per cent., and both sides in 2 per cent., of his cases.

Though the process may involve the walls (parietal or mural endocarditis), it most frequently attacks *the valves* (valvular endocarditis), which are often already indurated by previous simple endocarditis, are most washed by the circulating blood, and are subject to greater mechanical activity. The endocardium becomes opaque, and irregular deficits occur on the under surface of the aortic or the auricular surface of the mitral valves; and later there is a grayish deposit surrounded by some swelling and injection of the endothelial coat. Exudation is abundant on the mitral valves, but is late or scanty on the less vascular aortic or pulmonary valves. *Thrombi* and excrescences develop on the erosions, whose removal discloses subjacent *ulceration*. The thrombi consist of lamellated fibrin and bacteria. The *vegetations* may attain the size of a walnut and often extend from one valve to another by contact infection.

Some thrombi are apparently purely mechanical deposits of fibrin, but genuine endocarditis is probably always mycotic. Heiberg first found microörganisms (1869) and Weichselbaum first obtained pure cultures.

Efforts to classify endocarditis by the bacteriological findings are more or less impracticable. The following bacteria have been found: Streptococcus, staphylococcus, pneumococcus, and less frequently the gonococcus, colon, diphtheria, influenza, tubercle, and typhoid bacillus, meningococcus, a long bacillus (Alchame), Bacillus endocarditidis griseus and encapsulatus, Micrococcus endocarditidis rugatus, Bacillus pyogenes fœtidus, Bacillus fœtidus immobilis, and Bacillus pyocyaneus. The same organism may produce either the benign or malignant form.

Bacteria may cause the endocarditis or may only be deposited from the blood upon vegetations of other origin, for mixed infections are common. Experimental endocarditis (Wyssokowitch, Ribbert) has been produced by inoculations of various organisms, usually after wounding the valves by a sound introduced through the cervical vessels, but also without trauma. The pyogenic organisms are said to attack more frequently the mitral, and the pneumococcus rather selects the aortic valves. The mitral valves in general are more often involved than are the aortic. The process may invade the aorta, the pulmonary artery, or the lung. Destructive changes by ulceration through one leaf of the valve may produce acute valvular aneurysm, which, when aortic, projects toward the ventricle, and when mitral, projects toward the auricle. If the necrosis destroys both lamellæ of the valve, the perforation leads to valvular insufficiency. Ulceration of the chordæ tendineæ, or papillary muscles, also produces valvular insufficiency; necrosis of the parietes results in a "heart ulcer," which sometimes leads to communication between the ventricles, or to rupture of the heart. Dislodgment of tissue particles, thrombi, or bacteria, occurs frequently, and usually in the arterial circuit. The effects vary with the location of the *infarct* (See SYMPTOMATOLOGY) and with the character of the microbes; *i. e.*, they act mechanically and infectively. Streptococci are said to produce anæmic infarcts, while staphylococci result in suppuration. Pathological changes in other organs are those of (*a*) general sepsis, such as parenchymatous degeneration, or splenic tumor, (*b*) embolism, mechanical or infective, (*c*) stasis when the heart fails, and (*d*) the primary disease.

Symptoms.—The clinical features vary to such an extent, according to the basic disease, the special visceral involvement by embolism and the virulence of the microörganisms, that a general description is impossible. The onset may be violent or most insidious during convalescence from the primary disease. The autopsy findings in the *latent* cases may be surprising, as, for example, the discovery of a mural gonorrhœal endocarditis in a case which clinically resembled septicopyæmia. In the majority of cases, the septic element prevails. The *various types*, somewhat artificial though convenient for description, are:

1. The Typhoid Form.—The toxæmic symptoms, such as apathy, delirium, dry tongue, distended abdomen, splenic tumor, diarrhœa, and albuminuria, are suggestive of typhoid. The resemblance may be heightened by hemorrhage of the bowels, rose-like spots, and the response of the urine to the diazo reaction. Dilatation of the right heart, and cardiac murmurs, so important in endocarditis, are not uncommon in typhoid. Obvious embolism helps to establish the diagnosis. The pneumococcus is often the cause of this type. In the skin *embolism* appears as spots with a white or yellow centre and hemorrhagic periphery, or as pemphigus, gangrene, or suppurative foci. Analogous areas may be seen in the upper air-passages. Infarcts may sometimes occur in the conjunctiva, but in the retina simple spots of hemorrhage or actual infarcts with yellow centres are frequently detected. The entire eyeball may suppurate. The frequent splenic infarction is characterized by sudden pain, tenderness, enlargement, and sometimes perisplenitic friction. Embolism is often accompanied by fever, chill, and vomiting. Renal infarction is very frequent, and is attended by pain in the loins, hæmaturia, albuminuria, and sometimes anuria or dysuria. Staphylococci may be found in renal abscesses. Severe nephritis is common. Cerebral embolism usually occurs in the left Sylvian artery, and results in the usual shock and hemiplegia with aphasia. Pulmonary infarcts are caused by lesions of the right heart or by auricular thrombi, and occasion sudden pleuritic pain, dyspnœa, hæmoptoë, temperature, and possibly a small area of crepitant rales, pleural friction, or consolidation. Embolism also occurs in the liver, in the mesenteric artery (with enterorrhagia and peritonitic symptoms), in the muscles and joints, testes, and parotid; in the heart, and the various mucosæ; in the meninges, pleuræ, pericardium, peritoneum, and in the peripheral arteries, sometimes causing gangrene, or embolic aneurysm.

The *course* may be peracute (two to four days), acute (a few weeks), or chronic (several months). Death occurs from the typhoid state, from embolism, or from cardiac failure.

2. The Septic or Intermittent Type.—This form has an irregular fever-curve, with chills and drenching sweats; and the spleen is enlarged. Heart symptoms may be absent or indeterminate, the moderate relaxation of the right heart and the murmur being often called functional. The paroxysms are more irregular than in malaria and no response to quinine is obtained. The streptococcus is often causal. Cardiac findings, embolism, or clearly septic manifestations, may establish the diagnosis. The course is usually several weeks but may cover months.

3. The Visceral Forms.—These forms embrace the cases in which some one organ is especially involved. In the *cardiac form*, anæmia exists and the heart murmur seems accidental, until a typical valvular lesion develops, or, if one already be present from older valvular disease, until another appears as a diastolic following a systolic murmur. The diagnosis may be difficult even when chronic valvular disease is present. In the *cerebral form*, meningitis, apoplexy, coma, or psychoses, may dominate the clinical picture. The symptoms referable to any organ which is the seat of embolism (lung pericardium, etc.), may seem to indicate a primary disease. In this way acute yellow atrophy, cholera, etc., have been incorrectly diagnosticated.

Special Symptoms.—(1) All types of *fever*, from intermittent to continuous, have been noted, although it may be absent for a long time or may not develop at all. (2) The *heart* is always somewhat disturbed in its action, cardiac unrest being common. Its action is usually more frequent, but is quite variable, now being rapid, again slow, and often irregular. The heart seems to beat harder and the pulse may throb although the irritable overaction is attended by low arterial tension. The heart tones are dulled. Palpitation is frequent. Cyanosis is not common. (3) *Respiration* is always increased, from toxæmic centric stimulation, or from pain or respiratory complications (septic pneumonia, infarcts, pleurisy, pneumothorax, or gangrene). (4) The *digestive tract* may be affected. Anorexia is not invariable. Nausea, vomiting, and diarrhœa, may occur from the general sepsis. Embolism has been mentioned. The liver is enlarged, may be tender, or may be the seat of friction. Icterus may be marked, is ominous and is referable to duodenal catarrh or to oversecretion of bile (polycholia). (5) The *spleen* is almost always palpably enlarged from sepsis or embolism. (6) The *urine* shows evidences of the sepsis or stasis by albuminuria with or without cylindruria, of the "blood dissolution," and of the embolism by hæmaturia. Malignant endocarditis may run a course similar to that of subacute nephritis, which is present in 33 per cent. of all cases of the ulcerative type. (7) The *nervous system* may be irritated or dulled by the sepsis toxins, or embolic lesions, such as meningitis or encephalitis, may intervene. (8) The *motor system* is often affected. The muscles are frequently tender and actually inflamed, and may undergo suppuration. The bones are often tender and the joints tender or swollen, although the swelling is more frequently peri-articular than intra-articular. Bacteria are seldom found, and this may heighten the resemblance to rheumatism. (9) The *skin* may be the seat of embolism (*v. s.*) or of eruptions, such as sudamina, urticaria, erythema, purpura, polymorphous septic rashes from bacteria or their products in the circulation. Bullæ, gangrene, phlegmons, or bed sores, are also observed. (10) The *blood* is also affected. There is usually marked anæmia; when there are no heart findings, the anæmia and splenic tumor have been confused with pseudoleukæmia. The leukocytes are often but not always increased. Bacteria may be cultivated from the blood by modern methods of inoculation from a large quantity of blood in much bouillon. The latter and other points are considered under SEPTICOPYÆMIA (*q. v.*).

Bacteriological Forms.—(*a*) *Streptococcic Endocarditis.*—The ordinary streptococcus is the usual cause of rapidly fatal and of chronic ulcerative endocarditis, both with severe repeated chills and high irregular fever. Acute cases may result from the small streptococcus, which is said to usually cause moderate fever. (*b*) *Staphylococcic endocarditis* is less frequent. The course is nearly always acute and is attended by high fever, pustules in the skin, disseminated hemorrhages, and septic infarcts, especially in the kidneys. (*c*) *Pneumococcic endocarditis* (see PNEUMONIA) is usually acute; frequently follows pneumonia, less often cholecystitis; begins with gradual fever rise associated commonly with chills; and is often attended by suppurative meningitis. (*d*) *Gonococcic endocarditis* (Ricord, 1847) may result from Neisser's coccus or from mixed infection with the strepto-, staphylo-, or pneumococcus. Cases of recovery are instanced.

Diagnosis.—Bamberger's remark of half a century ago still holds: "The diagnosis of acute endocarditis is seldom easy, is usually difficult, and often impossible." Unless there are physical changes in the heart, the diagnosis is only presumptive. A proper etiology, temperature, signs of cardiac insufficiency, such as blue nails or cold extremities, irregular weak pulse with apparently strong, cardiac shock and dilatation are very suggestive and may be confirmed by continued observation, by embolism, by change in the valvular murmur, and by blood-cultures. Actual hypertrophy of the heart not only requires time for development but adequate nutrition, of which the disease seldom admits. In 37 per cent. of cases, the heart is negative, in 44 per cent. the murmur is systolic and in 5 per cent. diastolic. Absolute certainty rests upon our ability to diagnosticate by physical signs, the presence of a mitral regurgitation (*q. v.*) which is the most frequent form, or of an aortic regurgitation (*q. v.*) plus the *signs of sepsis* of which the disease is a part ("arterial pyæmia"). The valvular murmur may disappear after embolism. Confusion with typhoid or malaria is possible (see TYPHOID, differential table).

Diastolic murmurs are very rarely functional, and their appearance is therefore especially suggestive. Myocarditis may produce a systolic murmur, but is usually attended by marked early muscular insufficiency, weak-heart shock and irregular pulse, weak systolic murmur and a moderately accentuated second pulmonary tone.

Recurrent endocarditis shows not only the fever and other symptoms caused by the fresh lesions, but also the *hypertrophy* of the older valvular lesion. An aortic diastolic or a mitral presystolic murmur is more likely to indicate an *old* lesion. With repeated examinations, fresh murmurs may be heard in addition to the old ones.

Prognosis.—The prognosis is almost always unfavorable; Herrick (1902) collected 22 cases of recovery. It depends on the strength of the heart, the bacteriology, the number of infarcts and their location, the variability of the heart murmurs (which may entail danger of embolism), and the intensity of the sepsis. Recovery from the gonorrhœal form is relatively frequent. Musical murmurs with fever are usually regarded as ominous. Apparently light cases of endocarditis may become severe. The course may be rapid or severe; of but a few days duration; or it may have a slower evolution of even a year and a quarter.

Treatment.—(See BENIGN ENDOCARDITIS and SEPTICOPYÆMIA.)

(B). (VERRUCOSE OR) BENIGN ENDOCARDITIS.

This variety is more frequent (1.5 per cent. of clinical cases) and is always secondary. (See general prefatory remarks to ULCERATIVE ENDOCARDITIS.)

Etiology.—(*a*) *Rheumatism* (*q. v.*) is the most frequent cause, and 60 to 85 per cent. of the cases can be referred to it. Twenty per cent. or more of cases of rheumatism suffer from endocarditis. It occurs in the more severe rheumatic cases. In children it is very frequent, for in them the heart "resembles an articulation." Muscular rheumatism, angina, or erythema nodosum, may occasion endocarditis. Chronic rheumatism predisposes to it. (*b*) *Chorea* is frequently complicated by endocarditis (30 per cent. of clinical and 82 per cent. of fatal cases). By some writers chorea is considered rheumatic but the majority hold that it is only correlated. (*c*) Of other *specific infections*, scarlatina ranks first, (10 per cent. of the cases becoming involved). Endocarditis is far more uncommon after typhoid, diphtheria, tonsillitis, measles, erysipelas, gonorrhœa, variola, grippe, tuberculosis, or parotitis. (*d*) Occasionally it is associated with trauma, burns, syphilis, cancer, Bright's disease, gout, or diabetes. (*e*) It is more common in males between the years of twenty and thirty.

Pathology.—The vegetations are verrucose, wart-like, papular, or even pedunculated, are usually small, and occur at or above the lines of closure of the valves, whereby their function is interfered with and valvular insufficiency or stenosis results. At times the vegetations are cauliflower-like, or resemble condylomata. They are grayish, gelatinous, and transparent; they become whiter and firmer with age, and consist of two strata, the deeper being granulating endocardium and subendocardial tissue and the superficial layer consisting of thrombi from the coagulable elements of the blood. The signs of active inflammation, such as redness or swelling, are usually lacking, redness being sometimes confused with post-mortem imbibition of hæmoglobin from the blood. Vegetations must not be confused with Albini's nodes, which are seen in infants and children. The vegetations are at an early stage, quite cellular from leukocytic exudation and tissue proliferation and usually contain bacteria (see ULCERATIVE TYPE). Becoming detached, the outgrowths form *emboli* and produce mechanical hemorrhagic or anæmic infarcts, not suppurative infarcts as in the ulcerative type. The fate of the vegetations is (*a*) embolism; (*b*) organization with the bare possibility of restoration to normal; (*c*) organization with valvular lesions resulting from retraction, and from induration and calcification; (*d*) being a place of slight resistance, recurrent endocarditis may be observed; (*e*) the intensity of the inflammation may increase, leading to malignant endocarditis.

Location.—As in the first type, the vegetations are largely in the left heart, because of its greater physiological activity and its arterial blood, although these same conditions in fœtal life predispose to endocarditis of the right heart, either on the valves or on fœtal openings or defects. In general, the mitral valves are most affected (50 per cent. of the cases alone or 80 per cent. when combined with other lesions), next the aortic (13 per cent. alone or 40 per cent. when combined with other lesions), and far less frequently, the tricuspid, the pulmonary, and the walls of the heart, chordæ or parietes—(1) wall of left, or (2) of right auricle, (3) of left or (4) of right ventricle.

Symptoms.—The symptoms are not characteristic and a diagnosis is impossible or only presumptive unless the valves are considerably involved. Simple endocarditis therefore is found far more frequently at autopsy than at the bedside; *i. e.*, it is very often *latent*. With such heart symptoms as increased rate or palpitation, it is difficult or impossible to determine whether they are caused by a valvular lesion or a myocarditis, though the latter is the more likely cause. Systematic daily examination of the heart in such diseases as rheumatism may reveal the gradual involvement of the endocardium with moderate fever, and dyspnœa, cardiac unrest, oppression, palpitation, or insufficiency. Pain is more often pleuritic or pericarditic. The diagnosis depends on the valvular bruit, which often follows muffling or irregularity of the cardiac sounds, and is still later followed by the signs of mitral or aortic insufficiency (*q. v.*). The infrequent diastolic murmurs are more distinctive than the systolic, for they are rarely functional. *Embolism* is very distinctive, as it is in the first type, and is important mechanically rather than bacteriologically. It also occurs in parietal endocarditis; embolism may be so minute or undistinctive as to escape recognition.

Diagnosis.—Conservatism in making the diagnosis is necessary, because acute benign endocarditis is much too often diagnosticated. The murmurs are mistaken for functional murmurs accompanying febrile relaxation of the heart-chambers. (See MITRAL INSUFFICIENCY.) Confusion with pericardial rub (*q. v.*) is possible.

Prognosis.—(*a*) *As to life*, the outlook is generally good, unless (i) emboli lodge in important structures, such as the basilar or coronary arteries, or (ii) the process becomes malignant. (*b*) *As to complete recovery*, (i) valvular lesions are usual and, (ii) fresh recurrent endocarditis is very common.

Treatment.—The treatment of the two forms of endocarditis is considered together. (*a*) *Prophylaxis* resolves itself into the treatment of the causal *acute* infection, such as rheumatism or sepsis. Preventive treatment, such as the administration of salicylates and alkalies in rheumatism, is uncertain except that they shorten the rheumatic attack. (*b*) *Rest* spares the heart and probably lessens the percentage of cases involved. Complete rest, psychical and physical (cardiac), is the prime essential, and cardiac stimulants are to be avoided, at least in the early stages. Rest in bed must be enforced for weeks after temperature and signs of heart weakness have wholly disappeared. Should relative recovery occur, the general care of the heart's strength must be insisted upon as in chronic valvular disease, and, with intercurrent affections or with the appearance of temperature, the patient must be confined to bed. (*c*) The *sepsis* is treated by alcohol (see PNEUMONIA); quinine, gr. x to xv, once or twice daily, with three ounces of red wine every hour or two, is recommended by Fraentzel; bichloride of mercury and other antiseptics are futile; for pure or dominating streptococcic infections Marmoreck's antistreptococcus serum is indicated in doses larger than those usually prescribed, and should be frequently repeated. (*d*) The *irritable heart* may be quieted and slowed by the use of the ice-bag. Sinapisms sometimes act favorably but blisters are to be avoided. Cardiac stimulants should be employed with caution, and only coffee, ammonia, or brandy, should be given. Digitalis is usually to be avoided, but it is indicated when very tumultuous heart action threatens to induce embolism.

(*e*) *Symptomatic Treatment.*—For nervous symptoms, sodium bromide is indicated. Sulphonal and trional are to be avoided when the heart weakens. For the fever, hydrotherapy, aside from simple sponging, is inadvisable, for the joints and muscles are very tender, and movement, on account of the possibility of embolism, is dangerous. Diarrhœa of septic origin should not be checked at once, because it is nature's effort at derivation and elimination. Fluid diet should be given because of the fever. Potassium iodide is usually administered as a resolvent after the acute stage has passed.

II. Chronic Endocarditis. Chronic Valvular Disease.

Etiology.—Valvular heart disease constitutes 2 to 9 per cent. of all diseases. It often develops from (*a*) *acute verrucose endocarditis*, and therefore has the same etiology. Some cases develop without symptoms and are apparently primary or possibly hereditary; they are seen mostly in young or adolescent subjects and in women. (*b*) *Atheroma* is also an important cause; it is promoted by overexertion (as in the working classes), by advanced years, or by chronic nephritis, gout, diabetes, syphilis, alcoholism, and lead poisoning. It is a more common cause in males. Though chronic valvular disease may develop from other causes than chronic endocarditis, both topics can well be considered together. Valvular lesions less often result from (*c*) *rupture of the valves;* this occurs most often in the aortic valves during supreme physical efforts, and is promoted of course by previous valvular lesions, although it is sometimes observed in perfectly normal valves. Healing is most rare. (*d*) *Relative valvular insufficiency* is a condition in which the normal valves are unable to close the orifices to which they belong because the heart is dilated. It is most commonly tricuspid and mitral, although it also occurs in the aortic and pulmonary valves. Relative stenosis is not clearly established. (*e*) *Other rarer causes* are aortic aneurysms, neoplasms, valvular aneurysms, and cardiac thrombi or trauma.

Pathology.—Small grayish-red swellings are sometimes seen in the early stages; they somewhat resemble atheroma plaques. Foci of endocardial thickening and retraction (*endocarditis retrahens*) are seen, in which calcification and even ossification of the valves may be present. The valves become less translucent, are not infrequently rigid, unable to unfold, adherent to each other or distorted by their own thickening and retraction or that of the chordæ or papillary muscles. Mural changes are unimportant, except when associated with myocarditis. Acute exacerbations may occur and the ordinary vegetations appear. Embolism occurs in about 30 per cent. of the cases. The myocardial changes are hypertrophy, dilatation, and myocarditis. The valves most often involved are the mitral (78 per cent.), especially in adolescence, then the aortic (20 per cent.), which are especially subject to change in arteriosclerotics, and finally the tricuspid (1 per cent.), and the pulmonary valves. Combined valvular lesions occur in 33 per cent. of cases. The process often extends by continuity of surface from the mitral to the aortic valves. In fœtal cases (*v.* CONGENITAL VALVULAR DISEASE) the valves of the right heart are more frequently involved. When one valve is but moderately involved, the others of the same valvular orifice may compensate by stretching in order to cover the defect, especially in aortic disease.

Valvular insufficiency is usually caused by (*a*) thickening and retraction or even calcification of the valves and also of the chordæ tendineæ and of the papillary muscles; (*b*) massive vegetations; (*c*) growing together of the valves, or of the valves with the wall of the heart or aorta; or adhesions between the papillary muscles and tendons; (*d*) valvular aneurysm, perforation, rupture, or tearing of the tendons or muscles. *Valvular stenosis* (stenosis of the orifice) results from (*a*) adhesions between the valves, (*b*) retraction, calcification, fibrosis of valves or ring of insertion, (*c*) vegetations. Combined valvular insufficiency and stenosis in one valvular mechanism, especially the mitral, is most common.

Symptoms.—The symptoms of valvular disease are (*a*) the *cardiac findings* peculiar to each lesion, which will be first considered, and (*b*) the *general symptoms* more or less common to all forms of cardiac insufficiency, the description of which will follow the individual lesions.

III. Aortic Insufficiency. (Corrigan's Disease.)

Etiology.—**1. Endocarditic Form.**—The sinuses of the valves may fuse, vegetations are present, or the valves grow together at their margins and shrink; this is more frequent than valvular perforation or ulceration, which is rapidly fatal. If calcification develops, there may be coincident stenosis. This form usually follows a previous mitral lesion. The endocarditic may be combined with the atheromatous form.

2. Atheromatous Form.—Atheroma may be slight in the aorta, and located mostly on the valves. It is a slow, degenerative process (see ARTERIOSCLEROSIS) with marginal valvular retraction; it is caused by excessive use of alcohol, by continued hard work or athletics, and by syphilis, which may dilate the aortic ring (syphilitic aortitis), even without directly involving the valves. It is often therefore seen in middle-aged men. A relatively slight anatomical lesion may produce disproportionate symptoms. The atheromatous process may overlie or actually invade the coronary orifices. This is obviously dangerous. The valves may become adherent to the aorta. Their edges are rounded, and are not the seat of endocarditic masses. Coincident stenosis is infrequent. Relative insufficiency and congenital defects are less frequent.

3. Relative Insufficiency.—This form occurs mostly in cases of aortitis, aneurysm, and arteriosclerotic *dilatation of the root of the aorta*, which is a more or less physiological process, as the aorta usually relaxes one centimeter in circumference after the fortieth year. It may also occur in cases of *dilatation of the left ventricle*, resulting from idiopathic hypertrophy, from myocarditis, or from renal disease.

4. Congenital Defects.—These are rare; absence of a valve may be in part compensated for by the others, though anatomical anomalies predispose to sclerosing endocarditis. Valvular fenestration is unimportant clinically and pathologically.

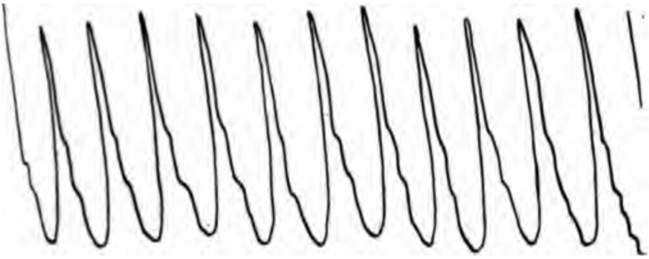
5. Trauma.—This is a most uncommon cause, only 25 cases being recorded (Trevisanello, 1904).

Mechanism.—From aortic valvular incompetence, a diastolic regurgitation of blood into the left ventricle takes place from gravity and contraction of the aorta. Blood currents and vibrations are caused by the blood flowing (normally from the left auricle and pathologically from the aorta) into the left ventricle, thereby producing the *diastolic murmur*. The *left ventricle becomes hypertrophied*, because it must propel the normal plus the regurgitated blood, and becomes *dilated* to accommodate its added volume. The size of the left heart becomes enormously increased, and may even weigh 50 ounces, whence the name "ox-heart,"—*cor bovinum*. The hypertrophied left ventricle suddenly forces an unusually large amount of blood into the arteries, and thus causes the many *arterial phenomena*.

Physical Signs.—1. **Inspection.**—(*a*) *Precordial prominence* is observed chiefly in plastic chests, and, therefore, is most common in women and children. Mensuration may confirm the prominence, although the right chest normally measures nearly an inch more than the left. (*b*) The heart *shock* is powerful and diffuse. (*c*) The *apex beat* is *dislocated downward* (6th, 7th, 8th interspace) and outward, beyond the nipple or anterior axillary line. The apex lies low in arteriosclerotics, from relaxation of the aorta. It is *heaving*, pulsating, and superficial. The lung is pushed back and some slight rotatory systolic retraction of the chest wall is seen. These findings are referred to the dilated and hypertrophied left heart. (*d*) Pulsation in the first and second right intercostal spaces is usually dynamic, because the extra volume of blood thrown with extra force into the aorta causes dilatation of the aortic arch. (*e*) The *peripheral vessels pulsate with unusual violence*, and the carotids hop; pulsation in the jugular fossa, or in the abdominal aorta, may simulate aneurysm, and the temporals and radials often attract attention before the patient undresses for examination; these are all caused by ventricular hypertrophy and the extra volume of blood thrown into the vessels. Pulsation may also be seen in the retina, either spontaneously or by pressure on the eyeball, as occurs also in Basedow's disease. The liver may pulsate, though this is rare because a strong heart action with stasis is necessary. The liver also pulsates in cases of tricuspid incompetency, but this throb is due to venous filling of the organ during the systole. Pulsation also occurs in the spleen, palate, and penis. (*f*) The *capillary* (*Quincke's*) *pulse*, occurs in over 80 per cent. of the cases, but is not pathognomonic, for it is also seen in health and in fevers or anæmias. It is a symptom of overfilling of the arteries and capillary dilatation, and is best observed when the hypertrophy exceeds the dilatation; it disappears when the heart weakens. It is elicited by exerting slight pressure on the tips of the finger nails, on the lips by a glass slide, or by rubbing the forehead until it is red; there is a systolic reddening and a diastolic paling in these parts. It is sometimes found in the retina, in the palate, or in cases of complicating erysipelas. (*g*) The passing over of the blood from the arteries into the veins may produce the *centripetal venous pulse.* This is seen most advantageously in the forearm, and is caused by (i) relaxation of the vessels, as in fevers, some nervous lesions, and chlorosis; (ii) by delicate skin; and (iii) full heart action. (*h*) The *arteries are actually lengthened.* (*i*) Patients often assume the dorsal decubitus, for gravity increases the leakage; subjects with mitral insufficiency usually sit.

2. **Palpation.**—(*a*) The hand corroborates the results of inspection,—as the heaving, *throbbing apex.* (*b*) *A diastolic thrill* over the base is infrequent, but when present is usually proportionate to the degree of leakage. (*c*) In the jugulum, especially in arteriosclerotics, the widened aorta can be felt. (*d*) The *pulse* findings may seem paradoxical, but it must be noted that (i) the arteries are filled violently (the pulsus durus), though (ii) the tone of the vessel wall is less than normal.

FIG. 22.



Sphygmogram of case of uncomplicated aortic leakage.

The radial artery is suddenly filled ("pistol pulse"), the *pulsus celer;* this is due to hypertrophy of the left ventricle. The condition of the heart muscle, and therefore of the pulse, depends largely on the type or etiology of the case; hypertrophy is usual, and marked in the endocarditic form, though in the atheromatous type coronary atheroma and fibrous myocarditis lessen the myocardial activity, render the pulse atypical, and make the prognosis less favorable. The sphygmographic tracing shows the high up-stroke with an acute apex (the *pulsus altus*). The dicrotic wave is ill-marked, and the pulse rapidly recedes (the *"collapsing pulse"*), due to the inability of the aortic valves to hold the receding blood. The falling away of the pulse is sometimes increased by lifting the arm. The pulse is not absolutely characteristic, because it also occurs in fevers or anæmias, but it is strongly suggestive. The typical findings are altered, (*a*) when atheroma of the aorta or vessels occurs; (*b*) when other valvular lesions develop, as aortic stenosis or mitral regurgitation; (*c*) when the heart is weak or degenerated. When the pulse findings precede the murmur it is claimed that the lesion is due to disease of the aorta. Thrills may be felt over various peripheral vessels. The *peripheral pulse is delayed* in about half of the cases, because the regurgitation continues into the first part of the systole.

3. **Percussion.**—(*a*) The heart area is *oval,* and is increased downward and *to the left,* as could well be anticipated from the mechanism.

Dulness reaches the second or third ribs to the left of the sternum (See Plate VIII, Fig. C). The heart is somewhat more horizontal than normal. The dulness extends slightly beyond the apex and is largely due to dilatation of the left ventricle. Because physical laws demand its equal dilatation in all directions, it is not surprising that the right heart may be somewhat crowded to the right, and the left auricle upward, although dulness to the right of the sternum is infrequent. The papillary muscles are found flattened and even thickened at autopsy, from the pressure of the regurgitant blood. (*b*) A small area of *dulness* in the first and *second right interspaces* denotes mechanical dilatation of the arch, which results from the cardiac hypertrophy.

4. **Auscultation.**—(*a*) *A diastolic murmur* is heard over the sternum at the level of the second or third rib. It is caused by the retrograde blood passing through the incompetent valves into the left ventricle. When heard over the so-called "pulmonic area," it is increased downward along the sternum, for the regurgitant column follows this direction (Plate VIII, Fig. C). It has a *blowing, deep,* and less often sawing, musical or whistling sound (which latter suggests a relative aortic leakage). When slight, it may easily escape recognition, but when well developed it is very characteristic, sounding like the word "*who.*" It sometimes disappears before death when the heart is weak and the lesion is great, but generally corresponds to the degree of leakage. It rarely reaches the neck or abdomen. Cases of valvular rupture have produced a murmur which could be heard at a distance. (*b*) *A slight systolic murmur* over the valves or aorta is not uncommon, but the too frequent diagnosis of aortic stenosis (see PULSE and THRILL under this topic) is not justified. It is soft in the atheromatous, and often harsh in the endocarditic type. Its various explanations are: Rough aorta or valves, physiological stenosis (the rigid valves lying between a wide aorta and a dilated ventricle); vibration of the aorta; or leakage continuing into the beginning of the systole. (*c*) *Weakness or absence of the second aortic tone* is explained by imperfect closure of the valves. Because the second tone is normally heard over the carotid artery it should be listened for there, because it is removed from the murmur and the second pulmonic tone. If it is present, the usual conclusion is that one or more valves are closing, though possibly imperfectly. By lifting the stethoscope a little from the skin over the aortic region, the second sound, which was previously obscured by the bruit, may be heard. (*d*) *The first mitral tone is often weak, or lost,* because the papillary muscles are flattened and indurated by the regurgitant column. If the change is pronounced, a mitral systolic murmur may be heard in place of the first tone. (*e*) *An apical presystolic* (*Flint's*) *murmur* is heard in 50 per cent. of the cases, and is due to aortic leakage continued in the systole, or to pressure on the mitral valves, which hinders the flow of blood from the left auricle into the left ventricle. Thayer found it in 60 per cent. of 74 cases, and in less than one-quarter of these was there a mitral stenosis. (*f*) *Arterial tones and murmurs* may result from vibration of the vessel wall; these tones may become murmurs, when

there is great difference in tension. The systolic vessel tone is increased from the larger volume of blood and increased ventricular action, and is most plain when the vessels are not sclerotic. It is also found in anæmia. Over the femoral artery, a systolic tone is heard which is convertible into a murmur on pressure. *Traube's double femoral sounds* consist of two spontaneous (*i. e.*, produced without pressure) tones, which are variously explained, probably most satisfactorily by sudden change in tension. They are heard best when the heart is strong and the vessels soft. They occur in other conditions and in valvular lesions, and may be confused with the double venous (Friedreich's) tones, which are heard over the vein in tricuspid leakage. *Duroziez's double murmur* is analogous, but consists of two murmurs, which may be elicited by slight pressure and are due to the progress and recession of blood in the artery. It also occurs in other conditions.

Diagnosis.—Physical examination by inspection, palpation, percussion, and auscultation, in the order named, prevents many errors in the interpretation of murmurs. The cardinal findings are: (*a*) Dilatation and hypertrophy of the left ventricle; (*b*) the *pulsus celer* and *altus;* (*c*) the arterial phenomena; and (*d*) the diastolic murmur.

Relative aortic regurgitation (see ETIOLOGY) is difficult to differentiate from the valvular; the murmur is not the typical protracted diastolic bruit, but rather a short presystolic (or postdiastolic) murmur (Rosenbach) which produces a short "huff" after the second aortic tone. This character is said to be almost pathognomonic. The pulse in relative regurgitation is frequently small, tardy, and tense, but not quick. Hypertrophy of the left ventricle is not very great. The murmur varies from time to time; it is sometimes short, then longer, sometimes blowing, and again coarse, or it may be difficult to fix as to systole or diastole.

Diastolic murmurs are rarely *anæmic* (*cardiopulmonary or functional*). Clear instances exist, however, of bruits in the cava and other veins. These murmurs are uneven, and are stronger in the beginning of the diastole. *Aortic aneurysm* (*q. v.*) may cause confusion. In *mitral lesions*, the capillaries are poorly filled and the skin is cyanotic; in aortic regurgitation the arterioles are overfilled and the skin is red. In mitral disease, congestion of the lung, brown induration, and dilatation of the right heart obtain; these are absent in aortic leakage; a hemorrhagic nephritis takes the place of the analogous brown induration of the lungs of mitral lesions.

Relative healing by vicarious stretching of sound valves, by growing stenosis, and by calcification, is possible but infrequent.

IV. Aortic Stenosis.

Aortic stenosis is the most infrequent of left-heart lesions. It may occur congenitally, in which case the conus arteriosus is often constricted (Dittrich's genuine heart stenosis). It occurs, however, thus leading to increased engorgement and blood pressure in the pulmonary circuit, because (*a*) of this backward pressure, and (*b*) of the onward pressure from the right ventricle, which hypertrophies. The lungs are enlarged and engorged, their vessels dilate and may become atheromatous, possibly causing infarcts in the lung, and connective tissue development (brown induration). When the final inevitable weakening of the right ventricle occurs from dilatation, *i.e.*, broken compensation, the right auricle is engorged and relative tricuspid leakage (*v. i.*) follows from dilatation. The venous system is overloaded, and this results in stasis and cyanotic induration of the viscera, liver, and kidneys, and often in œdema.

Physical Signs.—1. Inspection.—(*a*) The *apex* may be normally located or *displaced to the left*—even into the axilla when there is much dilatation. The heart may then lie lower than normal. The normal force of the apex beat in mitral insufficiency is evidence of *hypertrophy*. (*b*) Systolic *pulsation in the second left interspace* indicates a wide pulmonary artery from increased tension or stasis, and (*c*) a *diastolic shock* there is due to closure of its valves; (*d*) *precordial prominence* occurs in yielding chests from marked cardiac dilatation; (*e*) *pulsation*, either epigastric or to the right of the sternum, results from dilatation and hypertrophy of the right ventricle. (*f*) The *veins are enlarged* and pulsate during the diastole from stasis, and sometimes during the systole from tricuspid incompetency (*q. v.*) or patency of the foramen ovale.

2. Palpation.—(*a*) The *apex is dislocated to the left;* (*b*) the *pulmonary pulsation* is felt; (*c*) the *diastolic shock* of the forcibly closing *pulmonary valves* is very often palpable by placing one finger over the valves and another over the apex whereon the two shocks alternate; (*d*) *pulsation* is felt to the *right;* (*e*) a *systolic apical thrill* is often palpable after physical or psychical excitement, or when the patient lies on his left side, but it is not pathognomonic. (*f*) In pure cases the *pulse* is of about *normal tension* in compensation, or becomes lessened with rupture of compensation. Irregularity is not very common and the sphygmographic tracing is not characteristic.

3. Percussion.—This shows (*a*) *dilatation of the right ventricle* in compensated cases, which is commonly followed by *hypertrophy*. Dilatation shows dulness and palpatory resistance to the right of the sternum (See Plate VIII, Fig. B). Hypertrophy without dilatation is usually found in the early and well compensated lesion. It should be remembered that the right heart bears the stress of this lesion. (*b*) The *left ventricle* usually but not always *dilates* to respond to the increased amount of blood, and *hypertrophies* because of increased work or because of the raising of arterial pressure by carbon dioxide.

4. Auscultation.—(*a*) The *systolic murmur* is due to abnormal eddies in the left auricle or to the vibrations caused by them in the mitral valves; it most often gives a *blowing* sound, is best heard *over the apex*, which is less covered by the lung, but at times is best recognized over the pulmonary area or auricle, where it really originates and where it is heard especially in early lesions. It is *transmitted chiefly towards* the *left interscapular* region, and to *the left;* and more rarely to the aorta, carotids, and abdomen (See Plate VIII, Fig. B); it is at times heard intermittently, in certain postures only, and it may disappear with weakening of the heart. In very rare cases it is entirely absent (latent). (*b*) The *first mitral tone* is usually absent because the valves cannot vibrate; if present it is referable to conduction from the tricuspid valve or to contraction of the ventricle. When obscured by the murmur, it may sometimes be heard by lifting the stethoscope a little from the chest wall. In cases of extreme leakage, the murmur replaces all tones. (*c*) The *second pulmonic sound* is accentuated, "hammer-like," and is a measure and result of the right ventricular hypertrophy (Skoda). It sometimes reaches the vessels of the neck. When the tricuspid valves become relatively insufficient, it usually weakens; accentuation is absent in those rare cases where the right ventricle does not hypertrophy. The second tone at the base is sometimes split. (*d*) A *systolic murmur* over the pulmonary area is usually propagated from the apex or is due to vibrations in the distended pulmonary artery.

Diagnosis.—The cardinal features are (*a*) the systolic murmur, (*b*) the accentuated second pulmonic tone, and (*c*) the right-heart changes.

Excepting the etiological diagnosis, there are no positive criteria by which we can differentiate a *muscular* (*relative*) from a *valvular insufficiency,* though in the former the murmur is more variable, lessens with digitalis, and is not associated with a diastolic murmur which is common in valvular incompetency. Pure insufficiency is very exceptional, being usually combined with some mitral stenosis into which insufficiency may eventually pass. (A cure is thus sometimes effected.)

From the *functional,* accidental, hæmic, or cardiopulmonary *murmur,* the diagnosis is usually easy. This murmur is inconstant, is often basal, often depends on posture and coughing efforts, is circumscribed, sometimes disappears on pressure, is not typically transmitted, follows fever, anæmia, and emphysema, is often associated with the venous hum in the neck, but is never associated with typical mitral changes in the heart tones and right ventricle. The intensity, *timbre,* or *frémissement,* of the murmur must not alone be depended upon. So-called "functional" murmurs are usually systolic.

In *chlorosis,* we may obtain a hæmic murmur, apparently large heart (because the lungs are poorly expanded), and a displaced apex, since the diaphragm stands high in chlorosis; deep breathing rapidly decreases the heart's dulness.

Pericardial friction (*q. v.*) may simulate any endocardial murmur (Skoda), though most clinicians find little difficulty in distinguishing them.

Fraentzel's general rules are excellent and aid in accuracy: (1) Never make a diagnosis just before death. (2) Remember the great infrequency of right-heart lesions. (3) Avoid complicated diagnoses. (4) Murmurs are subordinate in value to physical findings by inspection, palpation, and percussion.

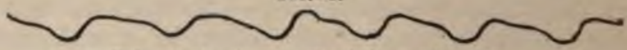
largely in the aged. The valves become interadherent or thickened, and calcarious masses or retraction at their insertion obstruct the orifice. Acute endocarditis, with massive vegetations, may produce some stenosis. It is usually associated with aortic incompetency, although aortic double lesions are far less common than mitral. Stenosis of the aortic orifice is a better term than aortic stenosis.

Mechanism.—The rigid valves make the orifice narrow, and at the obstruction a systolic murmur originates. The resistance to the onward flow of blood from the left ventricle, leads to its hypertrophy. The pulse becomes small and slow because of obstruction.

Physical Signs.—1. **Inspection.**—Some precordial prominence may be seen in yielding chests. The apex beat is sometimes strong, and at times somewhat dislocated to the left, but often no apex is seen, either on account of coincident emphysema or because the heart's recoil is lessened or absent. The apex sometimes retracts during the systole from lack of tension in the aorta and from lessened systolic dislocation of the heart.

2. **Palpation.**—(*a*) A most pronounced *systolic thrill* is often felt over the second right interspace, and sometimes is propagated over the heart and into the cervical vessels. (*b*) *The pulse* is infrequent (*pulsus rarus*), because the systole is prolonged and the coronary arteries are not well filled; it is small (*p. parvus*), from obstruction to its flow; it is tense (*p. durus*), from hypertrophy of the left ventricle, and from arteriosclerosis; and it is exceedingly slow (*p. tardus*) with tardy up-and-down stroke and some retardation, as compared with the apex beat. A *strong apex beat* and *a weak radial pulse* suggest aortic stenosis.

Fig. 23.



Pulse tracing in stenosis of the aortic orifice.

3. **Percussion.**—This may be wholly negative. Concentric hypertrophy (*i. e.*, hypertrophy, with the left heart chamber smaller than normal), is very frequent. Concentric and simple hypertrophy, which are the more common, as the stenosis is purer, naturally give no change on percussion. Dilatation is sometimes present, giving slight dulness to the left, especially with aortic regurgitation, renal contraction, or atheroma. In rare instances, hypertrophy and dilatation are both absent.

4. **Auscultation.**—Auscultation gives (*a*) a *systolic murmur* over the aortic area, which is sometimes soft but more often long, sawing, whistling, or musical, and is transmitted with the blood current into the aorta, the descending aorta in the back, and its vessels (Plate VIII, Fig. A). It is frequently heard over the entire heart, and at times is heard at some distance from the chest. It is not proportionate to the intensity of the lesion, and disappears with broken compensation. (*b*) *The second aortic sound is weak*, from poor filling of the aorta and weak closure of the altered valves, or it is *absent*, when combined with

aortic regurgitation; (*c*) when the second aortic sound is absent, the *second mitral tone*, or indeed all the heart tones, are *weak or absent*. (*d*) A systolic tone is heard over the aorta.

Diagnosis.—The lesion may be a favorable sign, because it is sometimes a healing of an aortic regurgitation. Aortic stenosis is a frequent surprise at autopsy, having run a *latent* course. The *cardinal symptoms* are (*a*) systolic murmur with propagation into the large vessels, which is frequently heard over the entire heart; (*b*) a systolic *frémissement* over the aortic area; (*c*) the small, tardy, slow pulse, in contrast to the strong apex beat. A diagnosis should be reserved unless these signs are present. As the lesion is uncommon, its frequent diagnosis implies *confusion* with (i) *accidental murmurs*, which are variable but which may resemble a slight stenotic murmur. (ii) *Acute endocarditic vegetations;* these usually give a soft murmur which becomes harsher if actual stenosis ensues. (iii) *Atheroma of the aorta*, or acute aortitis, where the murmur is more localized over the aorta, the second aortic tone is ringing or metallic, and the pulse is generally proportionate to the apex beat. Atheroma is the most frequent cause of diagnostic error; it is very common, while experienced clinicians see few cases of pure aortic stenosis. (iv) *Aneurysm* (*q. v.*), which is often distinguishable by its pressure symptoms.

V. Mitral Insufficiency.

Mitral insufficiency (incompetency, regurgitation) is the most frequent valvular lesion. It is (*a*) *valvular*, from adhesions between the valves or chordæ or ventricular wall, from vegetations, from thickening with retraction, from valvular aneurysm or perforation, or from shrinkage or rupture of the papillary muscles or chordæ; or it is (*b*) *muscular*, from relative insufficiency due to such relaxation of the ventricular muscle that the valves will not cover the orifice; it is most common in hypertrophy with dilatation, resulting from hard work, atheroma, nephritis, syphilis, alcoholism, myocarditis, adherent pericardium, and exophthalmic goitre; (*c*) rupture of the mitral cusps is very uncommon. Barié collected 35 cases of valvular rupture, of which 45.7 per cent. were aortic, 45.7 per cent. were mitral, and 8.6 per cent. were tricuspid.

Mechanism.—During the systole, a backward flow occurs through the incompetent valves into the left auricle, which, as it also receives blood from the lung veins, must first dilate and later hypertrophy, as far as its slight musculature can admit. The left ventricle dilates because it receives in the diastole more blood than normal (*i. e.*, the usual inflow plus the already regurgitated blood), and it hypertrophies because its work is increased. In aortic lesions, the first and greatest strain is felt in the left ventricle, but mitral lesions, by congesting the lungs, throw the most work on the right heart. The stasis in the left auricle is transmitted to the pulmonary veins, capillaries, and arteries, thus leading to increased engorgement and blood pressure in the pulmonary circuit, because (*a*) of this backward pressure, and (*b*) of the onward pressure from the right ventricle, which hypertrophies. The lungs are enlarged and engorged, their vessels dilate and may become atheromatous, possibly causing infarcts in the lung, and connective tissue development (brown induration). When the final inevitable weakening of the right ventricle occurs from dilatation, *i.e.*, broken compensation, the right auricle is engorged and relative tricuspid leakage (*v. i.*) follows from dilatation. The venous system is overloaded, and this results in stasis and cyanotic induration of the viscera, liver, and kidneys, and often in œdema.

Physical Signs.—1. **Inspection.**—(*a*) The *apex* may be normally located or *displaced to the left*—even into the axilla when there is much dilatation. The heart may then lie lower than normal. The normal force of the apex beat in mitral insufficiency is evidence of *hypertrophy*. (*b*) Systolic *pulsation in the second left interspace* indicates a wide pulmonary artery from increased tension or stasis, and (*c*) a *diastolic shock* there is due to closure of its valves; (*d*) *precordial prominence* occurs in yielding chests from marked cardiac dilatation; (*e*) *pulsation*, either epigastric or to the right of the sternum, results from dilatation and hypertrophy of the right ventricle. (*f*) The *veins are enlarged* and pulsate during the diastole from stasis, and sometimes during the systole from tricuspid incompetency (*q. v.*) or patency of the foramen ovale.

2. **Palpation.**—(*a*) The *apex is dislocated to the left;* (*b*) the *pulmonary pulsation* is felt; (*c*) the *diastolic shock* of the forcibly closing *pulmonary valves* is very often palpable by placing one finger over the valves and another over the apex whereon the two shocks alternate; (*d*) *pulsation* is felt to the *right;* (*e*) a *systolic apical thrill* is often palpable after physical or psychical excitement, or when the patient lies on his left side, but it is not pathognomonic. (*f*) In pure cases the *pulse* is of about *normal tension* in compensation, or becomes lessened with rupture of compensation. Irregularity is not very common and the sphygmographic tracing is not characteristic.

3. **Percussion.**—This shows (*a*) *dilatation of the right ventricle* in compensated cases, which is commonly followed by *hypertrophy*. Dilatation shows dulness and palpatory resistance to the right of the sternum (See Plate VIII, Fig. B). Hypertrophy without dilatation is usually found in the early and well compensated lesion. It should be remembered that the right heart bears the stress of this lesion. (*b*) The *left ventricle* usually but not always *dilates* to respond to the increased amount of blood, and *hypertrophies* because of increased work or because of the raising of arterial pressure by carbon dioxide.

4. **Auscultation.**—(*a*) The *systolic murmur* is due to abnormal eddies in the left auricle or to the vibrations caused by them in the mitral valves; it most often gives a *blowing* sound, is best heard *over the apex,* which is less covered by the lung, but at times is best recognized over the pulmonary area or auricle, where it really originates and

where it is heard especially in early lesions. It is *transmitted chiefly towards* the *left interscapular* region, and to *the left;* and more rarely to the aorta, carotids, and abdomen (See Plate VIII, Fig. B); it is at times heard intermittently, in certain postures only, and it may disappear with weakening of the heart. In very rare cases it is entirely absent (latent). (*b*) The *first mitral tone* is usually absent because the valves cannot vibrate; if present it is referable to conduction from the tricuspid valve or to contraction of the ventricle. When obscured by the murmur, it may sometimes be heard by lifting the stethoscope a little from the chest wall. In cases of extreme leakage, the murmur replaces all tones. (*c*) The *second pulmonic sound* is accentuated, "hammer-like," and is a measure and result of the right ventricular hypertrophy (Skoda). It sometimes reaches the vessels of the neck. When the tricuspid valves become relatively insufficient, it usually weakens; accentuation is absent in those rare cases where the right ventricle does not hypertrophy. The second tone at the base is sometimes split. (*d*) A *systolic murmur* over the pulmonary area is usually propagated from the apex or is due to vibrations in the distended pulmonary artery.

Diagnosis.—The cardinal features are (*a*) the systolic murmur, (*b*) the accentuated second pulmonic tone, and (*c*) the right-heart changes.

Excepting the etiological diagnosis, there are no positive criteria by which we can differentiate a *muscular* (*relative*) from a *valvular insufficiency*, though in the former the murmur is more variable, lessens with digitalis, and is not associated with a diastolic murmur which is common in valvular incompetency. Pure insufficiency is very exceptional, being usually combined with some mitral stenosis into which insufficiency may eventually pass. (A cure is thus sometimes effected.)

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Fraentzel's general rules are excellent and aid in accuracy: (1) Never make a diagnosis just before death. (2) Remember the great infrequency of right-heart lesions. (3) Avoid complicated diagnoses. (4) Murmurs are subordinate in value to physical findings by inspection, palpation, and percussion.

VI. Mitral Stenosis.

Pure stenosis of the mitral orifice is rare and is an almost constant anatomical associate of mitral leakage. The mitral valves are so grown together or the ring so constricted as to well justify the name used by Corrigan, "the button-hole contraction." The orifice in extreme cases is funnel-shaped. It is more frequent in the young and in women (76 per cent. of the cases).

Mechanism.—The stenosis obstructs the diastolic passage of the blood into the left ventricle from the left auricle, which consequently dilates and later hypertrophies. The enlarged auricle may compress the adjacent bronchus or recurrent laryngeal nerve. The stasis is propagated backward through the lungs to the right ventricle, whose dilatation and hypertrophy cause the large right heart and the increased pressure in the lesser circulation. The effects on the right heart and lungs are more severe than in mitral insufficiency. The right auricle also dilates when the later venous stasis follows. Thrombi in either auricle may dislodge and produce pulmonary or arterial infarction. The arterial system is naturally filled but poorly.

Physical Signs.—**1. Inspection.**—This reveals (*a*) *precordial distention*, (*b*) *epigastric pulsation*, and (*c*) a *diffuse cardiac shock;* (*d*) the *apex beat* may in comparison be *small*, and (*e*) two impulses or shocks may be seen in the second left interspace; one is systolic, from systolic filling of the pulmonary artery, and the other is diastolic, from pulmonary valve closure under high tension. All these findings depend on changes in the right heart.

2. Palpation.—Palpation confirms the above, especially (*a*) the apex beat, which is often weak in comparison with the diffuse heart impulse, (*b*) the snap of the second pulmonic sound, (*c*) the pulsating right heart, and also (*d*) a *presystolic* (more rarely diastolic) *thrill* (*frémissement cataire*) which is usually limited to the apex, ends with the *snappy first apex-tone*, and is felt sometimes only in the left lateral posture, after exertion or when the arms are raised. Its mechanism is identical with that of the murmur (*q. v.*). (*e*) The *pulse* is often *irregular* even with good compensation, especially in cases of pure stenosis, and is usually *small* and of *low tension*, because the aorta is poorly filled. It sometimes seems hard, and at times its weakness contrasts conspicuously with the active heart impulse. The pulse is not pathognomonic but merely suggestive; the sphygmograph confirms the palpatory findings.

3. Percussion.—(*a*) The *right ventricle* in the earlier stages may be only hypertrophied, which does not show on percussion. *Dilatation* without hypertrophy exists in few cases fortunately, for the prognosis is then poor. Most commonly hypertrophy and dilatation coëxist, the latter giving increased dulness to the right, as is the case in mitral insufficiency. (*b*) The *left ventricle* in many cases of dominant stenosis shows concentric atrophy, so that at autopsy it looks like a mere appendage to the huge right heart. It is sometimes both hypertrophied and dilated, for which the following reasons have been assigned: Marked concomitant mitral leakage; stasis with increase in the blood of carbon dioxide, which raises the blood pressure; coincident adherent pericardium, nephritis, atheroma, or aortic disease, all of which lead to changes in the left ventricle. (c) The *auricles* are distended, much of the dulness under or beyond the sternum being due to the dilated right auricle and that above and to the left, to the left ventricle. (The right conus arteriosus and the pulmonary trunk are found widened at autopsy.) Increased dulness, due apparently to dilatation of the various chambers, is often caused merely by their dislocation by the wide right ventricle.

4. **Auscultation.**—(a) An *apical presystolic murmur* is important. It occurs at the end of the diastole just before the ventricular systole, *i.e.*, during the auricular systole which forces the blood through the narrowed ring or rigid adherent valves. Corresponding with the energy of that chamber, it is loudest at the incipiency and ending of the murmur. It so closely precedes the ventricular systole that it is often called systolic by physicians and is almost invariably mistaken by students. It is rarely a blowing sound, but usually snoring, *rolling*, sounding like R-R-R-P, *after which* comes the loud first tone. It is poorly propagated as a rule, corresponding to the localized thrill (*v. s.*), but may in exceptional cases, be heard in the axilla or back. It is most variable, often being absent even with great stenosis, and appearing again only after exertion; it is absent when the heart weakens and before death, whence the importance of Rule 4 of Fraentzel (see DIAGNOSIS of MITRAL INSUFFICIENCY). It is rarely diastolic. (b) *Marked accentuation* of the *second pulmonic sound* is the proof and measure of hypertrophy of the right ventricle from pulmonary congestion. Its absence is a poor prognostic. It usually weakens when relative tricuspid leakage intervenes and thereby lessens the tension in the lungs. The accentuation rarely reaches the cervical vessels. (c) *Splitting* or *reduplication* of the *second pulmonic sound* is due to difference in tension in the two arterial trunks, the aortic valves closing first and more weakly. The doubling is best heard at the base and is often propagated into the neck or axilla. (d) The usually *loud first mitral tone* is explained in various ways; *viz.*, by difference in tension of the valves during the systole and diastole or by a vigorous contraction of the left ventricle upon little blood. It is sometimes heard at a distance of several feet. (e) The *second mitral tone* is weak because of poor filling of the aorta or because the right heart comes forward more than usual. This tone may be split at the apex (the "postman's knock"), and may be present early in this lesion; the cause of the splitting is the subject of variant discussion.

Diagnosis.—The cardinal signs are (a) the presystolic thrill and bruit; (b) the involvement of the right heart; (c) the accentuated second pulmonic sound; (d) the small pulse, which is less important and (e) loud first apical tone.

The thrill and bruit are also found in other conditions, as in aortic insufficiency (Flint's murmur), in which the vascular phenomena and the involvement of the left heart are sufficiently distinctive, though one out of every three cases of aortic leakage is complicated by mitral stenosis. The bruit has also been heard in lesions leading to dilatation of the left ventricle, such as adherent pericardium, whence the rule that the diagnosis of mitral stenosis must be conservative when there is a clear history of pericarditis or where the *left* ventricle is dilated.

When the thrill and murmur are absent, the diagnosis can very often be made by the other cardinal symptoms.

It has been held that (*a*) an absence of the murmur indicates a slight lesion, as also does (*b*) a presystolic murmur; (*c*) a diastolic murmur indicates a severe stenosis; and (*d*) absence of the murmur with irregularity is indicative of the most serious lesion.

DIFFERENTIAL TABLE OF THE COMMON CHRONIC VALVULAR LESIONS.

	AORTIC INSUFFICIENCY.	AORTIC STENOSIS.	MITRAL INSUFFICIENCY.	MITRAL STENOSIS.
ETIOLOGY.	Arteriosclerosis chiefly.	Often arteriosclerotic.	Endocarditis chiefly.	Ditto.
PULSE.	Pulsus celer. Water-hammer, collapsing.	Pulse, small, slow, tense.	No characteristic alteration—often normal.	Small and often irregular.
LEFT VENTRICLE.	Hypertrophied and dilated greatly; heaving apex.	Hypertrophy, usually concentric.	Some hypertrophy and dilatation.	No essential nor regular change; may be small.
RIGHT VENTRICLE.	No change.	No change.	Hypertrophy and dilatation marked.	Both marked, also in auricles.
MURMUR.	Diastolic; blowing over sternum near second rib; propagated toward apex.	Harsh systolic murmur over aortic area with systolic thrill; propagated into neck.	Systolic at apex, blowing, transmitted into axilla and behind scapula.	Presystolic (oftener than diastolic), apical, accompanied by presystolic thrill, fairly localized.
FIRST MITRAL TONE.	Usually weak.	Strong usually.	Usually present, perhaps covered by murmur.	Loud and sharp, clearly palpable and following murmur.
SECOND AORTIC TONE.	Weak or absent.	Weak only.	Somewhat weak.	Somewhat weak.
SECOND PULMONARY TONE.	Not accentuated.	Not accentuated.	Loudly accentuated, and often palpable.	Ditto; often split, reduplicated.
PERIPHERAL VESSELS.	Violent pulsation; capillary pulse; arterial tones; skin red.	Absent.	Absent. Skin cyanotic.	Absent. Skin cyanotic

In conjunction with this table, Plate VIII should be consulted.

VII. Pulmonary Insufficiency.

Pulmonary insufficiency, of which Barié has collected 58 cases, is most uncommon and is often combined with pulmonary stenosis or other rare right-heart anomalies. It is (*a*) *congenital*, resulting from fœtal endocarditis or from supernumerary, or rudimentary valves; (*b*) *acquired* from rheumatism, from sepsis, which destroys the valves by ulceration or from atheroma; or it is very rarely (*c*) *relative* from extreme intrapulmonary blood pressure, or from embolism of the pulmonary trunk or its branches.

Mechanism.—The mechanism is analogous to that of aortic insufficiency except that the right heart suffers instead of the left and the arterial phenomena are manifest in the area of the pulmonary artery.

Physical Signs.—1. **Inspection.**—The right heart is dilated and hypertrophied, and pulsates under and to the right of the sternum. It may slightly dislocate the apex to the right. A systolic dynamic pulsation may be seen over the pulmonary trunk, which is also dilated.

2. **Palpation.**—This determines the diffuse right-heart impulse in 20 per cent. of cases, and a diastolic thrill over the pulmonic area, though the apex beat is normal or weak and the radial pulse is practically normal.

3. **Percussion.**—Percussion confirms the schematic expectation that the *right ventricle* should be *dilated* because of the backward fall of the regurgitant column of blood and should be *hypertrophied* to maintain the onward flow under this burden. The left ventricle is normal or sometimes atrophic. (See Plate VIII, Fig. E.)

4. **Auscultation.**—A *diastolic murmur* is heard, which is propagated down the sternum over the right ventricle. This bruit may be confused with that of aortic leakage, aneurysm, a dislocated aorta, functional venous murmurs, congenital anomalies of the vessels, or patent foramen ovale. The *second pulmonic* and the second tricuspid tones are *weak or absent* and are replaced by the murmur. The bruit is but rarely heard in the neck and the normal second arterial tone is heard over the carotids (see AORTIC INSUFFICIENCY). A systolic murmur (heard in 25 per cent. of cases) over the pulmonary trunk is due to vibrations in its walls or to coincident stenosis of the pulmonary ring. A double tone has been heard over the lung, as well as variation in the respiration during the systole,—a capillary pulse in the pulmonary vessels.

VIII. Pulmonary Stenosis.

The etiology will be considered under diagnosis and under congenital heart lesions.

Mechanism and Physical Signs.—1. **Inspection.**—Inspection shows precordial bulging over the right heart; it also discloses a diffuse heart shock from hypertrophy and dilatation of the right heart. The apex beat is often weak or absent.

2. **Palpation.**—Palpation confirms the above and reveals a systolic thrill in the left second interspace. The pulse is negative or weak.

3. **Percussion.**—Percussion shows an area of increased *dulness* over the *right heart* from dilatation.

4. **Auscultation.**—On auscultation, a loud and usually whistling or musical *systolic murmur*, caused by the stenosis, is often but not always heard and the first and second pulmonic tones (and hence also the second tricuspid) are weak or absent from poor filling of the pulmonary vessel and from valve alteration. (See Plate VIII, Fig. E.)

Diagnosis.—The most frequent form is (*a*) the *congenital*, which is due to malformation, myocarditis, or endocarditis, and is usually associated with other kindred lesions (*e. g.*, open ductus Botalli or foramen ovale). The obstruction may be below the valves, from myocarditic scars narrowing the conus arteriosus, in which case the second pulmonic sound is accentuated; or it may be at the valves or in the trunk of the artery. Congenital cyanosis and acquired pulmonary tuberculosis are common. The murmur may be heard in the neck. (*b*) The *acquired* valvular form is rare. Atheroma or gummata may be causal. The thrill is also, though far less frequently, felt in *accidental murmurs* and a murmur over this area is most common, because the walls of the artery are so thin and compressible as to vibrate easily. These "functional" murmurs are so common in this locality as well to justify Fraentzel's rule to observe extreme caution in the diagnosis of right-heart lesions in the adult. Functional murmurs may occur (i) in health; (ii) after fevers; (iii) in anæmia; (iv) as the cardiopulmonary murmur. The *organic murmur* rarely reaches the jugular or cervical vessels. The left heart is frequently atrophic. When the acquired stenosis is beyond the valve in the pulmonary trunk or lungs, the murmur is heard most advantageously beside the right border of the sternum or in the back, and the second pulmonic sound is loudly accentuated, from which points a diagnosis may occasionally be made. In atresia of the orifice 80 per cent. of the subjects die in the first year.

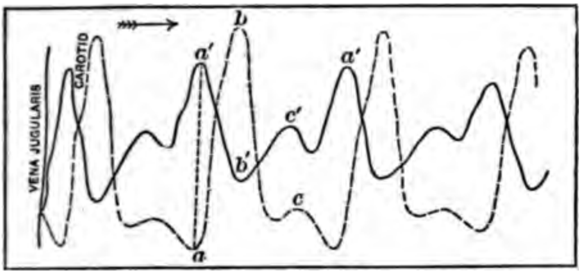
IX. Tricuspid Insufficiency.

Etiology.—(*a*) The *congenital* form is most rare, isolated tricuspid disease occurring in 0.8 per cent. of cases of valvular lesions. The venosity of the blood in the right heart tends to prevent endocarditis, except in fœtal life when the blood is arterial. Myocarditis or ulcerative endocarditis may exceptionally occur. (*b*) Of *acquired* forms, the *relative insufficiency* is very common, resulting from (i) valvular lesions of the left heart, especially mitral stenosis; (ii) obstructive lung lesions, such as induration or emphysema; (iii) possibly abdominal lesions which reflexly contract the pulmonary vessels, and (iv) in severe anæmias (Leube); in anæmic regurgitation cyanosis and dyspnœa are not observed.

Mechanism.—Whatever the cause of the high pressure in the lesser circulation, it leads to dilatation and hypertrophy of the right ventricle. Extreme dilatation stretches the tricuspid orifice so that the valves are incompetent to close it, and relative leakage occurs. The leak backward into the right auricle produces its dilatation and hypertrophy, a systolic murmur and a systolic pulse in the veins of the neck and liver.

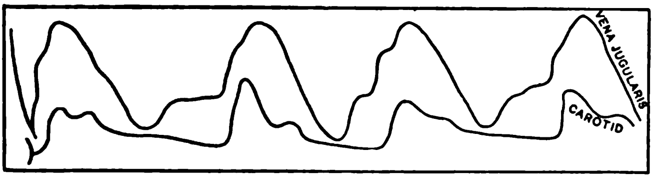
Physical Signs.—1. Inspection. (*a*) *A positive* (*systolic*) *pulse in the jugular veins* is absolutely pathognomonic and is due to systolic entrance of blood into the veins from the right ventricle through the insufficient valves. It is presystolic-systolic and is known as positive to differentiate it from the negative or diastolic venous pulse which is observed in many other conditions, and is either physiological, as on deep inspiration caused by temporary damming back of the blood by the auricular systole, or pathological, as in stasis. It is seen best on the right side because of the more direct (vertical) connection with the right innominate vein. A certain degree of heart force is necessary for its production, and it may cease therefore when the heart grows weak. Digitalis may cause it to reappear in cases of relative and sometimes of organic leakage. It is best seen when the patient is in the dorsal decubitus. To avoid confusion with pulsation transmitted from the carotids, the carotid should be compressed as low down as possible, or the pulsating jugular vein should be compressed at the middle; the upper part will then pulsate if the carotids beat against it (transmitted pulsation) and the lower part will beat if the pulsation is in the vein itself. Though it is seen most advantageously in the internal jugular vein, it is sometimes seen in the vena cava inferior or in the veins of the chest and extremities. It is difficult to determine when the heart is rapid or irregular. (*b*) *A liver pulse,* systolic in time, venous regurgitant in origin, is also pathognomonic of this disease. (*c*) *The right heart pulsates widely,* while the apex beat is weak.

FIG. 24.



Negative (diastolic-presystolic) venous pulse. (After Riegel and Sahli.)

FIG. 25.



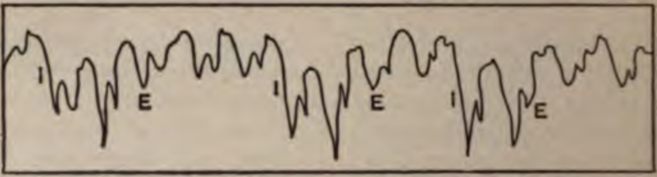
Positive (presystolic-systolic) venous pulse. (After Riegel.)

2. Palpation.—(*a*) This aids in determining the systolic phase of the venous pulse, and (*b*) it establishes the *systolic venous pulsation of the liver*, which expands in the bimanual examination. It is easier to find, and often earlier to appear than the jugular pulsation. It must not be confused with an arterial liver pulse, which occurs in cases of aortic leakage, exophthalmic goitre, and of liver inflammation, but is less expansile than throbbing. (*c*) A *systolic thrill* over the tricuspid area; (*d*) tones or thrills over the peripheral veins; and, (*e*) a weak apex and radial pulse are also found.

3. Percussion.—This shows increased dulness to the right, from distension of the right auricle and ventricle. Changes in the left heart may indicate an older valvular lesion which leads up to the tricuspid leakage. (See Plate VIII, Fig. D.)

4. Auscultation.—(*a*) A *systolic murmur* is heard over the tricuspid region; it may have a different quality from that of the causal mitral lesion. Unlike the murmur caused by mitral lesions, it is rarely heard in the back. The murmur is absent when the right heart is weak, because it then fails to produce the necessary vibration or eddy. (*b*) The *second pulmonic sound is weak* because the pulmonary circuit is poorly filled. This tone often varies with the changing activity of the right ventricle. It may remain accentuated even with tricuspid leakage when the ventricle is fairly strong. (*c*) Over the *cervical veins* may be heard a systolic tone referable to closure of valves in the veins; or the tone may be double, or presystolic-systolic; or venous murmurs may take the place of the single or double tone.

FIG. 26.



Illustrating the influence of respiration on the positive (presystolic-systolic) venous pulse of the jugular vein. (After Kovacs.)

Diagnosis.—An isolated lesion is most rare. The relative insufficiency is diagnosticated by means of (*a*) the etiology, (*b*) the large right heart, (*c*) the systolic murmur, (*d*) the positive venous pulse in the neck, and (*e*) the weak second pulmonic tone. Even when tricuspid incompetency is apparently the only lesion, it should be remembered that rest or digitalis very often brings out a causal mitral lesion. Much is said regarding the "safety valve" action of the tricuspid valves, which is hard to understand. Guarding as they do the door between the venous, and arterial circulation, their insufficiency indicates most serious cardiac failure.

X. Tricuspid Stenosis.

Tricuspid stenosis is rarer than tricuspid insufficiency; it occurs alone in only 10 per cent. of the cases; it is often associated with insufficiency, or with mitral or aortic stenosis. Obstruction of this orifice induces hypertrophy and dilatation of the right auricle, and this produces dulness to the right of the sternum. In the single lesion the other heart chambers and the large vessels are small. The pulse is weak. The second pulmonic sound is weak; a diastolic or presystolic murmur and thrill to the right of the sternum are detected if the heart is sufficiently strong, but they are often absent. The lesion is most serious, because it taxes the weak auricle and greatly engorges the venous system. The diagnosis is most difficult; indeed it is usually impossible. (See Plate VIII, Fig. D.)

XI. Combined Valvular Lesions.

These occur in 33 per cent. of valvular cases. From an anatomical standpoint, a pure lesion is rare, and double lesions are found oftener than physical signs would indicate. Pure lesions occur most often on the arterial valves. One lesion may follow another; for instance, stenosis may gradually result from insufficiency sometimes with good results; or combined lesions may develop simultaneously.

(a) *Combined mitral* lesions are most frequent, though one lesion usually predominates; (b) *combined mitral and aortic lesions, e. g.*, aortic vegetations reaching the nearest mitral cusp, are next in frequency and include 71 per cent. of the cases of combined lesions; (c) *double aortic lesions* are less common; (d) *combined aortic, mitral and tricuspid lesions, e. g.*, stenosis of each orifice with no signs other than slowing of the circulation, occur in 16 per cent., and (e) *combined mitral and tricuspid lesions* (especially relative tricuspid insufficiency) occur still less often (in 9 per cent.).

Diagnosis.—The diagnosis depends upon the character of the pulse, the quality and direction of transmission of the different murmurs, and the hypertrophy and dilatation of the right or left heart. Skoda's rule was that a murmur is usually most intense at its point of origin except in cases of aortic insufficiency; and Oppolzer held that when listening to the mitral and pulmonary valves, murmurs transmitted to them disappear as examination is made more to the left, though murmurs due to lesions on these valves remain; on listening further to the right, the true tricuspid and aortic murmurs remain and those which are transmitted disappear. In diagnosis, the seeking out of the *main lesions* and the avoidance of too complicated diagnoses lead to the most practical results.

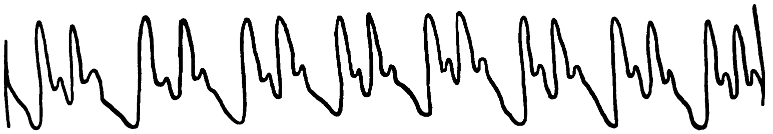
In cases of combined aortic and mitral disease: (a) *Aortic insufficiency* and *mitral insufficiency* lead to dilatation and hypertrophy of both ventricles; the combination is frequent in children; the aort findings as to pulse are frequently modified: the mitral insufficien may be relative (muscular) or organic (valvular), and this can determined by close and continued observation only. (*b*) *Aort stenosis and mitral insufficiency* is next in frequency and is the mo common combination in adults; less blood reaches the arteries than simple aortic stenosis; the right heart suffers disproportionatel whence the severity of this complication. The systolic murmu usually vary from each other in location, transmission, and qualit (*c*) *Aortic insufficiency and mitral stenosis* is a relatively frequent a favorable combination, because the left ventricle is usually less dilate both may produce presystolic apical murmurs and therefore the sta of the right heart and the second pulmonic tone are all-importa diagnostically. (*d*) In combined *aortic and mitral stenosis,* the great the aortic stenosis, the more is the hypertrophy of the left ventricl the more dominant the mitral stenosis, the less is the hypertrophy the left ventricle, and the greater is the dilatation and hypertrophy the right ventricle. Differentiation is often difficult.

General Symptoms of Valvular Disease.—The heart possesses n only a certain *reserve power* but in favorable instances its musculatu *hypertrophies.* Symptoms may easily be lacking in the early stage but later compensation to meet the lesion may fail, either tempo arily ("disturbed compensation"), or permanently ("loss or ruptu of compensation"). This is the *asystolie* of French writers. Eve hypertrophy is more or less temporary in its compensation, and ult mate cardiac failure is inevitable. All lesions, practically speakin tend to reduce arterial tension, increase venous pressure, retard t capillary flow and promote embolism. Circulatory disturbances ma be spontaneous or induced by psychical excitement, physical exertio intercurrent disease, recurrent endocarditis, malnutrition, excessi use of alcohol or tobacco, or changes in the heart muscle. The ge eral symptoms and physical findings are more or less common to a lesions, but some are more frequent in certain forms of valvula disease.

1. Constitutional Symptoms.—Anæmia is frequent (aortic insuff ciency); polymorphonuclear leukocytosis is present in half the mitr cases; Andral spoke of a "heart cachexia"; the body temperature normal or subnormal from slow circulation. Fever is due to recurre endocarditis, embolism, or intercurrent disease. The nails are ofte clubbed in congenital lesions. Skin hemorrhages are due to "blo dissolution" or less frequently to embolism.

2. Cardiac Signs and Symptoms.—Apart from actual change in t chambers, such as dilatation and hypertrophy, the heart itself is n well nourished in "breaking compensation," for it receives less arteri blood and is engorged with venous blood. The *heart muscle* is in so cases fatty, and in others is the seat of myocarditis about the arteries some cyanotic induration about the veins. These changes are less th cause than the effect of lack of compensation; the autopsy and th microscope may fail to demonstrate the cause of broken compensation. Here and there pressure atrophy occurs, as in the papillary muscles (aortic regurgitation). The *rhythm* is frequently disturbed, especially in mitral lesions (stenosis). The sphygmograph sometimes shows two ventricular contractions occurring very close together (pulsus bigeminus); when the pulse intermits to the finger, the tones of the abortive contraction may be heard over the heart with the stethoscope, the weaker wave failing to reach the periphery. In general, the pulse beats are fewer than the heart beats. Four tones corresponding to the two heart beats are sometimes heard. The *gallop-rhythm* (Bouillaud 1847) is sometimes heard. There may be such irregularity that tones and murmurs are indistinguishable (*delirium cordis*). The pulse is slow (the *bradycardia* of aortic stenosis) or faster than normal, *tachycardia* (mitral lesions).

FIG. 27.



Pulsus bigeminus.

Among the complications are pericarditis (aortic lesions) and recurrent endocarditis. *Palpitation* is frequent, either continuously or intermittently, and is sometimes induced by lying on the left side. It may be absent with the greatest heart alteration (as in aortic insufficiency). A sense of tension over the heart is not infrequent, and *pain* like that of angina pectoris occurs in aortic oftener than in mitral disease (Nothnagel).

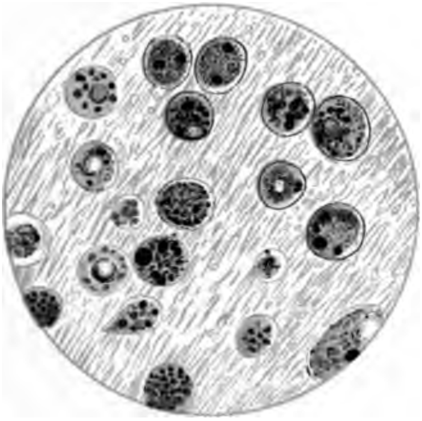
3. **Nervous Symptoms.**—These are not frequent. Syncope (aortic stenosis especially) occurs from arterial anæmia of the brain. Venous congestion is evidenced by repeated epistaxis, or vertigo, tinnitus, or *muscæ volitantes*. Brain hemorrhage is infrequent (mostly in aortic insufficiency). Chorea, epileptiform seizures, and embolism of the Sylvian arteries are sometimes observed as well as psychoses; the latter are delirium, mania, melancholia, and suicidal tendencies, and are usually of unfavorable import; stasis, inanition, carbon-dioxide narcosis and renal inadequacy are their cause. *Retinitis* hemorrhagica and simple hemorrhages result from stasis and anæmia.

4. **Respiratory Symptoms.**—Lung stasis is sometimes confused with independent lung disease on account of dyspnœa, cough, and expectoration. *Cyanosis* occurs more often in mitral than in aortic disease and is most intense in lesions of the pulmonary orifice. *Bronchitis* and *hæmoptysis* are most common in mitral affections. Bronchitis results less from congestion in the lesser circuit than from congestion in the cava superior, into which (and the azygos) the bronchial veins empty.

Though usually bilateral, it may predominate on one side, from such local causes as pleural adhesions. Hæmoptysis results from lung stasis with vessel rupture, or from embolism emanating from right-heart clots or thrombosis in the pulmonary and less frequently in the peripheral veins. The *sputum* may resemble that of pneumonia, though it is darker, more venous, and less viscid. It contains the so-called "heart disease cells" (E. Wagner). They are large granular delicate cells with oval nuclei, which often contain hæmosiderin or more rarely needles or plates of hæmatoidin; they are probably alveolar epithelial cells (possibly leukocytes) and also occur in other lesions than brown induration (as emphysema, asthma, pneumonia, and phthisis). See Plate IX. *Pulmonary œdema* is either (*a*) due to weakening of the left ventricle while the right heart beats more strongly or (*b*) far less frequently is inflammatory. Crepitant rales are frequently heard. In mitral disease the *lungs* frequently become *distended* and rigid from engorgement of the capillaries, and somewhat resemble the lung in emphysema (von Basch's *Lungenschwellung und Lungenstarrheit*). *Brown induration* (cyanotic induration) of the lungs has for its anatomical basis, (*a*) capillary engorgement often with fatty change in the intima, (*b*) increased inter-alveolar and perivascular connective tissue, (*c*) hemorrhage into the lung from vessel rupture, (*d*) consequent pigmentation by the altered blood-pigment of the lungs and sputum, and (*e*) the "heart-disease cells." *Dyspnœa*, the "cry of distress of the myocardium" (Sansom), constant, intermittent, or nocturnal, is caused by unbalanced slow circulation, the bulging of the turgid capillaries into the alveoli, pressure of the heart on the lower left lobe or left bronchus, ascites, hydrothorax, œdema, bronchitis, or infarcts. It may come on even without effort on the part of the patient when there are constipation and poor digestion; or it may occur especially at night when the voluntary deep and diaphragmatic respiration of the waking hours is not in play, whence the respiratory centre becomes anæmic. The dyspnœa is not expiratory as in asthma but is mixed in type (see CARDIAC ASTHMA under CARDIAC NEUROSES.) The wheezing which is heard is due to pulmonary œdema. The gallop-rhythm is rarely absent, and this threatens relapse of the dyspnœa. Arteriosclerosis and nephritis are important elements in its production. Exertion not only increases the heart's work but the carbon dioxide and fatigue substances from the muscles irritate the respiratory centre, and the respiratory muscles ultimately become tired. The breathing is more rapid. It may be *Cheyne-Stokes* in type (aortic stenosis); cerebral anæmia, arteriosclerosis, and uræmia are its causes. Hypostasis and lobular pneumonia may develop. *Hydrothorax* may hasten death within a few hours (Andral).

5. Gastro-intestinal Changes.—Gastralgia, anorexia, hemorrhagic infiltration and erosion, vomiting or hæmatemesis, result from passive congestion and may be mistaken for independent gastric disease, such as cancer or ulcer. Delayed digestion and constipation are usual. The secretion of hydrochloric acid is normal except in cases of marked

PLATE IX.



Heart Disease Cells, showing Alveolar Epithelial Cells, Loaded Down with Granules of Hæmatin. (Simon.)

weakness of the heart. Intestinal hemorrhage may result from capillary congestion, rarely from embolism of the mesenteric artery, and most infrequently from mesenteric venous thrombosis.

6. **Changes in the Liver.**—The liver suffers stasis through the congested cava and hepatic vein; this leads to cyanotic distention of the central veins of the liver lobules (appearing dark) and to cloudy and fatty degeneration of their periphery (appearing lighter), the "nutmeg liver." This occurs particularly in mitral disease. The liver is not uniformly affected. Pigmentation occurs and connective tissue develops in the liver and its capsule (perihepatitis, "iced liver"). Small granulations develop on its surface and in some instances a liver shrinkage results from "cyanotic induration" (cardiac cirrhosis, chronic central red atrophy). The liver is symmetrically *large* before œdema appears; when disturbed compensation is being restored, it is one of the last symptoms to disappear. Occasionally, from local changes, there may be tumor-like enlargement of the liver, which may simulate cancer. There is a *feeling of tension* and weight in the right hypochondrium and the organ is *tender* and *variable* in size; it may become smaller from rest or from administration of digitalis. Its edge is rounded and not hard except in the late stages. (See DIFFERENTIAL TABLE OF HEPATIC DISEASES). The *icterus viridis* develops gradually and is a combination of mild icterus and cyanosis; icterus is rarely intense but is of the "urobilin" type; it is rarely sudden except in the infrequent embolism, which may cause a clinical picture resembling acute yellow atrophy. *Ascites* is usually late and secondary to the liver congestion; its early appearance is indicative of independent liver disease or peritoneal affections. It is sometimes chyliform.

7. **Changes in the Spleen.**—The spleen is sometimes increased from embolism or liver disease, but is often harder and smaller than normal.

8. **Changes in the Kidneys.**—The kidneys are enlarged from hyperæmia, the capsule strips readily, and scattered areas of blood extravasation are seen. The *stellulæ Verheynii* are prominent, as also are the glomeruli. Some epithelial degeneration from malnutrition is seen and, in cases of long standing, "cyanotic induration" occurs. Deformity from the stellate scars of healed embolism and fresh infarcts are often encountered.

Renal symptoms are greater, the higher the venous and the lower the arterial blood pressure. The urine is decreased—its daily amount being an accurate gauge of the heart's force; it is of higher specific gravity (1025 to 1030), very acid, and is highly colored from urates and increased uric acid (even an eight-fold increase); the urea is decreased and also the chlorides when œdema exists; albumin is frequent (at the most 2 per 1,000[Krehl]), and there may possibly be a few red disks though these are more usually due to embolism or acute nephritis. The kidneys are slow to eliminate methyl blue or potassium iodide. Krehl has found granular as well as hyaline casts, contrary to the usual experience. (See DIFFERENTIAL TABLE OF DIFFUSE RENAL DISEASES.) Chronic nephritis may coëxist. Rest and digitalis increase the amount of urine and decrease or remove the albumin. In women, menstruation is frequently increased by passive congestion.

9. Embolism.—Embolism occurs as it does in acute endocarditis but its significance and effects are wholly mechanical. Embolism occurs in about one-third of the cases. Though it is most common in the kidney (77 per cent.) and spleen (54.7 per cent), it also occurs in the arteries of the brain (22.7 per cent.) (left Sylvian artery), in the extremities especially, in the branches of the left iliac artery, in the retina, liver, aorta (with paraplegia), and mesenteric vessels (in 1 per cent.). In the lungs it results from right-heart clots or thrombosis in the large peripheral or pulmonary veins. The special visceral features of embolism will be discussed under Diseases of the Brain, Lung, Kidney, etc.

10. Œdema.—This is a result of venous stasis, transmitted to th lymph-vessels and interspaces, and is also an evidence of changes i the vessel walls from actual blood alteration, for in some cases at lea the blood is thinned (hydræmia). It appears first in the connecti tissue of the lower extremities (right leg); it disappears over nigh and it extends upward to the vulva or scrotum, abdomen, chest a serous sacs (ascites, hydrothorax and hydropericardium) and brain ve tricles. The loss of fluid to the system is unfavorable; the anasarca a impedes the arterial circulation, threatens life from involvement of glottis or lungs, increases the tendency to secondary infections (erysi las, cellulitis, ulceration, or gangrene) and may lead to hypertrophy the connective tissue about the genitalia (cardiac elephantiasis). It most common in mitral disease. The prognosis is less favorable if th liver becomes very large before œdema appears.

11. Changes in the Joints.—Swollen joints and tabes dorsalis (aor insufficiency), are occasionally associated with valvular disease.

Course and Prognosis of Valvular Disease.—Patients may live f years with compensated valvular affections; a case of aortic leaka lived for twenty-five years and a case of mitral leakage lived for sixt six years. As a rule the course is chronic and unfavorable, and influenced by the following conditions:—

1. State of the Heart Muscle.—This muscle is the basis of hype trophy and compensation. Myocarditis is most unfavorable whethe associated or not with coronary disease, for digitalis has then no effe on the heart muscle. In cases where digitalis has immediate effect, th prognosis is favorable. The size of the heart is more important tha valvular murmurs. Great dilatation or dilatation without hypertroph is most unfavorable.

2. Sex.—The prognosis is generally better in women, because the are less exposed and less subject to physical strain and corona disease. Pregnancy as a rule is a rather unfavorable complicatio Peter advises, "In girls, (with valvular lesions) no marriage; in wive no pregnancy; in mothers, no nursing."

3. Age.—In the first decade of life the prognosis is poor, even thou the heart and coronary vessels are young, because hypertrophy ar dilatation are extreme, exercise is difficult to regulate, the valvular lesion tends to recur and the endocarditis tends to develop into pericarditis or pancarditis. After puberty the outlook is better, except, of course, in congenital types.

4. **Valve Involved.**—Opinions vary widely; for instance, aortic insufficiency is rated as the most favorable form by some, and as the most unfavorable by others. This may depend on the type, the outlook being fair in the endocarditic and most ominous in the atheromatous type. The best clinicians disagree as to the relative outlook in mitral and aortic lesions, although subjects of mitral insufficiency seem to have the more favorable prognosis. Broken compensation is more likely to be restored in mitral insufficiency than in mitral stenosis or in aortic lesions. Mitral stenosis is less favorable, although it is often found in women at an advanced age. Aortic stenosis is also found at an advanced age. Combined or multiple lesions, with some few exceptions, are unfavorable. The writer observed, in a young physician, a complete recovery from a double mitral lesion; in eight years after his causal rheumatism the outlines and tones became perfectly normal. Pulmonary lesions give a tendency to early tuberculosis.

5. **Mode of Life.**—The social scale, hygiene, necessity of hard work, psychical excitement, alcoholism, exposure, and the individual constitution, are most important factors.

6. **Unfavorable Symptoms.**—Cardialgia, angina pectoris, embolism, persistent palpitation, dyspnœa, and irregular or intermittent pulse are unfavorable symptoms. The "reserve heart power" of Martius is not present in all cases; this is due to failure of the coronary vessels to increase their calibre (Verney).

7. **Favorable Conditions.**—As described by Sir Andrew Clark, these are good general health, just habits of living, and absence of special rheumatic or catarrhal tendencies, endocarditic (not atheromatous) origin, no changes in the heart after three years' observation, sound ventricles and arteries, and freedom from stasis in the lungs, liver, and kidneys.

8. **Sudden Death.**—In most cases, sudden death occurs in aortic affections from coronary disease, but it may also result from cerebral or pulmonary embolism, from thrombosis cordis occluding an orifice, from heart rupture, from sudden ventricular dilatation, and from glottis œdema.

9. **Intercurrent or Associated Disease.**—Tabes and chorea have been mentioned. Valvular disease does not exclude cancer nor typhoid, as was once claimed. The acute fevers greatly tax the myocardium. Tuberculosis, which is said to be rare except in pulmonary valve lesions (since the lungs are hyperæmic), is found in 8 per cent. of autopsies on valvular subjects.

Treatment of Valvular Disease.—(**A**) **In Compensated Lesions.**—In compensated lesions medicinal treatment is usually unnecessary. With some exceptions, the patient is informed of the nature of his trouble that his coöperation may be enlisted. The *mode of life* is important, *i. e.*, regular orderly living, as free as possible from psychical or sexual excitement, worry, or hurry. Especial care should enforced in aortic cases because of danger of sudden death. Mu depends on the *social status* of the patient. Plenty of sleep is advisabl although heavy sleep after meals is said to promote fatty degeneratio The *diet* should be simple but not too schematic. *Tobacco* should interdicted, but like *alcohol* can sometimes be given with care ar moderation; beer and champagne are but poorly tolerated, thoug some red wine and brandy or cognac are permissible, especially adults and in the aged. Coffee may be given in the morning but shou not be strong, for cardiac irregularity frequently results. A breakfa of milk, with a little liquor, may be taken before rising, and the no meal should be the heartiest of the day. Nitrogenous food is be with such light carbohydrates as toast; foods which ferment, such cabbage, beans, or brown bread, should be avoided and the amount fluid ingested should be moderately restricted. Those things whi load the stomach heavily, are prone to elevate the diaphragm, ar every meal somewhat taxes the heart. A light meal at night is the be precaution against gastric and cardiac oppression during the sleepi hours. A milk diet is recommended in cases of stasis, especially wh there is much hepatic congestion, but it necessitates the ingestion of t large quantities of milk.

Exercise.—Exercise must be rational, and in moderate cases is advi able, for the muscles can not only accommodate all the blood of t body, but also by their activity reflexly stimulate the heart muscl Palpitation, recurrent endocarditis and intercurrent fever necessita keeping the patient in bed. Moderate *massage* is beneficial, but ca must be taken lest it produce dilatation of the abdominal vesse Deep breathing and singing are also beneficial. Plethoric patien should be given an enema of *salines*, which should be allowed to r main in the bowels over night. Tepid baths are more beneficial th those which are cold or hot.

Climate.—High altitudes are to be avoided, although modera elevation is tonic. The seashore often disagrees with rheumatic catarrhal subjects. A warm climate should be chosen for the winter.

(*B*). **Broken Compensation.**—Broken compensation may be acu gradual or terminal, as by coronary occlusion, and is evidenced dyspnœa which develops on exertion or spontaneously during t night, by anæmia, by irregular pulse (of special significance in aort cases), and by dilatation. The indications are as follows:

1. Absolute Rest in Bed.—This is frequently curative, especial in chlorosis or in pregnancy. Some patients are obliged to sit because of dyspnœa. Liebermeister's case of severe cardiac disea which recovered while in bed with a broken leg, is an example of co pensation restored without digitalis or other drugs. Rest is imperati in intercurrent fevers, bronchitis, recurrent endocarditis, and acu cardiac insufficiency. Bed sores can be prevented by the use of wat cushions, and by cleanliness. The head may be propped up by bac rests, but they should have side supporters so that falling to sleep w

not waken the patient. Rest should be combined with purgation (see below).

2. SALINE PURGATION.—Epsom or Glauber salts ℥ss-j in concentrated form should be given in the morning or compound jalap powder ʒss-j at night. These and kindred remedies deplete or bleed, as it were, the portal circulation and relieve the heart. Constipation *per se* often increases the size of the left heart.

3. VENESECTION.—This is necessitated by extreme venous engorgement evidenced by cyanosis, orthopnœa, dilatation, or tricuspid leakage. Six ounces to nearly a quart may be drawn. The effects are too often considered transient although the symptoms return sooner or later. In two cases apparently dying of valvular disease, where no blood could be obtained from the arms, the external jugular veins were opened, and the patients lived more than a year afterward. Venesection is not to be left until the patient is *in extremis.* It may be necessary to have the patient "actively move the arm so as to bring the blood from the capillaries into the veins" (Liebermeister).

4. THE OERTEL AND SCHOTT METHODS.—(See TREATMENT OF DISEASE OF THE HEART MUSCLE.)

5. DIGITALIS.—Digitalis was first recommended by Withering, and its physiological action demonstrated by Traube. Its *general indications* are (*a*) *dilatation:* digitalis gives or restores tone to the heart-muscle and its ganglia, and upon these the drug exercises its main influence; (*b*) *functional dissociation:* it is claimed that the ventricles have different innervation and that the drug is used most efficaciously when the pulmonary and venous circulations are engorged from too weak systole of the right and too strong systole of the left ventricle. When the *pulse is irregular,* digitalis coördinates the cardiac energy by action on the heart muscle and its ganglia or upon the terminal branches of the vagus. (*c*) *Rapid, weak, heart action:* digitalis slows the heart by action on the vagus and by increasing arterial pressure. It tends to abolish abortive cardiac contractions (*v. s.* pulsus bigeminus), and synergizes the systole of the chambers, as is shown by the strong systolic spasm of the heart in cases of digitalis poisoning. The slower and stronger systole empties more completely all the heart-chambers, forces the venous and arterial blood into the main arteries, and squeezes the venous blood out of the heart muscle itself, thereby improving the intrinsic heart circulation. The slower and more complete diastole allows of better filling of the heart and drainage of the engorged veins. Digitalis raises the arterial tension by increasing the working power of the heart and by its centric and local action on the vasomotors.

In the individual lesions: (*a*) in *mitral stenosis,* the longer diastole drains the left auricle and lung vessels and the stronger systole forces the retarded blood through the stenotic orifice; (*b*) in *mitral insufficiency,* the best results are obtained; the leaking cusps are better coapted and the stronger systole fills the aorta and pulmonary artery; (*c*) in *aortic stenosis,* when dilatation occurs or the hypertrophy is inadequate, digitalis is valuable; (*d*) in *aortic regurgitation*, it is theoretically contraindicated since it increases the diastole and hence the leakage; it is however practically valuable.

Digitalis is *contra-indicated* (*a*) in balanced compensation; (*b*) in adequate hypertrophy; (*c*) unless rest and purgation have first been tried; (*d*) when the vessels are fragile, as in aneurysm and advanced atheroma (on account of danger, *e. g.*, of brain hemorrhage); (*e*) in fatty degeneration of the heart, except when resulting from stasis and malnutrition; (*f*) in vascular contraction. It is in this case combined with nitroglycerine, strophanthus or potassium iodide, for the purpose of "unlocking the arteries" (Balfour). The drug is not absolutely contra-indicated in the high tension of arteriosclerosis and nephritis for it may relieve the condition, paradoxical as this may seem, by lessening dyspnœa and carbon-dioxide narcosis. (*g*) In idiosyncrasy to the drug, *e. g.*, when there are gastrointestinal symptoms which, however, are more often due to stasis. Digitalis also causes contraction of all unstriped muscular tissue (gastric, muscular). It may cause insomnia delirium, headache, diplopia, blindness to green colors, etc. Digitalis is said to dilate the left and contract the right coronary artery; therefore when the right coronary vessel is diseased, it may dilate, and cause excessive contraction of the right ventricle and thus dissociate the heart's action.

Cumulative action is averted (*a*) by intermittent administration, although some patients tolerate digitalis continuously for months or years, as Thomeyer's case who used digitalis eight and a half years taking in all over 15 ounces of the solid digitalis; (*b*) by great care in ambulatory cases; (*c*) by care in cardiac dropsy, on relief of which toxic quantities may be resorbed from the tissues; (*d*) by care in intercurrent febrile affections, for toxic effects often follow the fall of temperature; (*e*) by withdrawal on the appearance of vomiting syncope, reduced urine or irregular pulse (with twice as many heart as pulse beats). Twenty-seven cases of pulsus bigeminus from the use of digitalis are reported.

Digitalis may fail to operate unless free purgation, venesection, and mechanical removal of the anasarca have been first employed.

Preparations.—Some physicians prefer the powder, gr. j, others the tincture, ♏xv to xx, or the infusion ℥ss-j. The *infusion* is considered more diuretic (containing digitalein and digitonin).

℞

Infusi digitalis	℥iij.
Spts. ætheris nitrosi	ʒj.
Potas. bicarbonatis	ʒss.
Aquæ	℥ij.

S.—To be taken after meals.

Larger doses may be given though with risk. It is excellent for rectal use when the stomach is irritable. The *tincture* is thought to be the best cardiant because it contains digitalin and digitoxin. The writer has found digitalin almost inert. *Digitoxin* has seemed of doubtful value, but in the author's hands it has often slowed and strengthened the weak and irregular heart (gr. $\frac{1}{400}$ t. i. d.), when digitalis has failed; it easily disorders the stomach, and often cannot be given for more than six or eight doses. Digitoxin is often advantageously combined with champagne. For hypodermic use, the fluid extract, ♏ij to iv, is a non-irritant. Cloetta's digalen is a soluble non-irritant digitoxin, and may be given intravenously, 1 c.c. equalling 0.3 mg., or $\frac{1}{200}$ grain. The writer believes the preparation acts promptly, though it is somewhat irritating when given hypodermatically.

6. OTHER CARDIANTS.—(*a*) *Strophanthus*, ♏v to x of the tincture every eight hours, may be given. *Advantages:* it is more rapid in action than is digitalis, has no cumulative effects, perpetuates the effects of digitalis after the first five or seven days, and is indicated especially in the irregular heart of mitral disease, in children under twelve years, in cases where we are waiting for the digitalis to act, and in dilatation where the blood pressure is high. *Disadvantages:* it is less certain and less energetic; it is inferior in œdema and does not give tone to the vessels (which may be an advantage in atheroma). (*b*) *Strychnine* stimulates the vagus and heart muscle, besides being a splendid vascular tonic (see PNEUMONIA, TREATMENT) and tonic to the nervous, gastric, and blood-making tissues. Given carelessly, it may irritate the heart, decrease the urine by spasm of the renal vessels, or even induce rupture of the vessels. (*c*) *Spartein sulphate* (gr. $\frac{1}{4}$ to $\frac{1}{2}$ every six or eight hours) is given in capsule because of its bitterness; larger doses may be given, gr. j to gr. iss; it seems to quiet the nervous system and regulate the heart better than other remedies (Sée) but is very inferior to digitalis. It does not contract the vessels. Convallaria and adonis are very unsatisfactory.

7. SYMPTOMATIC TREATMENT.—(*a*) *Dropsy.*—Saline or hydragogue *catharsis* often relieves moderate œdema. *Digitalis*, alone or combined with calomel or blue mass, and pulv. scillæ (Guy's pill) is often efficacious—

℞

Pulv. digitalis	gr. x.
Massæ hydrargyri	gr. vj.
Pulv. scillæ	gr. x.

M. et ft. capsulæ x.
S.—One after meals for one or two days.

Mercurials, which were recommended by Jendrassik, must be used with the greatest care. The author saw, in consultation, a case complicated by nephritis in which the tongue sloughed off after five grains of calomel had been given. Desperate stomatitis may result in alcoholic, myocarditic, or nephritic subjects. Excess of digitalis, it must be recalled, is held to suppress the urine by inducing renal spasm. It is better to use potassium salts, as potassium acetate gr. xxx, t. i. d., which are less abundant in the tissues than the sodium salts; the potassium acetate, *e. g.*, passes through the tissues and kidneys, taking fluid with it (Bunge). Huchard proved that the diuresis following digitalis was associated with increase of sodium chloride elimination. *Caffeine* with digitalis is beneficial in dropsy when the pulse is slow, the mind dull, and the digestion sluggish; it may cause nervousness or insomnia (therefore should be given early in the day), irregular pulse (therefore should be combined with rum or brandy), or biliousness; gr. v should be given, but not for more than three days—

℞

Caffeinæ citratæ	ʒss.
Sodii benzoatis	ʒss.
Aquæ	ʒiij.

M. et S.—One hypodermicful (ʒss), as indicated.

Diuretin (daily dosage of 60 to 90 grains) may have marvellous effects or may absolutely fail; chemically it is sodio-salicylate of theobromine; it contains 50 per cent. theobromin, and operates on the renal cells directly, and possibly also on the heart and vessels. *Canadian hemp* (fluidextr. apocyni ♏ xv) is hard to obtain pure but sometimes justifies Benjamin Rush's term, "a vegetable trochar." *Sweats* and the use of pilocarpine are both dangerous and injurious. Mechanical drainage is recommended, by scarification, by multiple knife or pinpricks, or by Southey's capillary tubes; but the best method is by a single long *deep incision* down to the bone. Sodium chloride should be excluded from the diet (see DROPSY, TREATMENT OF CHRONIC NEPHRITIS). Ascites, from heart disease alone, rarely necessitates puncture: it is indicated chiefly in coincident liver cirrhosis or in chronic peritonitis. Hydrothorax is far more important, and thoracic paracentesis should not be delayed when dyspnœa is urgent.

(*b*) *Anæmia.*—The administration of digitalis with iron, arsenic, and zinc phosphide often produces remarkable improvement; iron and arsenic may be useful without employing digitalis at all.

℞

Pulv. digitalis	gr. xx.
Ferri redacti	ʒss.
Ac. arsenosi	gr. j.
Zinci phosphidis	gr. j.

M. et ft. pilulæ, xx.
S.—One pill after meals.

Cod-liver oil is excellent in children.

(*c*) *Insomnia.*—It is often difficult to relieve insomnia or unrestful sleep, broken by nightmare or sudden "heart starts." Spts. chloroformi, spts. camphoræ, and spts. ætheris compos. āā ʒss are often helpful. Sulphonal (gr. xx in hot milk at bed-time) may be tried but is somewhat dangerous in stasis; not more than three doses should be given. Chloralamide may be tried, but morphia is most efficacious.

(*d*) *Dyspnœa.*—The ice-bag, quinine and nitroglycerine gr. $\frac{1}{100}$ (though it gives better results in 5 per cent. alcoholic solution), are of value for high tension. *Morphine* may be given in the early stages, and without hesitation, and especially at night, for restlessness and dyspnœa; it is excellent while waiting for digitalis to act. It is almost specific and lessens the irritability of the respiratory centre. It is dangerous when the heart weakens, the lungs become œdematous, or Cheyne-Stokes breathing is present. Dyspnœa is often renal or due to (right-sided) hydrothorax, chronic bronchitis, or excessive dilatation, and appropriate treatment is indicated by catharsis, paracentesis, potassium iodide, or digitalis, respectively.

(*e*) *Cardiac Symptoms.*—Cough, cyanosis, pulmonary œdema, and hæmoptysis are usually true cardiac symptoms, and have for their anatomical basis pulmonary engorgement or brown induration; they are relieved by the cardiants. Balfour recommends the following combination for associated bronchial catarrh:

℞

Ammonii carbonatis	ʒj.
Tr. hyoscyami	ʒiv.
Potassii iodidi	ʒj.
Tr. digitalis	ʒj.
Infusi calumbæ q.s. ad	℥vj.

M. et S.—One teaspoonful every four hours.

Expectorants usually nauseate. Hæmoptysis is often beneficial; it may be regarded as a species of lung venesection and is seldom urgent or fatal. For cyanosis, alcohol and camphor are indicated. Pulmonary œdema necessitates phlebotomy; and a 30-grain dose of lead acetate, gr. $\frac{1}{100}$ of nitroglycerine, and gr. $\frac{1}{4}$ of morphine should be given.

(*f*) *Palpitation and Pain.*—For palpitation and throbbing, the ice-bag may be used; a precordial blister, or tapping several times on the back, or a few minims of tr. belladonnæ and tr. aconiti are often helpful.

℞

Fluidextracti belladonnæ	gtt. xv.
Tr. digitalis	ʒj.
Aq. laurocerasi q.s. ad	℥j.

M. et S.—One teaspoonful two or three times daily.

Care regarding the evening meal (see DIET) and the condition of the colon is important. Ten grains of iodide of potash with nitroglycerine (or gr. ss of erythrol tetranitrate which produces a more lasting effect), are valuable for the pain of aortic lesions, especially when these are accompanied by weakness, pallor, neuralgia, vertigo, or headache.

(*g*) *Gastric Symptoms.*—These are troublesome and ominous. They are largely due to portal stasis, for the liver is enlarged and there is usually not much œdema. They are more often caused by stasis than by a cumulative effect of digitalis, which should then be given by rectum. Small doses of carbolic acid, gr. $\frac{1}{4}$ every half hour for five to ten doses, and rectal nourishment are indicated.

(III). CONGENITAL HEART-DISEASE.

These cases are infrequent and are usually most complicated. They occur largely in the right heart, upon which more work is devolved in fœtal life. The causes are (*a*) *developmental errors* or deficits, and these are by far most frequent; (*b*) *fœtal endocarditis* which is nearly always sclerotic and very rarely verrucose; (*c*) *fœtal myocarditis* or (*d*) *adult endocarditis developing upon developmental anomalies.* They are most often seen in male children, and heredity seems occasionally a factor.

1. **Pulmonary Stenosis.**—This is by far the most frequent congenital lesion and occurs in various forms; (*a*) stenosis or actual atresia of the orifice itself from endocarditis (the orifice closed by a diaphragm or ring) or myocarditis. In this form the second pulmonic tone is weak or absent. (*b*) Changes in the *conus arteriosus* which obstruct the onward flow of blood. The second pulmonic sound is clearly heard, if the valves are normal. The constriction may cause the development of a second ventricle. (*c*) The pulmonary vessel beyond the valves is narrowed or even converted into an impervious cord; this is a grave type of pulmonary stenosis. The second pulmonic sound is usually heard.

The physical signs are hypertrophy and dilatation of the right ventricle, a thrill over the pulmonic area, and a systolic murmur propagated into the neck when there is a defect in the interventricular septum. These cases are more favorable than those in which such defect does not obtain, because in them the right ventricle is atrophic and the subjects rarely live over a year. In pulmonary stenosis other congenital abnormalties are most common. If the closure or stenosis exist before the end of the second month of fœtal life, the interventricular septum fails (in 75 per cent. of cases) to close, and is pushed to the left, so that the aorta may partly or entirely originate in the right ventricle. If the closure develops after the second month, the foramen ovale remains patent (66 per cent.). Upon these openings acute endocarditis is likely to develop. The ductus Botalli usually remains open and thereby conducts blood from the aorta to the pulmonary artery and lungs. Should the ductus Botalli and the pulmonary orifice be closed, collateral circulation is possible through the œsophageal, pericardial, and bronchial arteries. The right ventricle usually hypertrophies, especially at the conus, but hypertrophic changes in this chamber are not constant; the left heart is atrophic.

2. **Defects of the Interauricular Septum.**—The foramen ovale is open to some extent in 44 per cent. of all autopsies, and the membrane is deficient, cribiform, or wholly absent. The foramen ovale usually closes in the first week of life, but fails to adhere when low pressure in the left auricle exists. Entire absence of the septum is the most extreme defect possible; this is called the "*reptilian heart,*" or *cor biloculare* when the interventricular septum is also absent, or the

cor triloculare when the ventricular septum is present. Its patency aids compensation in this condition or when the valvular orifices are narrowed or closed or when the great vessels are transposed. Symptoms are absent or are those of the more important associated trouble, for its patency is almost a necessity in some congenital lesions. Rare occurrences are presystolic murmur at the level of the third or fourth ribs; "crossed or paradoxical embolism," which may occur, for instance, in the brain from venous thrombosis in the leg or even from particles of the liver or of neoplasms (the clots or tissue which pass directly from the right to the left auricle); and positive venous pulse in mitral regurgitation. Several cases have been found in living subjects over seventy years of age.

3. **Defects of the Interventricular Septum** (*Maladie de Roger*).—These have been spoken of under pulmonary stenosis. They may be single, associated with other anomalies, or may result from myocarditis. Sometimes a canal-like opening connects the aorta with the right ventricle. The deficit occurs oftenest in the upper "undefended" part of the septum. When it is wholly absent the heart has but three chambers, the "frilocular heart" (*cor triloculare biatriatum*). Symptoms are absent, complicated, or ambiguous. At times the signs are those of mitral leakage without the lung symptoms of stasis. A loud systolic murmur is heard over the upper third of the precordium but does not reach the large vessels.

4. **Patency of the Ductus Botalli.**—The duct is usually closed by proliferation of its coats within the first month of life, because the pulmonary blood-pressure is lowered after birth. Thus premature closure follows pulmonary stenosis, which necessitates poor filling of the lungs with blood. Permanent patency of the duct is promoted by other congenital lesions or by atelectasis or pneumonia in the newborn. It leads to increased blood tension in the lung. The second pulmonic sound is accentuated, and the artery is frequently dilated, whence the dulness in the second left interspace, with thrill, pulsation, and loud vibratory systolic murmur which may reach the cervical vessels or the fourth dorsal vertebra behind. Sometimes the vessel is so dilated as to compress the recurrent laryngeal nerve and thus cause hoarseness. The right ventricle is hypertrophied and dilated; it is distinguished in clear cases from pulmonary stenosis by the greater cyanosis of the latter lesion, the less frequent propagation to the neck, and the absence of the second pulmonic sound.

5. **Persistent Isthmus Aortæ.**—The fœtal, developmental, thrombotic or inflammatory *narrowing* at a point between the left subclavian artery and the ductus Botalli (46 per cent. below the duct, 37 per cent. at the duct, and 17 per cent. over it), is characterized (*a*) by hypertrophy of the left ventricle (50 per cent.) to overcome the stenosis, and sometimes by a systolic murmur in the interscapular region; (*b*) by largeness and fulness of the aorta above the isthmus, of arteries of the head (brain hemorrhage), neck and arms (full radials); (*c*) by a small, retarded, abdominal and femoral pulse, corresponding

to the anatomical smallness of the vessels below the isthmus; (*d*) by a pronounced collateral circulation between the branches of the aortic arch and those of the thoracic and abdominal aorta, especially the internal mammary, inferior thyroid, and transversalis colli arteries. Other anomalies, such as open foramen ovale, are present in 37 per cent. of cases.

The observant pathologist makes the diagnosis at once; he cuts through the large vessels in the skin of the chest and abdomen, and the clinician when he feels the large vessels (present in 25 per cent. of the cases) and the thrill in them or hears their tones and systolic (or less often, diastolic) murmur, suspects the lesion. Valvular lesions, especially aortic insufficiency, may coëxist. Broken compensation may intervene, though all cardiac signs may be lacking. Aneurysm and mediastinal tumor are frequently confused with it. The aorta sometimes ruptures. The lesion is nearly twice as common in the new-born as it is in adults. Vierordt (1898) collected only 135 cases from the literature.

6. Aortic Atresia or Stenosis.—The lesion is uncommon. The *conus arteriosus* may be greatly constricted. If the lesion is developmental, the interventricular and -auricular septa are open; if endocarditic or myocarditic, they are closed, provided the lesion develops after the third month of fœtal life. The left ventricle hypertrophies in stenosis or atrophies in atresia, while the right ventricle hypertrophies in order to force the blood from the pulmonary artery through the ductus Botalli into the aorta. The lesion may be associated with other cardiac anomalies, with myo- or endocarditis, or with chlorosis, tuberculosis, or hæmophilia.

7. Mitral Changes.—These are very rare.

8. Tricuspid Stenosis or Atresia.—This is developmental or inflammatory. In atresia the interventricular and -auricular septa are open and the right heart is atrophic. Tricuspid insufficiency is most rare.

9. Transposition of the Arteries and Veins.—In this lesion the foramen ovale remains open; the aorta originates in the right heart.

10. Valvular Anomalies.—These are largely of anatomical interest. The semilunar valves, especially the pulmonary, may be increased in number from three to five; or they may be decreased to two, which occurs especially in the aortic valves. Whether developmental or inflammatory in origin, accessory or abnormal valves are particularly prone to acute or chronic endocarditis.

11. Anomalies in Location and Development.—These are absence of the heart, *acardia*, double heart, or ectopia cordis, where the heart lies in the abdomen, or in the pectoral (with fissured sternum) or cervical regions.

Dextrocardia (dexiocardia) occurs with *situs viscerum inversus;* the mitral valve has three cusps, the tricuspid has but two; the pulmonary veins empty into the right auricle; the pulmonary artery arises from the left, and the aorta from the right, ventricle; the aorta runs to the right of the spine, to the right of the œsophagus, and to the left of the cava; the vena azygos is on the left and hemiazygos is on the right; the innominate artery is left-sided; the right lung has two lobes, the left three; the bronchial fremitus is greater on the left side, the left bronchus being larger; the spleen, liver, stomach, and colon, are transposed; the right kidney and testicle are higher than the left. The heart alone is rarely transposed. In one case complete transposition of all the viscera except the cæcum occurred.

General Symptoms of Congenital Heart Disease.—*Cyanosis* is present in 90 per cent. of cases whence the name "morbus cæruleus"; it is more intense than in acquired lesions. It usually appears early, and is either general or limited to the nose, fingers, etc.; the skin may be actually purple, and this is due to mixing of the arterial and venous currents, to a sluggish circulation in the small veins, or to impaired function of the red cells. Some subjects are anæmic, but a most striking increase of the red cells up to eight or nine millions (polycythæmia rubra) is seen in some cases, probably as a compensatory effort. The hæmoglobin may register 130 and the specific gravity 1,077. Dyspnœa, cough, convulsions, and marked clubbing of the fingers and toes, are frequent. The pulse is variable and the temperature often subnormal. Mental and physical development is incomplete, and exophthalmos, swelling of the face, lips and nose, and of the optic disk are frequent. Œdema and stasis are not frequent. There is a tendency to hemorrhage and pulmonary tuberculosis. Coincident maldevelopment occurs (in 10 per cent. of cases) in other organs, such as the stomach, colon, kidney, and spleen; imperforate anus, hypospadias, cryptorchismus, polydactylia, hare-lip, spina bifida, acrania, anencephalus, and uterus bicornis also occur. The murmurs are usually systolic and rough, although very complicated lesions may exist with normal heart tones. In a few instances the fœtal murmur was heard before birth (Peter). The dulness concerns the right ventricle chiefly.

Prognosis.—The prognosis is poor, for the lesion is incurable and compensation is incomplete. The patient usually succumbs to cardiac failure or to pulmonary tuberculosis (25 to 40 per cent. of cases) in the second decade (81 per cent.), hence Rokitansky's rule, that cyanosis protects the lungs from tuberculosis, is not absolute.

Treatment.—The treatment is wholly symptomatic. The body should be kept warm; baths should be employed to guard against colds; mental and bodily strain should be avoided; and in general the therapy of adult valvular disease should be followed, except that the heart stimulants must be used with considerable caution only.

DISEASES OF THE HEART MUSCLE.

DILATATION OF THE HEART.

Definition.—Dilatation means distention of the chambers of t heart; it may be partial or complete. Hypertrophy is thickening of t walls of one or more chambers. The two conditions are quite separa in themselves, though they are frequently combined.

Pathology.—Dilatation occurs (*a*) with thinning of the walls, *sim dilatation*, or (*b*) with eccentric hypertrophy (*q. v.*). It is most comm in the thin-walled auricles and less resistant right ventricle. The mu cle is lax and the heart, when held inverted by the basal vessels, m in extreme instances fall flabbily like a cap over the hand. The dila tion may be so extreme that the endo- and pericardium almost tou

There is much blood in the heart's chambers, in the cavæ and ot veins. Long-standing dilatation is not to be confused with dilatat occurring during the death agony from acute lung disease, suffocati etc., in which the right heart is chiefly affected and in which on remo of the post-mortem clots the chambers seem more nearly of normal s The papillary muscles and the trabeculæ are flattened as in aortic competency (pressure atrophy), and are not infrequently thicke (pressure sclerosis). Microscopically the heart muscle may be norm or may show evidences of fatty or cloudy (or amyloid) degeneration, acute or chronic myocarditis. The precise role of degeneration in t ganglia of the heart has not been determined.

Etiology.—**1. Mechanical Causes.**—The normal heart may yield increased blood-pressure or a weak heart may yield to normal pressu

The mechanical causes are: (*a*) *Valvular disease, e. g.*, dilatation the left ventricle in aortic insufficiency, or of the left auricle and rig ventricle in mitral stenosis. Hypertrophy is usually inversely pro portional to the dilatation. In combined valvular lesions, as aortic an mitral insufficiency, the heart is enormous—the ox heart, *cor bovinu bucardia.* (*b*) *Causes raising arterial blood pressure*, as stenosis of th aorta, arteriosclerosis, and nephritis. These causes produce hype trophy and dilatation of the left ventricle. Workers in caissons ma suffer from dilatation. (*c*) *Causes raising blood tension in the lesse circulation*, as spinal deformity, chronic disease of the lungs and pleur which produce hypertrophy of the right ventricle; of these emphysem is the most important. Abdominal lesions are thought by Frenc writers to reflexly raise the pulmonary blood pressure and induc hypertrophy and dilatation. Lichtheim's classic on the lesser circul tion proved that the lung vascular area must be reduced more than per cent. in order to affect the right heart. The size of the heart varie much physiologically; rapid heart action allows less complete physio logical dilatation than does slow action. When there is pulmonary obstruction with its consequent heart changes, the patient stands pneumonia, bronchitis, and other infections very poorly. When severe exercise is indulged in, the systole is incomplete and right-heart dilatation—even tricuspid regurgitation and marked venous stasis—may follow. Air embolism also produces dilatation. Physical training increases the power and reserve strength of the right heart; *i. e.*, it increases the "wind" and endurance. An equal effort in an individual out of training, or in a man with poor nutrition, "breaks his wind" and produces transient or permanent cardiac exhaustion.

2. **Nutritive Disturbances.**—Dilatation may be induced (*a*) by acute infections, leading to cardiac degeneration or acute myocarditis; (*b*) by anæmia, hemorrhages or loss of fluids; (*c*) by chronic myocarditis, fatty heart, or coronary atheroma, possibly following sudden heart strain as in athletics; by endo- or pericarditis invading the heart muscle by contiguity, pericardial adhesions, etc.; (*d*) by poisons, alcohol, tobacco, chloroform or anæsthesia; (*e*) it very frequently follows hypertrophy or is associated with it.

Symptoms.—The symptoms are those of cardiac insufficiency (*v.* UNCOMPENSATED VALVULAR DISEASE), as dyspnœa—which is especially great in pericardial adhesions—cardiac asthma, cough, syncopal attacks, and precordial anxiety. Cardiac failure begins when the systole is unable to expel all the blood from the dilated chambers.

Physical Signs.—It is not easy to separate the signs of hypertrophy and those of dilatation.

1. **Dilatation of the Left Ventricle.**—(*a*) *On inspection*, the *apex beat* is dislocated downward and to the left and is diffuse, undulating and weak, if indeed it is not absent. (*b*) *On palpation*, the apex may not be felt even though visible (Walshe). In other cases the apex and heart shock are snappy and may feel strong to the inexperienced hand; the sharp, snappy action contrasts sharply with the weak, rapid, and often irregular radial pulse. (*c*) *On auscultation*, the *heart tones* are weak, though sometimes snappy. The first apex tone is more valvular. The second aortic sound is weak because of poor arterial tension. In insufficient hearts, the heart tones sound alike, a condition termed embryocardia from its resemblance to the fœtal heart tones. *An apical systolic murmur* is frequent, especially in arteriosclerotic dilatation. It is sometimes due to relative insufficiency of the mitral valve whose normal circumference of three and one-half inches may be increased by one or two inches. Von Jürgensen holds that it is often due to irregular muscular contraction. Rest and the administration of digitalis frequently cause it to disappear. *Gallop-rhythm*, in which three tones are heard, like the rhythm of a galloping horse, consists of either doubling of the first (Potain) or second tone (Fraentzel), or of the addition of a presystolic tone (Rosenbach) caused by the auricular contraction. It is heard in the failing hypertrophied hearts of nephritics more than in all other conditions combined. It is also found in valvular lesions, atheroma, emphysema, and in acute diseases such as pneumonia, typhoid, and diphtheria. The auricle (causing the extra sound) cannot long compensate. Whatever its cause, it is a sign of great danger

although the heart's tone may temporarily improve with rest and with the use of heart tonics. (*d*) *On percussion,* the heart extends to the lef and downward, giving a more oval area of dulness.

2. Dilatation of the Right Ventricle.—This is more common. (*a* On *inspection,* a diffuse beat is seen to the right of the sternum indeed in the epigastrium; and (*b*) it may be *palpated.* (*c*) *Auscult tion* shows weakness of the heart tones, and (*d*) *percussion* outlin dulness to the right of the sternum. Extreme dilatation increases t tricuspid orifice from four and one-half to five or six and one-h inches, and the physical findings of (relative) tricuspid insufficien obtain.

3. Dilatation of the Auricles.—The deep location of the left aur renders it almost impossible to obtain physical signs of dilatati The changes usually ascribed to the left auricle are due to enlargem of the conus of the right ventricle, though a presystolic wave may seen in the second left interspace. The right auricle is freque distended and this is recognized by undulation to the right of the st num above the right ventricle, by dulness, and sometimes by a presystolic wave.

Dilatation of all the chambers, both right and left, combines t physical findings of each type, thus giving the heart a triangular or trapezoid conformation. The cardiac symptoms and physical findi of dilatation may develop gradually, or abruptly after violent exercis especially when the heart is congenitally weak or when its nutritio altered.

Diagnosis.—The diagnosis depends on the etiological factors, weak tones, the increased dulness, and cardiac and somatic evidenc of cardiac failure. The greater the dilatation, the less marked is t difference between the systole and diastole. The *absolute heart dulne* in middle life is bounded by the left border of the sternum, the four or fifth rib, and the left parasternal line, or it may extend even little more to the left. The *relative dulness* reaches from the third fourth rib above to the apex beat on the left and to a line drawn o liquely from the third left costal cartilage across the sternum to the fif right costal articulation. Percussion is difficult in distorted, fatty œdematous chests. Differentiation concerns the same group of co ditions discussed under the diagnosis of *effusive pericarditis* (*q. v* The Schott method of treatment (*v. i.*) frequently is of value in dia nosis; it helps dilatation but is without effect on pericardial exuda Rest and the use of digitalis greatly benefit dilatation and hydrope cardium, sometimes within a very short time, while pericard effusion is unaffected by this treatment. *Consolidation of the adjacent* lung margin can usually be recognized by the altered breathing and rales. *Mediastinal tumor* and *aneurysm* are suggested by pressure symptoms. If the margins of the *lung are adherent* from pleural adhesions, especial care in percussion is imperative, and dilatation is diagnosticated by the relative, more than by the absolute, heart dulness. Palpatory percussion, very lightly made, is most valuable and the *feel* of the increased dulness has greater diagnostic value than the sound elicited. *Emphysema* may greatly obscure the outlines of the heart, but the heart's outlines are usually disclosed by careful palpatory percussion for the relative dulness. *Encapsulated pleurisy* is differentiated by careful puncture, if other signs fail. The fluoroscope is frequently of aid. In short chests, the apex is seen normally at or even beyond the nipple. This occurs in 12 per cent. of women. Though *spinal curvature* frequently overtaxes the right heart and thus induces dilatation, the diagnosis must be made with care, for the topographical relations are much disturbed by visceral luxation, and the irregularity of the sternum and ribs makes percussion uncertain. In *fat individuals*, dulness may result from adipose deposit in the mediastinum, but differentiation is often easy by means of the normal, though perhaps distant, heart tones and by the character of the pulse. Dilatation must not be mistaken for dislocation due to gaseous, tumor, or fluid distention of the abdomen.

Prognosis.—The prognosis depends on the individual and on the etiology.

Treatment.—The therapy is as follows: (*a*) The cause must be treated. (*b*) The *dietetic, special* and *general indications* of *valvular disease* (*q. v.*) must be carried out. (*c*) *Oertel's treatment* consists of (i) *limitation* of the amount of *water ingested*, and stimulation of its excretion by sweats and deep inspirations. Beer must be interdicted, but many nervous subjects tolerate limitation of water very poorly. Water should be given in small quantities at frequent intervals. (ii) *Reduction of the body-weight* (see OBESITY). (iii) *Stimulation of the circulation by exercise.* Mountain climbing is said to stimulate the heart, develop the respiratory muscles, increase the lung capacity, and widen the vessels. The objections to active exercise are that rest in bed gives better results, and that exercise causes harm when the heart muscle or coronaries are diseased, or when endocarditis, pericardial adhesions, or Bright's disease exist. (*d*) The so-called *Schott method* was originated by Beneke and Saetherburg, and was elaborated by the Nauheim physicians. It consists of (i) *baths* in springs containing sodium and calcium chloride, iron carbonate, and carbon dioxide. The temperature should be a few degrees below body-heat, and a little carbon dioxide should be used in the baths at first. They should be given once daily for two or three days, and then intermitted; they should last from five to twenty minutes. They may be given artificially with almost equally good results, using 40 gallons of water, with 3 to 10 pints of salt, 10 ounces of pure calcium chloride and carbon dioxide generated in a vessel from sodium bicarbonate and hydrochloric acid. The temperature should be later reduced to 90° or less, and the amount of carbon dioxide should be increased. The bloodvessels are first contracted and later dilated. The pulse becomes slower and fuller, dilatation decreases, stasis is lessened, diuresis is increased, and the metabolism of the muscles and heart is improved. (ii) *Resisted movements* should be carefully and gradually employed. Though active exercise constricts the vessels, gentle movements by the patient, resisted by the operator, dilate the vessels.

Yeo (*Manual of Medical Treatment*) summarizes the resistance movements as follows:

Each exercise is made against slight resistance applied by the physician or a trained assistant.

1. The arms are extended in front of the body at the level of the shoulder, with the palms of the hands touching. The two arms are then moved slowly outward till they are in a line with each other; they are then brought back to their original position.

2. The arm and hand hanging down with the palm turned forward, the forearm is flexed upon the arm (which is kept still) until the fingers touch the shoulder. The forearm is then extended to its original position. This is first done with one arm and then with the other.

3. The arms, hanging down as in No. 2, are raised outward until the thumbs meet over the head; they are then returned to their original position.

4. With arms dependent, the fingers, at the first phalangeal joints, are pressed together, and the arms are then raised until the hands are above the head, afte which they are brought back to their original position.

5. The arms, hanging in the position of "attention," are advanced forward paralle to each other until they are elevated to a vertical position; they are then brough back to where they were before.

6. Same as No. 1, but with fists clenched.

7. Same as No. 2, but with fists firmly clenched.

8. The arms, starting from the position of "attention," describe a circle by movin forward and upward until they are raised vertically; then each palm is turned ou ward, and the arms descend backward to their former position.

9. The body is bent forward, and then brought back to the erect position, th knees not being moved.

10. The body is rotated, without any movement of the feet, first to the right a then to the left, and then back to its original position.

11. The body is flexed laterally, as far as possible, first to the one side and then the other, and afterward restored to its original erect position.

12. The patient, standing with the feet side by side and supporting himself leaning with one hand upon any object, flexes the opposite thigh as far as it is possib and afterward extends it until the feet are again side by side; then, leaning on t other hand, he carries out a similar movement with the other thigh.

13. The patient, supporting himself by one hand, as in 12, and the knee being k straight, each leg in turn is raised as high as possible in front of the body, and then the same way behind.

14. Supporting himself by placing both hands in front on the back of a chair, t patient first flexes one leg and then the other upon the thigh as far as he can.

15. Each leg in turn is abducted as far as possible, the knees being kept straig the patient resting on one or other hand the while.

16. The arms, held horizontally outward, are rotated forward and backward the shoulder joint.

17 and 18. Flexion and extension, first of the wrists, and second of the ankles.

In resisting these movements the operator places the palm of his hand on that s of the patient's limb or body toward which the movement is to be made. In movements of the wrist the operator closes his thumb and forefinger round that jo

(iii) *Passive movements and massage.* Contraindications to Scho method are aneurysm, angina pectoris, acute cardiac insufficien tendency to hemorrhage and embolism, and possibly nervousness.

HYPERTROPHY OF THE HEART.

Pathology.—The muscle fibers increase even tenfold in size (*hypertrophy*) and increase in number (*hyperplasia*). Hypertrophy may exist alone—*simple hypertrophy.* It more frequently occurs with dilatation

—*eccentric hypertrophy*. *Concentric hypertrophy*, *i. e.*, with decrease in the size of the ventricle, is rarer and some regard it as merely a post-mortem condition; the ventricle may be firmly contracted in death from violence or from cholera, or hemorrhage, but it can be distended by the finger. The size of the normal heart is that of the fist (Laennec), and the normal weight is about 9½ ounces (300 gm.). The heart is sometimes increased four times in weight, even to 59 ounces (1,980 gm.) (Stokes)—the *cor bovinum.* Its consistence is increased, and its color is normal or darker. In total hypertrophy the heart is round; in partial, the right or left ventricle is larger. The left ventricle normally measures nine to ten millimeters, but this may be more than doubled in hypertrophy; the right ventricle measures three or four millimeters; the left auricle measures three, and the right two, millimeters. In the fœtus the ventricles are equally thick. Up to the eighth year, the left ventricle is relatively thicker than in the adult, because of narrowing of the aorta at its isthmus.

Etiology.—The causes of *hypertrophy* of the *left ventricle* are usually mechanical resulting from (*a*) *valvular lesions* (aortic stenosis and insufficiency, mitral insufficiency); (*b*) *renal disease;* usually from a chronic interstitial nephritis but occasionally from other forms, from hypoplasia of one kidney, from hydronephrosis, etc. It is caused by mechanical obstruction to the flow of blood or by retention within the blood of waste substances which excite arterial spasm; (*c*) *arterio-sclerosis* (*q. v.*), developed by Traube; (*d*) *congenital narrowness* (*hypoplasia*) *of the arterial system*, or of the *aorta at the isthmus*; (*e*) *dilatation of the aorta*, diffuse or aneurysmatic, or occurring rarely from general arterial dilatation (Nothnagel and Fraentzel); (*f*) *pericardial adhesions;* (*g*) *myocarditis* (*q. v.*); (*h*) *pregnancy;* it is claimed that the placental circulation or increased intraäbdominal pressure increases the heart's work. The high diaphragm apposes the heart to the chest wall and tips the apex outward so as to simulate hypertrophy; (*i*) *over-exertion;* the "irritable heart of soldiers" (Da Costa, Myers, Fraentzel) is caused by forced marches, cold, and mental excitement; the so-called *idiopathic* hypertrophy occurs in hard workers, and especially in Munich from excessive use of beer and from the resulting plethora; hypertrophy results from continued hard work while dilatation follows sudden cardiac strain; (*j*) *nervous causes;* these are exophthalmic goitre, and the use of tobacco, especially strong Havana cigars, which causes rapid irregular heart action. Pujatin, Ott and others have found degenerative and sclerotic changes in the cardiac ganglia.

Hypertrophy of the right ventricle depends on (*a*) *hypertrophy consecutive to left heart disease;* the entire heart usually hypertrophies from the intimate connection between the muscular fibres of both sides; (*b*) *right-heart valvular lesions* and *congenital heart disease;* (*c*) *increased pulmonary pressure* from disease of the pulmonary artery, or pressure upon it; (*d*) *emphysema, chronic bronchitis, spinal deformity,* pulmonary induration, bronchiectasis, and extensive *pleural adhesions.*

Hypertrophy of the auricles always coëxists with dilatation (stenosis of the auriculoventricular orifices).

Symptoms.—Hypertrophy of the left heart may exist without any subjective sensations, or there may be palpitation, inability to lie on the left side, cough, cardiac oppression or pain or cerebral congestion (tinnitus, flashes before the eyes, headache, or vertigo). There is frequently a tendency to hemorrhage (epistaxis, cerebral hemorrhage, excessive menstruation). Polyuria results from high arterial tension. The causal disease (nephritis, valvular disease, arteriosclerosis, emphysema) presents its own symptoms. Right-heart hypertrophy is attended by brown induration of the lungs, hæmoptysis, cyanosis, or bronchial catarrh.

Physical Signs.—Much confusion in the clinical description prevails because the signs of hypertrophy are very often confounded with those of dilatation, although both conditions very frequently coëxist.

Hypertrophy of the Left Ventricle.—1. *Inspection.*—This discloses (*a*) a *strong*, sometimes heaving, diffuse *apex*; essential dislocation to the left or downward indicates coincident dilatation, because thickening of the heart muscle alone scarcely gives any increase in the size of the heart. Rotatory retraction of the apex beat is sometimes seen. (*b*) *Precordial prominence;* (*c*) *forcible pulsation* of the *vessels*.

2. *Palpation.*—Palpation confirms (*a*) the strong *apex beat*, detects (*b*) the *palpable second aortic tone* due to high tension (or atheroma), and sometimes (*c*) a *systolic apical thrill* (transmission of the strong first tone). (*d*) The *pulse* is regular, full and hard in simple hypertrophy, but in the eccentric form it is softer and more rapid. It is sometimes weak or almost absent in the arms; this is due to loss of vessel tonus. (*e*) *Thrills over the peripheral vessels* from high tension vibration may be felt.

3. *Percussion.*—It is usually stated that the left heart is dislocated downward to the left but this is infrequent, as enlargement means dilatation (or an atheromatous relaxed aorta which allows sinking of the heart).

4. *Auscultation.*—This brings out (*a*) usually the *loud first tone*, over the apex (but not always, as for example in aortic leakage); it discloses (*b*) the *accentuated* and frequently metallic *second aortic tone*; (*c*) *systolic arterial bruits* from tension and vibration of the walls; (*d*) the *gallop-rhythm* in cases of failing hypertrophy, especially in nephritics; (*e*) a *peculiar clink*, which is sometimes heard to the right of the apex; (*f*) an *apical systolic murmur* is sometimes heard, and is frequently of the cardiopulmonary type. Seitz, Gendrin and others have recorded murmurs resembling the pericardial rub, due to the violent beating of the heart or to muscular contraction (Jürgensen).

Hypertrophy of the Right Ventricle.—(1) *Inspection* shows a *strong, systolic impulse under the sternum* which is (2) *palpable*, as is the *strong second pulmonic tone.* (3) *Auscultation* brings out the accentuated second pulmonic; and (4) *percussion* is negative, unless there is coincident dilatation.

Diagnosis.—In *left-sided hypertrophy*, the cardinal signs are (*a*) the tense pulse, (*b*) the strong, hard apex beat, and (*c*) the accentuated second aortic tone; in *right-sided hypertrophy* they are (1) the strong second pulmonic tone, and (2) the substernal pulsation. Percussion is useless, for it discloses dilatation only.

Hypertrophy of the left heart must not be confused with nervous overaction of the heart in which the apex is not heaving. Retraction of the left lung merely exposes more of the left ventricle, but the heart in this case seems enlarged upward and to the left. In narrow chests with poor lungs, the heart's dulness is wide, but the cardinal symptoms of hypertrophy are lacking. The same caution in the diagnosis of hypertrophy at puberty is necessary because at this period the heart is nervously unstable, the chest is thin, and the heart is normally wider than in the adult. Perplexity may be caused when abundant casts and blood are found in the urine; these are usually indicative of *nephritis*, but, if there are other evidences of stasis, such as congested liver, they are compatible, in the minds of some writers, with stasis alone. Nephritis may cause hypertrophy; nephritis and hypertrophy may be due to a common cause; or a failing heart may produce renal stasis (see NEPHRITIS, Differential Table).

Prognosis.—The immediate outlook is good because hypertrophy is usually a compensatory process; the ultimate outlook is bad, because cardiac insufficiency is the ultimate issue. Symptoms usually appear first either gradually or suddenly in the stage of broken compensation from intercurrent fevers, malnutrition, or mental and physical strain. The best outlook is in hypertrophy caused by tobacco or athletics, for this may regress. Gallop-rhythm is ominous as is the occurrence of the first and second apical tones close together. Hypertrophy leads to fatty and other change in the heart, to sclerosis of the vessels, and in the pulmonary circuit to pulmonary atheroma, acute congestion, and œdema.

Treatment.—The therapy is eminently causal and dietetic; alcohol, tea and coffee, tobacco, and cold baths, are to be avoided, and constipation must be prevented. The symptomatic treatment and the use of cardiants in the uncompensated stage are identical with the indications in valvular disease.

(C). ATROPHY OF THE HEART.

Atrophy of the heart presents more pathological than clinical interest.

Etiology.—The heart is at times congenitally weak and small. It may then be associated with small vessels, chlorosis (Virchow), or with hypoplasia of the genitalia (Rokitansky). Pressure atrophy, coronary sclerosis, pericarditic adhesions, the senile involution, and cachectic diseases, are the most frequent causes. *Anatomically*, the heart is reduced in size and weight (even to 1½ ounces) the subpericardial fat disappears, the pericardium wrinkles, the coronary arteries are tortuous, and the heart muscle is pigmented. The valves, especially the aortic, may waste. The atrophy may be partial, as that of the left ventricle in some cases of mitral stenosis. Simple, concentric, and eccentric atrophy are described.

The *symptoms* are indeterminate, and the *diagnosis* impossible.

FATTY HEART.

Etiology and Pathology.—Fatty heart is no distinct pathological nor clinical entity. Two forms are recognized, (1) fatty infiltration, and (2) fatty degeneration.

1. Fatty Infiltration (Lipoma cordis capsulare, Virchow).—This form consists of invasion of the heart muscle by the subpericardial fat, which is normally found about the heart, in its furrows, at the tips of the ventricles, and around the basal vessels. The fat extends in parallel lines between the muscle fibers, which it may wholly replace. The fatty infiltration was considered primary by von Leyden; Struempell believes muscle atrophy is primary and the fatty infiltration secondary; Rosenbach holds that fatty infiltration and muscular atrophy are secondary to a common nutritive disturbance. The fat, which is usually most developed in the outer layers of the myocardium, may reach to the endocardium. Fatty infiltration is most common in obese males over forty years of age, but has been seen in young subjects. It may occur after the menopause or in sterile women. Plethora, high living, excessive use of alcohol, and heredity, are factors. The heart is reduced 25 per cent. in weight compared with the body-weight (Hirsch).

2. Fatty Degeneration.—This form is caused by deficient oxygenation which is either (*a*) *general*, resulting from acute infections, old age, cachexia, marasmus, anæmia, from such poisons as phosphorus, chloroform, or mercury, and from loss of fluids from diarrhœa; or (*b*) *local or cardiac*, from pericarditis; from coronary disease, which more frequently produces myofibrosis; and from cardiac failure following hypertrophy. Most degeneration occurs in the left heart. The fatty heart is large and flabby; it is yellow-brown—the "faded leaf" color of Laennec; the splashes and streaks of fatty degeneration contrast with the normal areas, giving the variegated "tiger" or "tabby-cat" appearance; oil globules are found in rows in the muscle fibers and may replace them in part or in whole. Frequently the muscle fibers appear dry and granular ("albuminoid" degeneration) and this is probably the first step in fatty degeneration. The striations of the muscle disappear later. Its consistence is decreased and its musculature is friable.

The extent of the process is in direct ratio to the intensity of nutritive alteration; parenchymatous and fatty degeneration are observed in those segments of the heart where the greatest work and the most nutrition are demanded, as in the papillary muscles of the mitral valves, and where pressure is greatest, *viz.*, the interventricular septum and the left conus arteriosus. In embryonal life, these conditions obtain in the right heart. The early investigations of von Leyden and those of more recent date by Goebel have determined that these changes are first focal and later diffuse.

Symptoms and Signs.—No symptoms are characteristic. Extensive fatty changes may occur without clinical symptoms (*latency*), as in pernicious anæmia. Leube details a case without symptoms in which the apex was completely fatty. In some obese subjects, cardiac embarrassment develops with dyspnœa, palpitation, a somewhat characteristic wheezing, etc. The autopsy may reveal fatty heart, but far more frequently myocarditis, idiopathic hypertrophy, or coronary disease is found; *i. e.*, there is no uniformity in the pathological findings, nor parallelism between them and the clinical signs. Though percussion of obese individuals is unsatisfactory, the heart is found dilated. In fatty heart, the tones are weak and distant, though pure, the apex beat is weak, diffuse, or absent, and sometimes an apical systolic murmur is heard, which is probably due to irregular systolic vibration. Gallop-rhythm is heard when the heart is fatigued. The pulse may be rapid from brain anæmia; it may be somewhat dicrotic, small, and arrhythmic. Low temperature, sweating, and bronchitis are common.

Much stress was formerly laid on the *Adams-Stokes syndrome*, a triad of symptoms including (*a*) a slow pulse, even 14 to the minute, due to anæmic stimulation of the vasomotor centre, or to heart changes, and followed by (*b*) *pseudo-apoplectic seizures*, unconsciousness, temporary paralysis, or convulsions (pseudo-epilepsy), which are clearly dependent on cerebral anæmia, for one of Stokes's cases warded off these attacks by assuming the genupectoral position; and (*c*) *Cheyne-Stokes' respiration*, in which respiration becomes slower, and is followed by a period of apnœa lasting a half minute or more, with respiration gradually returning to the normal. Narcotics increase or induce the condition. The pupils narrow during the apnœic period and muscular twitching is common. This triad is observed in uræmia, cholæmia, and diseases of the vagus and medulla as well as in heart disease. It seems likely that in Stokes' case the brain was also diseased. The syndrome is based on the bradycardia which appears to be most frequent in disease of the bundle of His (see BRADYCARDIA, CARDIAC NEUROSES).

Diagnosis.—The diagnosis is at best uncertain. As stated, fatty heart may cause no clinical symptoms, or the symptoms observed in the obese may be due to other lesions. Apparently increased dulness is often due to mediastinal obesity. The main point is to exclude valvular lesion and confine the diagnosis to a myocardial affection (*v.* MYOCARDITIS).

Prognosis.—The prognosis depends upon the cause. It is grave in diabetic or arteriosclerotic subjects, or when coronary disease, angina, lung œdema, or an irregular or very slow pulse intervene.

Sudden death is not uncommon from acute dilatation, pulmonary embolism, brain anæmia or hemorrhage, "heart block" (described under bradycardia), or rarely from heart rupture.

Treatment.—The treatment is that of obesity in fatty infiltration, but distinction must be made between (*a*) *obesity with anæmia*, in which the subject is obese and pale, with cold extremities, much sweating, scanty urine, weak and easily fatigued muscles, and dyspnœa; and (*b*) *obesity with plethora*, in which the color is red, the urine increased and uratic, the heart stronger, the muscles fairly developed, and exercise possible with little dyspnœa. Schott's treatment is beneficial in the first type, and Oertel's method in the latter (see DILATATION OF HEART). Saline cathartics, total abstinence from alcohol, and the use of potassium iodide for general or coronary sclerosis are indicated. The brain symptoms of anæmia and the sudden syncope are met by the ætherial alcoholics. Cardiac insufficiency is treated as in uncompensated valvular lesions and dilatation. Morphine should be avoided entirely or used only with great circumspection.

ACUTE MYOCARDITIS.

Etiology and Pathology.—Acute myocarditis is of the greatest clinical and prognostic importance.

Acute diffuse parenchymatous myocarditis corresponds to granular and fatty degeneration (*v. i.*); to the "mushroom" heart of Stokes (described under TYPHUS) which, when held inverted by the base, would fall like a cap over the hand; and also corresponds to the "softened heart" of Laennec and Louis.

Acute diffuse interstitial myocarditis has been most studied of late years, while formerly the degenerations attracted most attention. Round cells are found in the interstitial tissue, with or without muscular alteration. Vacuolization, nuclear multiplication, pigmentary deposit, blood extravasations, waxy and other changes are also found. Though it was first discovered by Leyden in scarlatina, it is found in a very large percentage of cases of diphtheria and typhoid by Rhomberg; it also occurs in acute endocarditis and rheumatism (10 to 20 per cent.), less frequently in variola, influenza, gonorrhœa, quinsy, and other infections. Though the typhoid bacillus, the gonococcus, and Bacillus pyocyaneus have been found in the myocardium, they are chiefly important as causing toxæmia. In some instances acute myocarditis is seemingly primary.

H. Martin has found inflammatory thickening of the intima in the coronary radicles, also minute thrombi, lamellar splitting of the elastic tunic, and proliferation of the media. Rhomberg found a perineuritis in one-half of typhoid and diphtheria autopsies. Virchow, Hayem, and Renault, hold that inflammation is primary and degeneration secondary; Weber, Blind, and others regard the process as degeneration rather than inflammation.

Symptoms and Signs.—During the progress of an acute infection, the heart's strength decreases; its tones, especially the first mitral, become fainter; the apex is less distinct and is dislocated somewhat to the left by dilatation; and the pulse grows weaker and more irregular. Vasomotor relaxation is probably a very important element in the heart failure. Gallop-rhythm and embryocardia develop, and there is usually a relative (muscular) mitral leakage. Cardiac pain may be severe. These changes may develop during the infection as is the case in typhoid, scarlatina, and rheumatism; or they may occur several (two to ten) weeks after the infection, as in 10 to 20 per cent. of the cases of diphtheria. Sudden death without symptoms may occur during convalescence. Rheumatic myocarditis often leads to chronic fibrous myocarditis.

Diagnosis.—The diagnosis is made from the etiology; acute myocarditis is distinguished with great difficulty from acute endocarditis (*q. v.*).

Treatment.—1. **Prophylaxis.**—Hydrotherapy is indicated in typhoid and antitoxin in diphtheria; the patient should have absolute rest well into convalescence, because sudden heart insufficiency, or unexpected death may follow effort, difficult defecation, or other exertions; sexual intercourse should be forbidden. The heart must be carefully watched in every convalescence.

2. **Cardiants.**—These are indicated as in valvular incompetency, although digitalis and strychnine are apparently of little use.

Acute Circumscript Myocarditis.

Acute circumscript myocarditis (the disease of gladiators, Galen) is usually septic and embolic. It is one form of sepsis, and its heart foci are usually multiple. It is usually caused by the pyogenic organisms. The issues are (*a*) resorption with fibrosis, (*b*) caseation or calcification, and (*c*) rupture into the pericardium or into the heart cavity, thus producing "heart ulcers" and pulmonary or arterial embolism, heart aneurysm, abnormal communications between the chambers, or sudden valvular insufficiency. The latter is sometimes suggestive, though it is usually impossible to make a diagnosis. The treatment is that of the fundamental septicopyæmia and the resulting cardiac insufficiency.

Fragmentation of the Heart Muscle.

Fragmentation of the heart muscle (Zenker, Virchow, Rindfleisch, Coats, Renault, and Landouzy) occurs in two forms (*a*) segmentation, or separation from each other of the heart fibers, and (*b*) fragmentation, or rupture across the normal or degenerated muscle fibers. It has also been called segmentary myocarditis, and though it most equently occurs in death from violence, it may have some clinical ignificance in cases of sudden or gradual heart weakness (see Myolysis under Diphtheria).

CHRONIC MYOCARDITIS (CHRONIC FIBROUS OR INTERSTITIAL MYOCARDITIS, MYOFIBROSIS CORDIS).

Etiology and Pathology.—Chronic myocarditis is rarely a disease *sui generis*, and the term is somewhat ill-chosen. It is a sequel of some previous retrogressive lesion—a secondary process. It compensates for destruction of heart tissue, and is in itself a measure of that loss. It is more often a fibroid degeneration than an inflammation. The longitudinal incision of the heart muscle discloses the scar-like tissue which is often unseen in the ordinary transverse sections (Koester and Rühle). This tissue is seen most frequently in the left ventricle (lower two-thirds of its anterior wall near the apex and the upper two-thirds of its posterior wall near the auricle) as glistening sunken areas under the pericardium or endocardium. It also occurs in the papillary muscles or interventricular septum. The scars appear as streaks, flecks, stellate plaques, sometimes measuring three or four centimeters, or even larger. The foci may be macro- or microscopic, circumscribed or general. Large areas may form from fusion of small foci. *Gross changes in the heart* include the following:

1. **Hypertrophy.**—This is vicarious and occurs when the fibrosis is not great. The relation is twofold; (*a*) hypertrophy often compensates for muscular destruction and maintains the heart; or (*b*) fibrosis develops in tired hypertrophied hearts. Hypertrophy is inversely proportional to the number of scars.

2. **Dilatation.**—This follows extensive fibrosis and decreased power to resist pressure. Fibrosis of the tissue can neutralize dilatation to some extent, and is therefore a conservative agent.

3. **Atrophy of the Myocardium.**

4. **Valvular Disease.**—This has no necessary affiliation with myocarditis, though they are indirectly associated. A relative mitral insufficiency may follow myocarditis in the mitral papillary muscles (Hamernjk). Dittrich first described the genuine heart-stenosis, a cicatricial contraction of the left conus arteriosus in the adult, or of the right conus in the fœtus, following myocarditis at those sites. Relative aortic insufficiency may follow enormous dilatation and myocarditis of the left ventricle (personal observation).

5. **Partial Heart Aneurysm and Heart Rupture.**—Partial *heart aneurysm*, a localized bulging of the heart wall, is seen largely near the apex (left ventricle, 68 per cent. of cases, Pelvet), where acute myomalacia cordis or chronic fibrosis occurs most frequently; it is seen more rarely at the base, or in the interventricular septum which may bulge into the right heart; and it occurs most infrequently in the right ventricle or auricle. The heart aneurysm is usually small, but

may equal the size of the heart; it is usually single (though four were found by Thurnam), and may communicate with the heart cavity by a neck-like constriction; its walls consist of connective tissue which is rarely ossified as in Corvisart's case; it often contains thrombi and is adherent to the pericardium. In unfavorable cases it ruptures or tears loose a valvular attachment. During twenty years ending in 1903, D. G. Hall found 112 cases recorded (26 had aneurysm in the valves, and 25 in the coronary vessels); Voelker alone made a diagnosis. *Rupture* of the heart occurs through the left ventricle (80 per cent.).

Pathogenesis.—The *most frequent cause is disease of the coronary arteries*, which are incessantly active and are end-arteries, anastomosing with each other only through their capillaries. The vessels of Thebesius, entering the muscle from the auricles and ventricles as well as some supernumerary vessels, may carry on a collateral circulation when the coronary arteries are occluded. *Coronary narrowing* may be *gradual*, by slow obliterative endarteritis in the trunk or by the occlusion of the vessel from an atheromatous plaque in the aorta; or it may be *sudden* and fatal by embolism or thrombosis in an already narrowed vessel. The most common location for coronary obstruction is in the left ventricle (anterior coronary artery which is most active, and relatively small) and in the interventricular septum. In fairly acute coronary obstruction, the heart muscle becomes ischæmic, pale yellow or white, and opaque; and the necrotic area becomes swollen and prominent (coagulation necrosis). A wedge-shaped anæmic (or hemorrhagic) *infarct* is less common than an irregularly outlined necrosis. The muscle fibers become fragmented, lose their nuclei, and disappear; this process may lead to rupture of the heart. Ziegler named it *myomalacia cordis*. White cells surround the focus. Sudden death is very common. If the subject lives, scar-tissue develops to replace the deficit, and is known as *dystrophic sclerosis*. Such "myocarditis" is not inflammatory; it is often an uncomplicated coagulation necrosis with substitution by scar-tissue. Huchard employs the terms, arteriosclerosis of heart and arterial cardiopathy, to emphasize the causal role of arterial disease. *Coronary arteritis* most frequently runs a subacute or chronic course. Its causes are those of arteriosclerosis (*q. v.*). A less frequent cause is *actual chronic myocarditis* following *acute myocarditis* due to acute infections, such as rheumatism, typhoid, or diphtheria. Koester, Ruehle, and others have found myocarditis in cases of cardiac insufficiency, resulting from valvular disease, and also in idiopathic cardiac hypertrophy. French writers describe an acute *endarteritis* following acute infections and leading to myofibrosis. Dehio holds that *myocarditis, or myofibrosis, is a result of dilatation*, that it occurs where the muscle is destroyed, and is therefore a necessary and protective process. This view conforms with Thoma's conception of arteriosclerosis (*q. v.*). Dehio considered that myofibrosis has no affiliation with acute myocarditis, that it occurs in all cavities of the heart, and often in the weaker and more dilated auricles, that dilatation is the determining factor, and that it is always a process secondary to the degeneration of hyperplastic muscle, to pressure, and to venous stasis.

Symptoms.—(*a*) In the *latent type* there are no symptoms, and sudden apoplectiform death occurs, while the subject is apparently in perfect health. (*b*) Most cases present chronic symptoms of *cardiac insufficiency*. The cervical veins are enlarged and there is general venous congestion. The pulmonary circulation suffers especially, as is shown by cyanosis, by duskiness of the skin, by infarcts which are often large (Rühle), by hydrothorax, œdema of the lungs, bronchitis, and by dyspnœa which is often nocturnal and develops on exertion. The heart symptoms are precordial oppression or dull pain, palpitation, cardiac asthma, or genuine angina pectoris. Examination of the heart sometimes gives negative results, but dilatation and hypertrophy are usually present (cardiomegalia). The heart tones at first are often clear, but later become faint and irregular, so that in the resulting delirium cordis, the tones are disorderly, and the phase of the murmur, due to relative muscular insufficiency or to arteriosclerotic dilatation, cannot be distinguished. The second pulmonic sound is accentuated or split, and the second aortic tone is weak, because the arterial tension is low. The pulse is usually irregular (Rühle, Riegel, Bard, Phillippe), and may be so either from the beginning or may become so later, with paroxysms of arrhythmia which some consider an extension of the degeneration and sclerosis. Irregularity usually precedes the cardiac insufficiency and persists during restoration of compensation. A regular pulse may be present throughout (Ebstein and Renoy), or a number of regular strong beats is followed by a wave of rapid tumbling heart action. Simple intermission and the pulsus bigeminus are also seen. The pulse rate is often slow at first, and gradually increases to 90 or 100, with ominous crises of tachycardia. In some instances the pulse is persistently slow and this is associated with syncope from brain anæmia (*v. i.* BRADYCARDIA). It is seen, then, that the rate and rhythm of the pulse vary, and though irregularity is of important diagnostic value there is no constancy in the pulse findings.

Œdema is present, but as a rule is less common and intense than it is in valvular disease. Embolism may occur. Digestive disturbances are almost constant (Rühle), and Huchard maintains that they have an important bearing on the heart's action; alimentary autotoxæmia from carelessness in regard to diet, may cause paroxysmal cardiac symptoms. Potain and Barie hold that digestive disorders induce a reflex contraction of the lung vessels and thus lead to dyspnœa. The urine is that of stasis in the later stages, but early in the disease polyuria may prevail from hypertrophy and high blood pressure. Renal insufficiency may lead to renal toxæmia (Houchard).

Diagnosis.—There Are Five Cardinal Points.—(*a*) In the majority of cases *arteriosclerosis* is most important. Its causes should be sought in the anamnesis, and the peripheral arteries and aorta should be examined for its usual signs (*q. v.*). Arteriosclerosis is usually general, but may exist in the heart or brain without being seen in the radial arteries, and the converse is also true. (*b*) The heart is usually dilated and hypertrophied; *i. e.*, its transverse measurement is increased. (*c*) *No valvular murmur exists* (in uncomplicated cases). The systolic apical murmur of arteriosclerotic dilatation may be heard or the soft variable bruit of relative mitral leakage, which often disappears with rest and catharsis. (*d*) *The pulse is fast and irregular.* A regular pulse does not absolutely exclude myocarditis. Arrhythmia, whether congenital, neurasthenic, or caused by tobacco, must be excluded. (*e*) *Dyspnœa* and pulmonary stasis are suggestive.

Differentiation is often so difficult that many able clinicians limit the diagnosis to *muscular insufficiency*; for fatty heart, idiopathic hypertrophy and dilatation, or pericardial adhesions, may be found at autopsy instead of the anticipated myocarditis. Sometimes when myocarditis is suspected, the heart shows nothing microscopically or macroscopically, thus demonstrating that the causes of heart failure are often beyond the detection of the microscope. A diagnosis is made by exclusion, and then is often only a "probability diagnosis." In distinguishing myocarditis from the "heart of *renal disease*," long observation, the gallop-rhythm (more common in the *cor renale*), and uræmic manifestations are helpful. Myocarditis and renal disease are rather frequently associated. From *mitral stenosis:* both lesions cause irregularity and a weak, radial pulse and apex beat; when the presystolic stenotic murmur is absent, the loud second pulmonic sound, the loud, snapping, apical tone and the wide, right heart are most suggestive of mitral stenosis. Digitalis very frequently fails to act in myocarditis. From *cerebral apoplexy:* the sudden "stroke" or "cardiac apoplexy" of coronary disease may be the first sign of myocardial disease and may be mistaken for hemorrhage of the pancreas, cerebral apoplexy, or embolic processes. The autopsy, in cases of sudden death or death after a few hours, often discloses coronary closure with myomalacia cordis or fibrous myocarditis.

Prognosis.—The prognosis is that of uncompensated valvular lesions with the extra consideration that the muscle itself and most often its nutrient arteries are involved. Because of the facts that digitalis is often inefficient in these cases, and that angina pectoris, cerebral anæmia, heart rupture, or coronary closure may occur, the outlook is most unpromising. Some patients, especially elderly subjects, may live a decade or more, until intercurrent pneumonia or another secondary affection causes their death. Early digestive symptoms are less favorable than early œdema or congested liver.

Treatment.—The therapy is (*a*) that of the *causal factor*, arteriosclerosis (*q. v.*) being the most common cause. Potassium iodide relieves pain, and lessens the atheroma; it should be given in doses of 5 to 10 grains after meals, freely diluted in milk or water—

℞

Potassii iodidi	ʒiiss.
Syr. sarsaparillæ co.	℥iv.

M. et S.—One teaspoonful in a glass of water after meals.

(*b*) *Dietetic;* stimulants should be used carefully, and overexertio and excitement should be avoided. (*c*) *The Schott treatment* (*v. s.* (*d*) *Cardiants.* Digitalis is beneficial in the less developed, but n in the advanced cases. It is to be avoided when the pulse is slo Strychnine is frequently valuable. When there are anginal seizur or hard vessels, nitroglycerine should be given (see THERAPY OF VA VULAR DISEASE). Nitrite of amyl is usually ineffectual. Caffein camphor, morphine, and alcoholics should be administered for cardia asthma; morphine is considered somewhat dangerous in angina pe toris (*q. v.*).

RUPTURE OF THE HEART.

Etiology.—(*a*) Spontaneous rupture is practically unknown. (*Rupture through the altered heart muscle* occurs in myocarditis, hea aneurysm (in which case it may even perforate the chest wall), myom lacia cordis (from coronary atheroma and embolic occlusion), corona aneurysm; fatty heart, abscess, gumma or neoplasm of the hear ulcerative endocarditis or obstructive valvular disease, and stenos of the isthmus of the aorta. It occurs largely in men over sixty yea of age. It may occur during sleep or may be caused by trauma, vomi ing, great effort, excitement, delivery, coitus, or by the use of digitali Kroll collected 332 cases of rupture of the heart.

Pathology and Symptoms.—(*a*) *Partial rupture* of the valves i duces sudden valvular insufficiency. (*b*) *Total rupture* of the heart usually single (though a case having five rents is recorded), and tak place in the left ventricle (80 per cent.) where the pathological facto (*v. s.*) come most into play; it occurs less frequently in the interve tricular septum, right ventricle and auricles. *Sudden death* may occ without prodromes, or death may result after a few hours to elev or seventeen days. The symptoms are a sense of something givi way in the chest, great precordial pain, incoercible vomiting or di rhœa, cerebral anæmia or irritation of the vagus from hæmoperic dium, collapse, and death from pressure of the blood on the ve cavæ (Cohnheim) or on the heart. Hæmopericardium (*q. v.*) may be s pected because of greatly increased triangular dulness, faint heart ton and absent or weak apex beat within the left border of the dulness.

Diagnosis and Treatment.—The diagnosis is rarely made. depends on (*a*) the *etiology*, (*b*) *increased dulness*, (*c*) *cardiac ins ciency*, and (*d*) *symptoms* of *internal hemorrhage*. The *outlook* is b and the *treatment* wholly symptomatic; the most absolute rest sho be enforced.

Heart injury was first operated on by Farina (1896). The cardiorrhaphy for rupture was by Rehn (1897). Frazier (19 collated 102 operations for injury, of which 42 per cent. recovered.

Disease of the Coronary Arteries.—This has been sufficiently consi ered under myocarditis (heart aneurysm) and rupture of the hea It will be considered again under angina pectoris, which anatomicall belongs to disease of the heart muscle though it is usually described under cardiac neuroses.

TUMORS OF THE HEART.

These are most rare because the heart is rather insusceptible even to neoplasms in its immediate vicinity. They cannot be diagnosticated. Carcinoma and sarcoma are the most frequent, and lipoma, myxoma, fibroma, and myoma, are infrequent. They are usually secondary. Cardiac insufficiency, valvular compression or other disorder and embolism sometimes develop.

Parasites are most often accidental post mortem findings. In Mosler's monograph echinococcus is the most frequent and occurs twice as often in the right ventricle as in the left. Grulee (1905) collected 55 cases of echinococcus of the heart. Cardiac rupture, sudden death from occlusion of the pulmonary trunks, relative pulmonary insufficiency, or cardiac incompetence may occur. The cysticercus, the pentastomum and trichina are of purely anatomical interest.

SYPHILIS OF THE HEART (See SYPHILIS).

CARDIAC THROMBOSIS—THROMBOSIS CORDIS.

This usually occurs at the time of or after death, and is of little clinical interest.

Symptoms.—(*a*) Entire latency. (*b*) Thrombosis sometimes develops during life and leads to pulmonary or arterial embolism. (*c*) *Sudden cardiac weakness.* (*d*) *Sudden valvular* stenosis, a ball thrombus (*kugelthrombus*) forming in the auricle, and occluding, for instance, the mitral orifice; it is beyond clinical recognition, although according to von Ziemssen, it is frequently associated with gangrene of the lower extremities; in Curschmann's case an ascites resembling chronic peritonitis was the sole finding.

NEUROSES OF THE HEART.

PALPITATION OF THE HEART.

Palpitation is also known as cardiopalmus, cardiogmus, or hyperkinesis cordis, and is purely a symptom. In health and in many cases of severe heart disease the patient is not conscious of his heart-action.

The most extreme irregularity (delirium cordis), or the pounding hea caused by aortic insufficiency, may escape the patient's notice, thoug the normal heart's action may cause palpitation after overwork, fro abuse of coffee and other causes.

Etiology.—**1. Nervous Causes (Hyperæsthesia of the Centripe Fibers from the Heart to the Brain).**—Nervous excitability is commo especially in medical students, whose hearts must therefore be examin repeatedly when they first study physiology or physical diagnos Even physicians are not wholly free from heart egotism. Peter Fra had violent palpitation while writing his "Diseases of the Heart so that he thought he had aneurysm. It is common in school ch dren, and results from nervous tension, fear of examinations a public exercises. It is more frequent in women and may be connect with puberty, the climacteric, or menstruation. It occurs almost co stantly in exophthalmic goitre and very commonly (50 per cent.) neurasthenics, so that a special neurasthenia cordis has been reco nized, which is attended by turgid face, red eyes, contracted tempo arteries, and angioneurotic œdema, which symptoms disappear wh the seizure is over. Hysteria is a less important cause. Nervo exhaustion, chlorosis, pernicious anæmia, acute infections, lactatio poor hygiene, the beginning of tuberculosis, and sexual excess or pe version, may produce palpitation. The "irritable" heart of soldie observed by Da Costa in our Civil War, which is associated wi arrhythmia, diarrhœa, emotional causes, and physical strain, is oft placed under this head, but is better classified as muscular insufficienc

2. Reflex Causes.—This type might be included under nervo palpitation. It follows dyspepsia, especially after a heavy meal, a peptones or other products of digestion have been thought caus Dyspepsia may cause it by mechanical gastric distention or by toxæm It may also result from constipation, intestinal parasites, gall-sto (Kehr), renal calculi (Potain and Houchard), puncture of ascit prostatic, uterine, or ovarian disease, floating kidney, and disease the teeth and nose.

3. Toxic Causes.—Palpitation may result from the use of cof tea, alcohol, tobacco, narcotics, and from gout.

4. Cardiac Causes.—Palpitation may occur in organic heart dise *viz.:* valvular disease, acute myocarditis, hypertrophy, dilatat coronary atheroma, and in obesity. It may be symptomatic of h failure, as in the heart of nephritics.

Symptoms.—The patient complains of "feeling the heart be "goneness," "fluttering," forcible, irregular, or intermittent actio the heart, dyspnœa, anxiety, vertigo, tinnitus, and spots before eyes. In some instances, the symptoms recur periodically. In mittent epigastric throbbing is not infrequently present. Vasomo symptoms, as flushing of the face, spastic urine, etc., are not uncommo

Physical Examination.—Sometimes the heart is absolutely norma Conceptions of palpitation differ widely, and some authors describ it under tachycardia. The heart's rate is often increased, going u suddenly after exertion or fright to 100 or 150, with violent arterial throbbing. The increased rate may be due to stimulation of the accelerator nerves or of the vagus. The heart is rarely slowed, as in Binswanger's case. The rhythm may be normal or disturbed. A series of rather rapid beats followed by a series of slower beats is possibly somewhat characteristic of nervous palpitation. If the pulse is irregular when the patient is quiet it sometimes becomes regular after exertion. The first apex tone is often accentuated, is even metallic, or may be heard by the patient, or by the physician at some distance from the patient's chest. In some nervous subjects the systole sounds short (Müller). The second tone at the base is more often strong than weak. A murmur over the pulmonary region is usually functional.

Diagnosis.—(*a*) The *etiology* is of the greatest importance. It is confirmed by (*b*) *negative results* of examination of the heart during the attacks, but particularly in the intervals. Coronary disease, arteriosclerosis, myocarditis, etc., are usually excluded with ease, but it should always be remembered that nervous palpitation may co-exist with organic difficulties. Palpitation is held by some to lead to dilatation or hypertrophy, but this is probably confusion of cause and effect. (*c*) If pain be present in nervous palpitation, it is more often lower (an "*epigastric cramp*") than that of organic lesions, the pain of which radiates to the shoulder or arms.

Prognosis.—Though palpitation is sometimes lifelong, the outlook is generally favorable, but this depends on the etiology. The old belief that fright could cause death is as unfounded as most popular conceptions, and few such instances are recorded (Bollinger, Laache).

Treatment.—Treatment must be *based on the cause.* (*a*) If the cause is *nervous, suggestion* is valuable and repeated examinations of the heart and body are essential, in order to assure the patient and to avoid error. Regulated exercise, tepid baths with active friction, light meals, regular habits, and possibly the rest cure of Mitchell, are indicated. It seems from personal experience that isolation often increases introspection. In anæmic cases full doses of iron, arsenic, and strychnine should be given. Goddell's combination of the three valerianates is valuable:

℞
Ferri valerianatis.
Quininæ valerianatis.
Ammon. valerianatis . . . . .āā gr. xx.
M. et ft. in pil. xx.
S.—One pill after meals.

In cases of violent throbbing, a few doses of veratrum often quiet the general and cardiac nervousness. A belladonna plaster or a few drops of the fluidextract helps the patient greatly by anæsthetizing the peripheral fibers of the vagus. Sexual hygiene must be strictly observed.

℞

Fluidextr. veratri	gtt. x.
Fluidextr. belladonnæ	gtt. xv.
Aquæ	q.s. ad ℥j.

M. et S.—One teaspoonful every half hour for two or three doses.

(*b*) When the case is *reflex*, flatulency and constipation must be treat and the nose sometimes should be cauterized. Bromides are of va but must be given well diluted or in pepsin elixir, in order to av added gastro-intestinal disorder.

℞

Sodii bromidi	ʒj.
Spts. ammon. aromat.	℥ss.
Syr. zingiberis	ʒiij.
Aquæ	q.s. ad ℥ij.

M. et S.—One teaspoonful in hot water every half-hour for four doses.

A light evening meal of non-nitrogenous food is beneficial in child afflicted with night terrors or palpitation. (*c*) *The toxic type* is eas treated when the *noxa* is recognized. (*d*) *In cardiac disease*, the c diants, morphia, belladonna, aconite and the ice-bag are indicat (*v.* VALVULAR DISEASE). Digitalis is of little use in the truly nervo type.

TACHYCARDIA.

Definition.—Tachycardia is the usual term for rapid heart actio although polycardia and pyknocardia are better etymologically, f tachycardia implies quick (not rapid) heart action like the *pulsus cele* Tachycardia, like palpitation, is only a symptom. Some hearts no mally beat 90 to 100 (120 in the case of Congreve).

Etiology.—Larcenia's division, somewhat modified, is as follow (*a*) *Heart and vascular disease*, coronary or valvular affections, a acute or chronic myocarditis. (*b*) *Nervous affections*, (i) brain a cord disease, as of the medulla (vagus centre), tumors, softeni hemorrhage, multiple sclerosis, acute ascending paralysis, bu paralysis, injury especially to the cervical spine, myelitis and ta (ii) neuritis, compression of the trunk of the vagus by tumors, tation of the accelerator nerve, neuroses as exophthalmic g emotional tachycardia, and reflex tachycardia from uterine or ov disease or from the menopause. (*c*) *General diseases* such as f chronic troubles (tuberculosis, cancer); and *convalescence, exha* or *excesses*. (*d*) *Toxic* causes, resulting from tea, coffee, alcoho digitalis, atropine, amyl nitrite, etc. Cases are recorded (Tarc in which individuals by voluntarily moving the ear or skin mu the neck can raise the heart's rate, through the spinal accesso and the vagus.

A special variety is the *tachycardia paroxysmalis*, describe Proebsting (1881) and later by Nothnagel, Bouveret, and of rapid heart action occur abruptly, last from a fe even weeks, and usually end abrup type is seen mostly in strong, hard-working men, who indulge in excesses in alcohol, tobacco, etc. Broadbent saw a case at ten years of age.

The pulse becomes small, is usually weaker and soft, and its rate reaches 150, 250 (Bowles), or even 280 (Bristowe). It is at times strong (Brieger, Nothnagel) and is usually regular, though arrhythmia may develop at the beginning and end of the seizure. The sphygmogram reveals more beats than does palpation. The heart often dilates (Martius), for the tachycardia and dilatation are coördinate to the causal factor. The pause of the heart (the time between systoles) is lessened (François Frank) and the systole is incomplete. The systolic tone is often clicking. The apex is apparently strong, but throbbing is an index of weakness and dilatation. Embryocardia (Stokes, Huchard) is sometimes present while heart murmurs are rare. The vessels, especially the carotids, pulsate violently, and the patient is anxious, depressed, and frequently shows signs of venous stasis or cyanosis, although the face is at times flushed. The urine is of low specific gravity. The lungs are usually normal but Riegel, Tuczek, Edinger and Kredel, describe an acute distention of the lungs like that observed in valvular disease, which may be caused by vagus or phrenic irritation or tonic contraction of the diaphragm.

Diagnosis.—Permanent tachycardia suggests organic disease of the centres in the medulla or nerve trunks. The physiology of the vagus and accelerator nerves from the clinical standpoint is still obscure. It has been held that (*a*) increase in the pulse up to 120 indicates sympathetic irritation (accelerator stimulation), which is relieved by morphine; (*b*) increase from 120 to 180 indicates paralysis of the vagus, and this is relieved by pressure on the vagus trunk; (*c*) increase over 180 is indicative of sympathetic irritation in addition to paralysis of the vagus. Sympathetic symptoms may prevail, as protrusion of the eyeball, wide pupils, and increase in the interpalpebral fissure; other evidences of vagus involvement may exist, such as respiratory, laryngeal, or gastric symptoms. The *diagnosis* of *vagus neurosis* rests upon (*a*) the exclusion of organic heart and brain disease, (*b*) a high pulse rate, *e. g.*, 150; (*c*) occurrence in paroxysms with intervals of normal pulse rate; (*d*) inhibitive action upon the paroxysm of manual compression or of electrical stimulation of the vagus.

Prognosis.—The prognosis varies with the cause and type; the permanent variety is less favorable than the paroxysmal and temporary forms. The outlook is generally good. Bouveret holds that permanent cure is rare and has reported four cases of sudden death. The infrequent autopsies (only 14 in 135 cases collected by Hoffmann in 1900) show degenerations, etc., in the heart muscle. It is well to regard with doubt and caution all cases of tachycardia other than those resulting from emotional causes.

Treatment.—Stimulation of the vagus is effected by faradization of the neck, holding of the breath, by Valsalva's experiment, by pressure on the right vagus, the use of the ice-bag, lying down, swallowing ice, or by induction of vomiting by apomorphine. Morphine, asafœtida, valerian, and the bromides, are frequently of value. The causal factors mu receive consideration.

BRADYCARDIA.

Definition.—Bradycardia, spanocardia or brachycardia is the usu term for *slow heart* but *oligocardia* is a better designation. A *puls* *tardus* is normal to some individuals and in certain families may seen in successive generations. It is often an accommodative effort in aortic stenosis. The apparent pulse rate must be verified by cardi auscultation because the pulse record is often but half as frequent as t actual heart rate. The pulse rate may fall to 20 or even to 6 beats minute. Paroxysmal bradycardia may occur with weak heart, pal tation, difficulty in breathing, or gastric distress.

Following Riegel's *classification*, two types are distinguished: (*Physiological* bradycardia, which is rather infrequent in occurrenc it is observed in starvation, and during the puerperium; the rate from 34 to 60. (2) *Pathological bradycardia* may show 6 to 9 beats minute (Norfleet), and the pulse and breathing may run parallel.

Etiology.—The etiological factors of the pathological type are: (*Acute infections;* bradycardia is very common during or after typhoi pneumonia, diphtheria, and rheumatism. Traube explained the slo pulse as due to exhaustion, but it is probably caused by acute my carditis, which induces lessened conductivity, or "heart-block." (*Digestive disorders,* which caused the majority of Riegel's collectio jaundice, especially light forms, constipation, gastric cancer or ulce vomiting (which causes bradycardia and arrhythmia in children), ar gall stones are the most frequent causes. (*c*) *Respiratory disease* is less frequent cause; emphysema occurs frequently with a slow stro pulse, which is sometimes an unfavorable symptom. (*d*) *Circulato* *disease,* such as aortic stenosis, fatty heart, *coronary atheroma* or chron myocarditis, is an etiological factor and lessens the conduction an contractibility of the heart muscle. Dehio uses atropine to distinguis between (i) disease of the heart muscle, upon which the drug has b little effect, and (ii) slow heart due to inhibition of the vagus (trunk centre) in which it brings the heart to or above the normal rate. (*Toxic conditions* such as poisoning from lead, alcohol, tobacco (corona spasm), canned meats, coffee, muscarine, digitalis and other drugs ma cause bradycardia; it also occurs in cholæmia and early in uræmia, i which cases it is unfavorable. (*f*) *General troubles* are also causativ such as diabetes, cachexia, anæmia or exhaustion, all of which lesse the irritability of the heart muscle. (*g*) *Nervous conditions,* frigh apoplexy, epilepsy, meningitis, brain tumor, disease or injury of t medulla or cervical cord, general paralysis, melancholia, hysteria, vag stimulation by tumors, and compression of the vagus trunk, are al factors in its development. Jacob describes an angiospastic for (bradycardia with chills, wide pupils, cold skin, loud second aort tone, etc).

The *Adams-Stokes syndrome* (described under fatty heart) results chiefly from arteriosclerotic changes in the medulla and heart, but rarely from toxins or neuroses. The causal condition is "*heart block,*" in which the transmission of the peristaltic contraction wave from the auricle to the ventricle is "blocked"; the ventricles contract two to four or more times less frequently than the auricles, whose movements may be seen in the pulsating cervical veins or by the fluoroscope. The syndrome consists of (i) bradycardia (with arrhythmia and hemisystole); (ii) cerebral disturbance (syncope, apoplectiform or epileptiform seizures); and (iii) accessory symptoms (Cheyne-Stokes respiration, angina pectoris, etc.).

In 1903 His, junior, found a band-like muscular bundle connecting the auricles and ventricles, which is known as the "bundle of His." It measures 18 millimeters in length, 2.5 millimeters in width, and 1.5 millimeters in thickness, and courses posteriorly in the septum ventriculorum, whence it reaches the right auricle and its valves. According to Erlanger (1906), slight compression of His's bundle increases the interventricular pause; marked compression causes intermittency in the ventricular contractions; complete compression results in independent contraction of the auricles and ventricles. In "heart-block" the vagus loses control of the ventricle but the accelerator does not. The epileptiform attacks of the syndrome are due to cerebral anæmia and the apoplectiform attacks result from cerebral venous congestion. In a case of the writer, myofibrosis was found; in the case of Stengel, an endocarditic patch; and in the case of Jellick, Cooper and Ophuls, a focus of acute anæmic necrosis (myomalacia cordis.).

Prognosis.—The constant is less favorable than the paroxysmal type.

Treatment.—The causal therapy alone is of value. Morphine and atropine have been recommended, but must be given with care where there is disease of the central nervous system. Strong cardiants, such as digitalis, are usually contra-indicated and in the Adams-Stokes syndrome are actually dangerous. Alcohol is injurious. In the Adams-Stokes syndrome, nitroglycerine is indicated, while bromides are deleterious.

ARRHYTHMIA.

In an irregular heart the individual beats are irregular in rhythm, in strength, or in both.

Etiology.—The cases are (*a*) *nervous*, (i) resulting from brain disease, as meningitis, concussion, or pressure from hemorrhage or tumor, (ii) psychical, (iii) reflex, usually from the digestive tract, lungs and kidney; or (*b*) *cardiac*, (i) *toxic*, from tobacco, alcohol, tea, coffee or digitalis, or (ii) *organic*, from changes in the peri-, myo- or endocardium. Changes in the heart ganglia, which have been considered causes, are probably in most cases the result rather than the cause. The recent studies of Gaskell, Englemann, Mackenzie, and Wenckebach, have shown that the heart's function resides in the heart itself (myogenic contractility, conductivity, excitability, tonicity, and rhythmicity).

Varieties.—Varieties are distinguished as follows: (*a*) *Intermittence.* The dropping of a beat may be complete or rudimentary (a slight indication of the deficient systole is recognized in a faint first tone). Some persons can voluntarily drop a beat by contracting the neck muscles, probably through reflex action upon the vagus by the spinal accessory nerve which supplies the muscles (Donders). (*b*) *Delirium cordis*, in which the greatest irregularity prevails—"*folie cardiaque.*" (*c*) *Embryocardia*, the fœtal heart rhythm of Stokes, and (*d*) the *gallop-rhythm* of the heart in cases of renal disease, hypertrophy, etc., which has been described under DILATATION. (*e*) *The alternate heart beat*, in which a strong heart beat is followed by a weak one, with corresponding findings in the pulse (*pulsus alternans*). When the irregularity recurs at even intervals the term allorrhythmia is employed. (*f*) *The bigeminal heart beat* consists of a strong beat quickly followed by a weaker one, with a pause before the next similar group; the second beat is often weak, and may not reach the radial vessels; this variety occurs frequently in mitral disease and in the cumulative effects of digitalis. The trigeminal heart beat and pulse are similar, except that each group comprises three beats. They usually indicate heart weakness. (*g*) *The pulsus paradoxus*, intermitting with inspiration, has been considered under *concretio cordis* (*v.* PERICARDITIS). It may be marked in young persons, in convalescence, neurasthenia and brain disease.

ANGINA PECTORIS.

Angina pectoris (stenocardia, breast-pang) was first described b Heberden and then by Hunter. Edward Jenner first recognized th connection between angina pectoris and coronary disease. By far th best treatises are Osler's classic on *Angina Pectoris and Allied State* and Neusser's Heft 2, *Ausgewählte Kapitel der klinischen Symptomatologie.*

Definition.—An arteriosclerotic cardiopathy, characterized (*a*) b paroxysms of excruciating pain over the heart, which radiates to th neck and arms, (*b*) by mental anguish, a sense of dying, and (*c*) fr quently by sudden death.

Etiology and Pathology.—The chief and practically constant path logical finding is arteriosclerosis involving the heart muscle directly b coronary atheroma, or indirectly by plaques in the aorta at the corona orifices. The aorta is often atheromatous without coronary involv ment and coronary sclerosis is frequent without much change in t aorta.

Though it is usually described under cardiac neuroses, its prop place is under arteriosclerosis. It is notable that atheroma is freque and angina pectoris is rare. The etiology is that of arteriosclerosis (*q. v.*), syphilis, gout, alcohol, contracted kidneys, sometimes infections such as rheumatism or influenza. A history of its occurrence through several generations of a family is sometimes elicited. It occurs chiefly in men (80 to 90 per cent. of cases or more), usually after the fortieth year (although cases in the second decennium have been reported) and in the well-to-do classes, professional men, hard workers, and luxurious livers. Angina is far more common in private than in hospital practice.

Symptoms.—Mode of Onset.—(*a*) In some cases the *onset is gradual*, and is preceded by dyspnœa (especially when dressing or undressing, lacing the shoes, emptying the bowels, sitting up, or beginning to exercise), by short deep respiration, precordial oppression, pulmonary œdema, bronchial catarrh, strong beating in the second or third left interspaces, and difficulty in moving the left arm. This cardiac weakness ("cardiac asthma"), is followed by genuine angina. Heart weakness and angina are coördinate phenomena, secondary to coronary disease. (*b*) In other cases *angina is the first important symptom* of cardiac disease. It is excited by (i) muscular effort, as walking up hill; (ii) mental excitement, as anger or coitus; (iii) chilling or bathing; (iv) flatulent dyspepsia or overloading the stomach, which may induce the attack when the patient is quiet or just going to sleep. (*c*) *Death* may be the first symptom.

Paroxysm.—The attack itself consists of (1) *sudden agonizing pain*, which is lancinating and feels like an iron hand squeezing the heart. Its explanation is disputed and nearly one hundred theories have been elaborated. The pain emanates from the cardiac plexus lying behind the aorta, and in the nerve branches from it to the coronary arteries, whence its reflection to other parts, which will be mentioned later. The best explanation, pathologically, is that during the seizure the *heart muscle is ischæmic* from coronary obstruction or possibly from superinduced spasm of the vessel (Allan Burns, Potain). In man and in the horse, atheroma or thrombosis of the abdominal aorta or its branches to the legs, shuts off the blood necessary during exertion; with moderate exercise the collateral circulation is sufficient, but prolonged exertion produces tingling, cramps, and transient paraplegia ("intermittent claudication"). The heart-pang is caused by an analogous coronary ischæmia when unusual stress is thrown on the diseased coronaries. In the case of the author's father, two seizures of angina were followed by "claudication," albuminuric retinitis and the Adams-Stokes syndrome; the abdominal aorta was clearly to be seen and felt as a hard uneven rigid tube. The "unutterable" pain is usually *substernal* and *radiates* to the left shoulder, neck and arm. Irradiation may be observed to the chin, ear, jaw, occiput, larynx, and even to the gall-bladder and renal region, spine, stomach, left leg and testis (which may swell). In aortic disease, pain is propagated to the first four dorsal areas; in angina it also reaches the cervical and lower dorsal areas. Pain *along the ulnar nerve* to the fingers may precede the angina; it is usually associated with some motor weakness and, in Eichhorst's remarkable case, with ulnar atrophy. Numbness or hyperæsthesia of the arm, chest, or mammary regions is frequent. The anginal pain produces vasomotor symptoms, such as great pallor, low temperature, and profuse sweating. The patient may fall, perhaps in a fatal syncope, or more often may support himself for a few seconds or minutes until the paroxysm has passed.

(2) The *mental anguish*, the *angor animi*, which was first recognized by Latham, is the second element of the seizure and consists of a sense of dying, with great anxiety, complete consciousness, immobile attitude, and perhaps inability to articulate.

(3) The *danger of sudden death* is the third element. Death may come without pain, in syncope (syncope anginosa) in the first seizure, after subsequent seizures which may occur years after the initial paroxysm, or after constantly and closely repeated attacks (the *état angineuse* of Houchard, which is analogous to the status epilepticus). Osler has called the anterior branch of the coronary artery the "artery of sudden death." Death in angina is analogous to death from experimental injury to Kronecker's point in the septum. It usually occurs in the second or third attack.

Other Signs and Symptoms.—The *heart is variable*, is sometimes normal in rate and strength, and again shows a tense or weak, slow or rapid, or irregular pulse. The left radial may be smaller than the right. A slow pulse usually results from partial "heart-block." Dunin found the arterial pressure low in 45 per cent. and high in 20 per cent. of his cases. Delirium cordis, gallop-rhythm, and shortening of the long pause, are but signs of cardiac fatigue.

The second aortic tone is often loud and metallic; a rough or blowing systolic (and sometimes a diastolic) murmur is heard over the aorta, which is sometimes dilated; and the left ventricle may be dilated, thus sometimes relieving the attack by relative mitral leakage (Broadbent and Musser). These incidents are simply evidences of arteriosclerosis and its sequences. *Constitutional disturbance* very frequently occurs, as disturbance in nutrition, loss in weight, color, and physical endurance. *Dyspnœa* is not a part of angina pectoris, but is solely symptomatic of heart insufficiency. Involuntary evacuations or unsuccessful straining at stool are not very frequent. The urine is often pale and abundant after the seizure. Dysphagia, singultus or gastric symptoms are due to sympathetic participation of the pneumogastric and phrenic nerves.

Diagnosis.—When the attack is typical and severe, there is little difficulty in making a diagnosis. Mild or early attacks may be confused with cardiac asthma, because angina may be preceded by cardiac distress, or associated with it. Seizures of angina involving the abdomen (angina abdominis) or legs (angina cruris) are easily mistaken unless the case is closely watched; these forms may appear first or may alternate with the more usual type—angina pectoris. The classical picture, together with hardness of the arteries and atheroma of the aorta or aortic valves, makes the diagnosis practically certain. The respiration is essentially normal during the attack. Atheroma with cardiac asthma is not angina pectoris.

Pseudo-angina (Latham and Walshe) is the most frequent cause of diagnostic error. Its varieties are (*a*) *nervous pseudo-angina*, which occurs in cases of neurasthenia and hysteria, in other neuroses, and in the crises of tabes (which in Oppenheim's case was due to vagus neuritis). Huchard's widely quoted table is substantially as follows:

TRUE ANGINA——*vs.*	——PSEUDO-ANGINA.
Most common in men between forty and fifty.	Commonest in women, at any age.
Paroxysms induced by exertion, etc.; diurnal; few in number.	Spontaneous; often nocturnal and periodic; frequent.
Pain intense, of short duration, precordial, inarticulate; immobile attitude; sensation of cardiac compression.	Less severe, lasting hours; epigastric; agitation, activity; sensation of distention.
No nervous symptoms.	Neurasthenic or hysterical stigmata.
Vasomotor form rare.	Common.
Prognosis grave or fatal.	Never fatal.
Lesions: from coronary sclerosis.	From neuralgia.
Arterial medication.	Neuralgic medication successful.

(*b*) *Vasomotor angina* (Nothnagel) which is not uncommonly attended by vasomotor spasm, coldness and numbness in the extremities, a feeling of faintness and great pain in the heart. It may be reflex in origin.

(*c*) The *toxic pseudo-angina* (Beau, Graves and Stokes) is due to coffee, etc., but especially to tobacco. Vasomotor symptoms, amaurosis, dyspepsia, weak, rapid, or irregular heart action, palpitation, and anginal pain are common. Functional angina from tobacco, which results according to Huchard from coronary spasm, heals when tobacco is withheld; but actual coronary disease, due to tobacco and other heart poisons, is not curable. (*d*) The crises of tabes and precordial pain due to brachial neuritis, vagus involvement or gout, etc., may be difficult to diagnosticate.

The **prognosis** is self-evident. Death is frequent. In one instance the patient lived forty years. Few clear recoveries are reported.

Treatment.—The treatment concerns (*a*) *the etiology*, in which arteriosclerosis is the essential factor. Vierordt advises that the possibility of syphilis should always be considered. Gout and diabetes necessitate appropriate diet and therapy, as colchicum. Attention must be directed to the question of stimulants. Aside from syphilis, the iodides are valuable and should be given two-thirds of the time, for months and years. Small amounts are frequently tolerated less well than 10 or 20 grain doses. Care in their use is necessary in nephritis, and even when they are well diluted gastric disorder may result and possibly induce an attack of angina. Nitroglycerine or its derivatives

are indicated in the intervals, in doses of gr. $\frac{1}{100}$ to $\frac{1}{50}$ four or
times daily, but it should be remembered that its effects last less
an hour. It may cause headache, flushing, or (in larger doses) cya
or pallor, muscular relaxation, dyspnœa and irregular pulse. Ery
tetranitrate in doses of gr. ss. t. i. d. has a more lasting effect
nitroglycerine. These vasodilators will be again considered u
ARTERIOSCLEROSIS. (*b*) *The causes precipitating paroxysms.*
phylaxis should be maintained regarding (i) exertion, (ii) exciter
worry, (iii) coitus, (iv) chilling, (v) constipation, and (vi) dyspe
late suppers and foods favoring flatulency should be avoided,
a light evening meal should be taken. The patient should be prov
with a mixture of aromatics in order to dispel gastric flatulency; a s
dose is contained in the following:

℞

Spiritus ammoniæ aromatici	ʒss.
Spiritus chloroformi	ʒss.
Spiritus glycerylis nitratis	gtt. i.
Spiritus ætheris compositi	ʒss.

M. et S.—To be taken in hot water.

(*c*) The *paroxysm. Amyl nitrite* was first advised by Brunton
an analgesic and a vasodilator; the patient should carry pearls o
drug, which were invented by Solger and contain three minims o
remedy. They often operate powerfully and instantaneously, th
sometimes they are useless. It may be better to have the patient
a hypodermic tablet of nitroglycerine grain $\frac{1}{100}$, for the physician
rarely sees the attack. Some member of the patient's family shoul
taught how to give a hypodermic injection of morphine. Morphine
chloroform inhalations have been held dangerous, especially
brain trouble exists, as is sometimes the case in the Adams-Stokes t
Nevertheless they must be used in some cases. Yeo called atter
to the large doses of morphia required and tolerated by subjects ha
angina. (*d*) *Cardiac weakness,* which is treated by diffuse stimul
such as ammonia, camphor, alcohol and strychnine. When art
tension is low, digitalis may be administered between paroxysms:

℞

Tr. digitalis	ʒiv.
Tr. strophanthi	ʒij.
Tr. valerianæ	q.s. ad ℥iv.

M. et S.—One teaspoonful after meals.

Vierordt says that morphine should be carried in one hand
stimulants in the other. Coffee is said to be prophylactic against car
weakness and the paroxysms. *Pseudo-angina* is treated accordin
the type. Electricity and arsenic are valuable.

DISEASES OF THE ARTERIES.

ARTERIOSCLEROSIS.

Synonyms.—Atheroma, arteriocapillary fibrosis (Gull and Sutton), endarteritis deformans (Virchow), angiosclerosis (Thoma).

Definition.—A regressive change, evidenced first by malnutrition in the walls of the large or small vessels and later by compensatory thickening, either localized or diffuse.

Etiology.—1. **Age.**—Arteriosclerosis is the essential element in senile involution, although some individuals attain their hundredth year with no arterial change. Though usually found after the fiftieth year, hereditary influences or certain infections, as syphilis, may develop hard arteries between the twentieth and fortieth year. Thoma frequently found a moderate degree of hardness after the thirty-sixth year. In adolescence or even in infancy it is sometimes found as a result of the acute infections; Chiari found it in 27 per cent. of those under 25 years.

2. **Sex.**—Eighty per cent. of cases occurs in men, from causes to be enumerated below. In women it usually occurs a decade later than in men. In factory women over fifty, Jusserand found even a larger percentage than in men.

3. **Arterial Poisons.**—*Chronic alcoholism* causes 25 per cent. of all cases (Edgren); it is not known how the arterial poisons operate; they do not raise arterial pressure; coffee, tea, and tobacco (which is said to affect especially the coronary vessels), are causative factors. Angiosclerosis has been produced experimentally by adrenalin.

4. **Overwork.**—This is second only to alcohol in importance. Physical exertion brings one-third of the circulating blood to the muscles, thus lowering the tension in other vessels (*v. i.* THOMA'S THEORY). According to an older theory exertion raises arterial tension.

5. **Poisons Produced Within the Body by Perverted Metabolism.**—*Interstitial nephritis* bears a threefold relation to arteriosclerosis: (*a*) it results from diffuse arterial hardening; (*b*) it raises arterial pressure mechanically (Traube) by increased peripheral resistance in the arterioles, or by alteration in the quality of the blood (Bright); (*c*) both processes are coördinate to a common cause. *Gout* certainly leads to arteriosclerosis. *Diabetes* bears a double relation, being either a rather rare result of arteriosclerosis of the pancreas (Fleiner), and possibly of the brain (Grabe), or itself causing arterial hardening. *High living, overeating, obesity,* and overindulgence in meat may be included under this head.

6. **Specific Infections.**—Edgren and Camac consider syphilis of first etiological importance in 33 per cent. of the cases. It operates by chronic toxæmia and endarteritis. Tuberculosis (in 16 per cent.) may produce changes in the vessels; for instance, tuberculous meningitis

may attack them from the perivascular lymphatics, especially those about the middle cerebral artery and its branches. Typhoid, rheumatism, influenza, scarlatina and other infections are held by French writers to have some bearing on vascular sclerosis and endarteritis. Oppenheimer has recently described arteriosclerosis in children nine and ten years old, obviously due to acute infections.

7. Other Causes.—Nervous factors (worry, high tension, neurasthenia) cause variations in blood pressure. Excessive brain effort tends toward cerebral localization precisely as digestive excesses may develop changes in the mesenteric vessels, just as working with the arms (as in the case of butchers) may develop changes in the brachials, and as excessive walking may produce arteriosclerosis in the femorals. Extreme heat, as in the work of bakers and stokers, is thought to induce sclerosis.

Pathogenesis and Pathology.—(*A*). Thoma considers angiosclerosis a compensatory process which follows widening of the vessels and alteration of the blood. To restore the caliber of the widened vessels, the tunica media contracts, or when it cannot contract, the vessel increases in thickness by growth of the subendothelial connective tissue of the intima. Thus in the fœtus there is no connective tissue in the tunica intima, but it appears when the fœtal circulation changes at birth, new areas of circulation being opened up. All bloodvessels have a nervous supply ending in the Vater-Pacini bodies. They respond to persistent low pressure, or dilatation, by increased proliferation of the connective tissue. Thoma observed arteriosclerosis develop in cases of experimentally produced aortic insufficiency. (*B*). Others regard arteriosclerosis as due to *inflammation in the nutrient vessels,* the vasa vasorum, leading to growth of connective tissue (Huchard, Koester, Hipp, Martin), *i. e.*, mesarteritis and periarteritis. When arteriosclerosis is seen in vessels with no vasa vasorum, it must be a simple regression or degeneration.

Conceptions of its morbid anatomy differ widely, atheroma of the aorta and endarteritis or arteriocapillary fibrosis being sometimes described under separate headings, but the difference is rather anatomical than pathological. Large vessels, such as the carotid, contain more elastic than muscular tissue, but the small vessels are largely muscular. Two main forms are distinguished, (1) the *diffuse,* and (2) the *nodular.* Without distinction as to form, Rokitansky found that arteriosclerosis occurred in the following order of frequency: ascending aorta, arch, thoracic, abdominal aorta, splenic, iliac, femoral, coronary (next after the aorta, according to Huchard and Sansom), cerebral, uterine, brachial, spermatic and carotid arteries. It is rare in the gastric and in the mesenteric and hepatic vessels and least frequent in the pulmonary trunk. Bergmann found the two varieties in the following percentages:

	DIFFUSE FORM.	NODULAR (NODOSE) VARIETY.
	Per cent.	*Per cent.*
Radial	89	2
Ulnar	88	8
Tibial	87	8
Popliteal	64	25
Splenic	64	17
Femoral	58	14
Axillary	53	16
External iliac	49	15
Brachial	49	9
Subclavian	45	43
External carotid	44	22
Cerebral	40	43
Internal carotid	28	60
Ascending aorta	19	56
Common carotid	10	69
Abdominal aorta	0	75

(Both forms may coëxist in the same vessel).

It will be noted that where the diffuse variety is most frequent the nodular form is least observed and that the converse is true. The *diffuse* form is most frequent in the extremities where arterial tension is most variable and more collateral circulation prevails; the *nodose form* is most common in the aorta and its primary branches, but is also seen in the circle of Willis, where arterial pressure varies greatly (Meynert).

To the *naked eye* the intima may be normal and smooth but most often is uneven and thickened. Small gelatinous or yellowish fatty areas are seen which though at first circumscribed often fuse into large plaques; they occur where arterial pressure is greatest, as in certain places in the aortic arch (See ANEURYSM), or where the vessel divides or gives off branches, as the subclavian or intercostal branches. These plaques of atheroma or nodular arteriosclerosis represent nature's effort to fill out areas of degeneration and dilatation and thus make even the caliber of the vessel. *Microscopically* the intima may be increased three or four fold from swelling of its tissue, increase of connective tissue and round-cell infiltration. Granular detritis, fatty needles and cholestearin plates are seen in the *atheroma plaques*, a name indicating their pulpy, grumous appearance. Formerly they were wrongly called *atheromatous abscesses*. An atheromatous area rupturing into the lumen produces the *atheromatous ulcer*, which may cause embolism, thrombosis or occulsion. Secondary deposit of carbonate or phosphate of lime in the atheromatous areas leads to *calcification* of the arteries or may produce rough areas in the aorta which break on manipulation or if handled carelessly may cut the finger. The lumen of the smaller vessels is often decreased or obliterated (endarteritis obliterans). The middle and outer coats of the smaller vessels are at times thickened from hyaline degeneration and fibrous tissue; the muscular coat may waste and all elastic tissue may disappear; the essential changes are those in the elastic tunic. In the larger vessels dilatation or even aneurysm may develop where thickening of the intima fails to protect the vessel wall.

The general results of arteriosclerosis are (1) *vascular,* and consist of loss of arterial elasticity, deformity, tortuosity, obliteration, aneurysm and high arterial tension, and (2) *visceral,* and occur in the heart, brain, kidney, and other organs (*v. i.*).

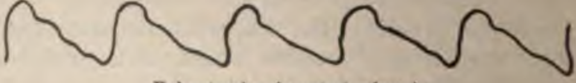
Sclerosis in the *pulmonary artery* is most common in the stasis and in the increased pulmonary tension of mitral lesions and emphysema; aside from these cases, the older writers maintained that it was always the result of general arteriosclerosis. The rare cases of Rhomberg, Klob and Neusser have shown that pulmonary atheroma may occur alone, sometimes with aneurysmatic dilatation of its branches and with pulmonary regurgitation.

The term angiosclerosis (Thoma) also includes *phlebosclerosis* ,which frequently accompanies arteriosclerosis and sometimes occurs independently; it is most often seen in the lower extremities and sometimes in the lung and in the portal tributaries in cases of liver cirrhosis or pylephlebitis.

Symptoms and Diagnosis.—In diffuse forms there may be no symptoms *i. e.* the process is *latent.* When it is local, visceral symptoms may develop. The clincial picture is extremely variable.

1. The *peripheral arteries* may be involved alone or there may be changes in the aorta and in the heart, brain and kidneys. The small arteries are visible and appear *tortuous, pulsating and lengthened,* as do the temporals or brachials when the elbow is flexed and the arm is rotated outward. Lifting the arm is said to decrease the pulsation, while this maneovre increases it in cases of aortic regurgitation. A distinction must be made between *hardness* of the vessel walls and *increased arterial tension,* although both conditions usually occur together. The normal systolic blood pressure measures 125 mm. (Strassburger, Sahli, Fellner) and the diastolic measures 29 mm. Dunin holds that the systolic pressure ranges between 130 and 150 mm., and that 21 per cent. of cases of arteriosclerosis have normal or decreased arterial tension. It is asserted that increased arterial tensio precedes actual sclerosis of the vessels but in these early cases it i probable that some angiosclerosis has already developed. Pressing o the radial with one finger should obliterate the pulse beyond that poi if the arteries are not sclerotic, unless there is an anastomotic or rec

Fig. 28.

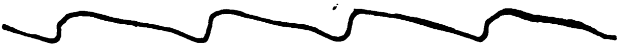


Pulse tracing in arteriosclerosis.

rent pulse from the ulnar to the radial vessel; this source of error easily eliminated by simultaneous pressure on the ulnar vessel. Th wall of the artery is firm, hard, often uneven or nodular, and the serial atheromatous plaques sometimes feel like a rosary or the trachea of a goose. In extreme instances the vessel feels calcareous—the "pipe stem artery." The Roentgen rays clearly show the arterial change.

The pulse is hard (*pulsus durus*), retarded (*pulsus tardus*), or slow (*pulsus rarus*); it rises slowly and is long maintained (*p. rotundus*), the vessel is firm between the pulse beats (Broadbent), the elastic wave is lessened or absent, the tracing has a rounded or flat top, and the pulse in the two radials may differ (*pulsus differens*). Marked slowing of the pulse calls attention to the coronary vessels. The bigeminal pulse is most rare unless there are heart, coronary, or brain changes. In some cases, either from vessel obliteration or because the walls are absolutely rigid, the pulse cannot be felt. Very slight pressure on the vessels often elicits a systolic murmur (Litten). The right radial is normally larger and more often shows changes than the left. It must be remembered that changes may occur in internal vessels without change in the radials (and conversely).

FIG. 29.



Pulse tracing in extreme arteriosclerosis (Sahli).

2. There may be *heart changes*. (*a*) *Simple hypertrophy* of the left ventricle may occur, with heaving apex, loud second aortic tone, tense pulse, and thickness of the arteries, which lead to a certain and immediate diagnosis. Some " consensual " right heart hypertrophy is common because of anastomosis between the ventricular muscular fibers or because of stasis in the lungs or emphysema. Those cases in which there is increased tension in the aorta without increased tension in the radials are more difficult of diagnosis. The heart is not hypertrophied in all cases nor, according to Rhomberg, in the majority of cases; hypertrophy, he maintains, does not necessarily follow arteriosclerosis in the extremities or brain, but most often occurs when the splanchnic vessels are involved; Hasenfeld also believes that cardiac hypertrophy only occurs when these vessels and the aorta are sclerosed. The apex may sink outward and to the left when the patient sits, from relaxation and lengthening of the aorta (Traube). (*b*) *Hypertrophy plus dilatation*, with heart insufficiency, dyspnœa, scanty urine, and hydrothorax, is very common. When the patient in this stage has a systolic murmur, a wrong diagnosis of mitral insufficiency may be made. Interstitial nephritis (*q. v.*) may be the cause, in part at least, of the hypertrophy and dilatation. Œdema is not frequent. Gallop-rhythm is uncommon without coincident coronary or renal disease. The heart muscle may show various changes, such as atrophy, myocarditis, myofibrosis, myomalacia cordis, the *cor renale*, senile heart, heart aneurysm, angina pectoris, coronary embolism, cardiac asthma, fatty heart, and arrhythmia, or sudden death may occur. Two forms of visceral sclerosis may be distinguished: (*a*) inflammatory, occurring about the vessels, and (*b*) dystrophic (malnutritive) sclerosis, which is often somewhat distant from the vessel involved; the visceral sclerosis is best illustrated by the heart changes, such as myofibrosis or myomalacia cordis, induced by coronary atheroma.

3. In *the aorta* dilatation may occur, giving slight *dulness in the second (and third) right intercostal spaces;* this is confirmed by the fluoroscope; the aorta may be felt in the jugulum or abdomen. The normal aorta may be felt in these situations when the heart action is strong, but when there is atheroma, plaques or even a crackling may sometimes be felt. The subclavian, or innominate arteries may be felt even through their entire length when the aorta is dilated. *A short systolic murmur* over the aorta often results from atheromatous roughening or dilatation, and the *second aortic tone is then accentuated* and metallic. Its vibration may sometimes be felt; when much emphysema is present it is best heard in the jugulum. The accentuation may be caused by increased tension, widening, or rigidity of the aorta. The murmur and sharp tone are often heard in the neck and back. Aside from nervous sensations, a burning pain back of the sternum may indicate involvement of the outer coat of the vessel. Atheroma of the arch may, by downward extension, cause aortic stenosis or more often aortic regurgitation.

4. *Renal findings* are next in frequency. The kidneys are often contracted (the senile arteriosclerotic kidney and the genuine contracted kidney). The discussion of the question whether the renal findings are primary or secondary is a separate literature in itself (see INTERSTITIAL NEPHRITIS). The urine may be increased as a result of simple increase in the blood pressure.

5. *Brain symptoms* are next in importance. Syncope, vertigo, insomnia, headache, tinnitus, neurasthenic symptoms, transient aphasia or hemiplegia are difficult to explain, though they are said to be caused by transient ischæmia. Brain hemorrhage and miliary aneurysms are frequent sequences. Softening of the brain (encephalomalacia) results from embolism or thrombosis of the cerebral arteries. Brain atrophy, dementia (cortical sclerosis), epileptic seizures and the Adams-Stokes triad have been attributed to cerebral atheroma. Vascular brain lesions occur fairly often in cases which show arteriosclerosis in the extremities and temporal vessels, though sclerosis of the aorta, coronary, or splanchnic vessels occurs more often without peripheral angiosclerosis.

Charcot's *claudication intermittente* (Erb's dysbasia intermittens angiosclerotica, intermittent limping, angina cruris) occurs especially in nervous or neurasthenic subjects. It is due to atheroma in the abdominal aorta and its branches in the limbs. It is seen in abdominal aneurysm in the horse. Erb, reviewing 127 cases in 1904, found that 94 per cent. were in men over 40 years of age; heredity is sometimes causal: 33 per cent. were Jews; syphilis (in 23 per cent.) was less frequently causal than tobacco; alcohol was not proven an etiological factor; most cases developed in the upper classes. Pain, paræsthesiæ, and sometimes local cyanosis, occur in the legs, which "give way" after moderate exertion. The legs are most comfortable when dependent. The dorsalis pedis, sometimes also the tibial and femoral vessels, usually cannot be felt. Gangrene may result.

Some of the vasomotor neuroses—as erythromelalgia, Reynaud's disease, acroparæsthesia—and some forms of neuritis and neuralgia are sometimes associated with angiosclerosis.

6. *The gastro-intestinal* symptoms probably rank next in frequency. Hasenfeld, Huchard, Allbutt, Litten, Edgren and others have described dyspepsia, round ulcer, arteriosclerotic gastritis, gastro-intestinal hemorrhages, intestinal perforation and ulceration. Severe abdominal pain (angina abdominis) has attracted some attention lately; it is due, as is angina, to anæmia (Oertner's intermittent claudication of the abdominal vessels, Schützler's intermittent dysperistalsis); it develops in two or three hours after eating, and is often accompanied by dyspnœa. Abdominal angina, in the writer's experience, is rather often associated with angina pectoris and angina cruris; in three cases these conditions appeared alternately.

7. The *retinal vessels* are not infrequently embolized, narrowed, or aneurysmatic, which lead to softening, hemorrhage, and exudation.

8. In the *respiratory tract*, emphysema is common and is sometimes regarded as a compensatory effort. The breathing is often somewhat irregular. Hæmoptysis is referable to stasis, infarction, or possibly to atheroma of the pulmonary arterioles.

9. In the *peripheral vessels* the so-called spontaneous or senile gangrene results from obliterating endarteritis. *Thrombosis or embolism* may occur in the aorta, brain, or in the coronary, pulmonary, mesenteric, pancreaticoduodenal and other arteries.

10. The *general nutrition varies*. Some writers distinguish between (*a*) *a plethoric type*, which occurs in elderly individuals and the symptoms of which are good color, wide vessels, increased thirst, hemorrhages from the nose, increased urine, respiratory catarrh, profuse sweats, muscular rheumatism, headache, enlargement of the liver, emphysema, tense pulse, thick bloodvessels and marked hypertrophy of the heart; and (*b*) *an anæmic type*, with chlorotic habitus, anorexia, dyspepsia, few external vascular findings, dilated heart, and endarteritis. This type occurs in individuals between 30 and 50 years of age. French writers speak of a *cachexie arterielle*, in which the pallor is distinguished from the dusky anæmia of cardiac disease. Alfred Stengel has noted that the anæmia is more often apparent (pseudo-anæmia) than real.

Prognosis.—The prognosis is relatively unfavorable, because the symptoms are frequently latent until the process is well established or far advanced. In the early stages arteriosclerosis may be arrested or may remain stationary for a long period, but there is constant danger of apoplexy, angina pectoris, myocardial paralysis, "heart block," heart rupture, or uræmia. It is a most common finding after death from acute infections, so that many deaths attributed to acute affections are more properly deaths from arteriosclerosis. Increased blood pressure may overcome moderate obstruction. The prognosis is most favorable in angiosclerosis of the peripheral vessels, and, as a rule, is rather unfavorable in atheroma of the aorta, because of the danger of coronary disease and aneurysm. Intestinal hemorrhage is ominous. A slow pulse is less favorable than arrhythmia and moderate increase in rate. The plethoric form is less dangerous than the anæmic.

Treatment.—*Prophylaxis* regarding the etiological factors and the patient's mode of life is most important. Alcohol and tobacco should be forbidden, iodides should be given in syphilis, the diet should be regulated in diabetes, colchicum should be given in gout, etc. In early cases the process may sometimes be arrested. Many cases seem to improve on a milk diet, although Rumpf claims that many of the salts ingested are deposited in the arteries. He found that milk sufficient to meet the needs of metabolism (70℥ or 2,175 c.c.) for 24 hours contained over one dram (5 grams) of calcium and magnesium. *Iodide of potash* has been advised, especially by Huchard, who gives 1 to 15 to 45 grains daily for twenty days; then after ten days intermission, *nitroglycerine* is used. (See ANGINA PECTORIS AND TREATMENT OF SYPHILIS for details.) Iodides are also used in cases where no suspicion of syphilis exists. Some physicians oppose its use and find on experimentation that it has no vasodilator effects. Rosenbach claims that it actually irritates the endothelial tissues. In some cases at least it has beneficial effects and in a non-syphilitic series of personally observed cases the walls of the nodular and hard vessels distinctly improved with administration of iodides. Rhomberg thinks that iodides operate less as vasodilators than they do to decrease the viscosity of the blood. It has been recently claimed that nitroglycerine dilates the peripheral vessels only, but on this assumption it is hard to explain the cerebral flushing, etc., which follows its use. *Massage, a warm climate, passive exercise,* and the *Schott method* of treatment may be employed as outlined under DILATATION OF THE HEART. It is interesting to note that Skoda employed hot hand- and foot-baths to reduce peripheral resistance. Coffee may relieve anginal and abdominal pain.

Iron and digitalis not only are, as a rule, unavailing but also are directly injurious. When the heart action is weak, Huchard advises the following pill:

℞

Sodii iodidi	ʒj.
Sparteinæ sulphatis	gr. xv.
Pulv. glycyrrhizæ	q. s.

M. et ft. in pil. xl.
S.—One after meals.

Rest is more efficacious than are cardiants. Morphine is a valuable nervous sedative and tonic for severe cases of cardiac asthma, but must be used with great care.

ANEURYSM.

Definition.—A dilation, usually localized and sac-like, of an artery.

Etiology.—(*a*) *Arteriosclerosis* is the most important cause. The importance of syphilis is estimated at 29 per cent. (M. Schmidt), 36 per cent. (Fraenkel), 53 per cent. (Gerhardt), 80 per cent. (Malmsten), and 100 per cent. (Drummond). The author thinks the relationship between syphilis and aneurysm is as well established as that between syphilis and locomotor ataxia, and probably with the same percentage, for the majority of cases coming under his own clinical and autopsy observation give a clear history or show indubitable stigmata of syphilis. Sudden effort in strong men with a weakened aorta is a direct factor in partial rupture of the aorta and formation of aneurysm. (*b*) *Trauma* is an important factor but is probably overestimated. (*c*) *Sex:* aneurysm is from two to eight times as frequent in males as in females. (*d*) *Age:* the old statement that aneurysms occur before the fiftieth year and arteriosclerosis after that age is still valid; they occur between 30 and 40 (Crisp), 30 to 50 (Orth), 30 to 60 (Rokitansky), 40 to 50 (v. Schroetter); later compilations, however, set the age higher, 50 to 60 (Emmerich), 60 to 70 (Juda), and even after 70 (Barsdorff). It is said that but 60 cases under 20 years of age have occurred. (*e*) *Embolic and mycotic aneurysms* are usually small and may be multiple. Arterial tuberculosis was found by Weinberger. (*f*) The *traction aneurysm* (Thoma) at the ductus arteriosus Botalli is rare.

(*g*) The *arrosion aneurysm,* in which the inner coats bulge out when the adventitia is eroded, occurs largely in tuberculous cavities. The "aneurysmal diathesis" is spoken of by older writers; Pelletier found 63 in one case. Dieulafoy considers that multiple aneurysms of the arch are clearly syphilitic.

Pathology.—In relation to the lumen, aneurysms are *axial* or *peripheral.* According to Quain, the varieties are: (*a*) *true aneurysm,* in which the sac consists of one or more coats. Strictly speaking, a true aneurysm should contain all three arterial coats in its walls, but this is most uncommon. The sac is usually (i) fusiform and diffuse, or (ii) sacculated and circumscribed. (*b*) *False aneurysm,* in which all the coats rupture, or in which, according to some, one coat ruptures through the others, like a hernia. These are circumscribed or diffuse, especially in the transverse arch and in the abdominal aorta. (*c*) *Dissecting aneurysm,* wherein the intima of the vessel ruptures and the blood makes a channel down the vessel between the intima and media or between the layers of the latter. It is a partial rupture. The rupture back into the aorta is an attempt of nature toward recovery; the patient may live for years. (*d*) *Varicose* aneurysm, wherein the sac ruptures into a vein, especially the innominate vein or vena cava; this form is also called *arteriovenous* and aneurysmal varix, because the main change is in the vein.

Atheroma is held to be a common cause, though it occurs very frequently in comparison with aneurysm. It is thought that intimal changes induce aneurysm when the outer coats do not undergo vicarious thickening quickly enough to prevent dilatation. Koester held that mesarteritis was the principal change, but Eppinger found the greatest change to be loss of the elastic and muscle-tissue in the media. Syphilitic changes are discussed under SYPHILIS and are very important. In the sac thrombi form from the roughened intima and slowed circulation and to a small extent guard against extension and rupture. Aneurysm occurs at those points in the aorta on which the blood stream impacts most strongly; 59 per cent. of thoracic aneurysms occur on the anterior surface of the ascending aorta, 29 per cent. on the convexity of the arch and 12 per cent. on the posterior surface of the descending segment. In 915 aneurysms the seat was in the pulmonary artery in but 0.4 per cent. (Crisp).

Symptoms.—Bramwell distinguishes three classes of cases: (*a*) Those which are entirely *latent*, and become manifest only by rupture or at the autopsy. (*b*) Those presenting *symptoms* of *intrathoracic pressure* but giving no positive physical signs. (*c*) Those *signalized by distinct physical signs, e. g.*, by pulsating expansile tumor.

The Purely Subjective Evidences of Aneurysm.—The clinical signs are most variable, and often change or disappear entirely when the aneurysm changes the direction of its growth; the latency of a large proportion of aneurysms gives a disproportionate value to symptoms *per se*.

1. PAIN.—Pain is either intrinsic, resulting from subacute aortitis, varying with the arterial tension, relieved by reduction of the circulation, localized, and dull or aching in character; or it is extrinsic, resulting from pressure on nerve trunks especially in deep-seated tumors, or from the weight of the aneurysm, as illustrated by a case of Stokes in which pain was relieved by the use of crutches. Pain in addition to progressive enfeeblement and emaciation is of some diagnostic value. Sudden pain in the left chest, whether anginal or not, if it does not yield to treatment, is always suggestive of aneurysm. Pain in the majority of cases is the first, the most notable, and often the most enduring symptom. It is often alleviated by iodides; it is usually paroxysmal, sharp, lancinating, or boring in character (when, for instance, the spine is eroded); it is augmented by movement, but may cease when the part compressed is entirely eroded. The pain is local or is reflected into the brachial plexus, the arm, neck, or along the intercostal nerves and may cause numbness and paresis of the arm. Though it is more usual anteriorly, it is sometimes felt in the interscapular region. Anginal pain follows involvement of the cardiac plexus. Intercostal neuralgia is always suggestive of aneurysm. When there is tenderness, care is imperative lest palpation induce embolism.

2. DYSPNŒA.—Dyspnœa is frequently out of all proportion to the findings. It is often a vagus neurosis, but is also due to narrowing of the chest space by compression of the aneurysm upon the heart, phrenic nerve, trachea, bronchi, lungs, or pulmonary artery; or to compression of the plexus cardiacus or pulmonicus, producing bronchial spasm, and of the recurrent nerve, leading to laryngeal spasm or paralysis. Dyspnœa may occur with change of posture (Leube) and is paroxysmal; it is at times more or less urgent, or may sometimes subside entirely.

3. COUGH.—Cough is frequent, but is one of the least distinctive features. It results from pressure on the air passages, vagus irritation, bronchitis or laryngeal involvement. It is often unproductive. Like

all other eccentric pressure symptoms, it varies with the size and direction of the aneurysm; it is paroxysmal (Morgagni) and is prone to appear or disappear hand in hand with the pain and dyspnœa.

4. DIFFICULT SWALLOWING.—Dysphagia is relatively infrequent. It results from direct compression or from pressure on the recurrent laryngeal nerve, inducing œsophageal spasm. It occurs particularly in low aneurysms of the "recurrent type" (*v. i.* LARYNX) and may produce distinct pulsations in a water tube introduced into the œsophagus. One of the earliest aneurysms reported by Stokes was the size of an egg, and produced pain, dysphagia, and compression of the left bronchus, with rupture into the latter. Aneurysm may be confused with carcinoma of the œsophagus. Traube described a case of obstruction of the subclavian artery and compression of the left recurrent laryngeal nerve in which aneurysm was suspected; but the autopsy revealed carcinoma of the œsophagus embracing in its regional invasion the left subclavian artery and the recurrent laryngeal nerve. Dysphagia may be intermittent or present only when the patient is in one posture. Its infrequency is explained by the ready lateral luxation of the œsophagus; dysphagia is most often due to adhesions between the aneurysm and the œsophagus. The danger of sounding the œsophagus is well known, for distressing lethal hemorrhages have occurred in the physician's consultation room.

5. COMPRESSION OF THE VAGUS.—Vagus compression results in asthma, laryngeal spasm, vomiting, stenocardiac attacks, hiccough, and pulmonary symptoms.

The Physical Signs of Aneurysm.—1. INSPECTION.—Stokes described *two areas of pulsation* in the chest, each distinct "*like two separate hearts.*" This is most significant when the cardiac impulse is the weaker. Inspection is most advantageously made with the eye just below the level of the shoulder and near the chest. The typical *expansile pulsation* is best detected in superficial thin-walled aneurysmata in the ascending arch, or in those presenting in the jugulum. Pulsation is less a question of the size than of the location of an aneurysm, and therefore visible pulsation may be absent even in the largest varieties, for deeply situated aneurysms give diffuse or no pulsation, or the pulsation appears only during expiration. Throbbing over the aorta is least important when there is also marked arterial pulsation (aortic regurgitation). The *pulsation* must be *expansile* to render the diagnosis absolutely sure, though at autopsy the author saw one case which presented clinically an expanding throbbing aorta and dulness over the arch which was due to an enormous mass of callous connective tissue involving the pleura and pericardium. Another instance of a truly expansile pulsation low down in the left side of the chest as well as near the spine was one observed in pernicious anæmia; the hands which had been placed over it were lifted widely apart, and the pulsation was seen at a distance of forty feet; the autopsy revealed no aneurysm. Pulsation lent to a tumor, a collapsed lung, the liver, or a pleural exudate, can usually be distinguished without difficulty.

2. PALPATION.—Pulsation is felt slightly later than the heart shock; this interval is more obvious the slower the heart rate becomes. The *expansile character of the pulsation* is sometimes more easily detected by the fingers than by the eye. Palpation may reveal remittent tension of the sac, *tenderness* on deeper pressure, resistance caused by thick lamellated thrombi, and, in rare cases, elasticity or fluctuation of the tumor during the diastole. Pulsation in a deeply situated aneurysm is best elicited by placing one hand anteriorly and the other posteriorly on the chest. A *thrill* may be felt which is most commonly systolic. *A diastolic shock* sometimes occurs in the jugulum and over the aortic area (Lancisi), and this is not due to recoil from the aortic valves, but to retraction of the hypertrophic aorta lying between the heart and the aneurysm (Rosenbach). It is possibly due to the retraction of the vessel wall.

3. PERCUSSION.—*Dulness* (Walshe) is an important finding only when it is present on either side of the sternum, but not when confined to the sternum. Dulness may be detected in the left interscapular region, though in this case percussion is less valuable, because of the thickness of the chest walls. Aneurysms are often obscured by an emphysema, and they may grow from their deep origin into the lung substance. The dulness may so merge with that of the spine, heart, liver, or spleen, that no certain statement can be made. Dulness when once detected *may cease or shift* to another place, and this is an almost certain indication of aneurysm, though it may occur in cysts (Bramwell).

It is inexplicable that aneurysms often grow toward and press upon relatively unyielding structures, while the softer parts in their immediate vicinity, as the lungs, are less compressed (v. Schroetter).

4. AUSCULTATION.—Auscultation is sometimes wholly negative, even in large sacs—explained by thrombi in the sac, and weak heart action. *Two clear tones are most frequently heard;* the second tone, heard over a circumscribed area, is pathognomonic of aneurysm (v. Schroetter), and originates in the aneurysm itself; some writers attribute it to closure of the aortic cusps; it is most often heard in aneurysms near the ascending arch. Two tones in the abdominal aorta are scarcely ever heard normally. A *systolic murmur* is frequent, and is usually blowing, roaring, or scraping; it is due to vibrations in the aortic wall, to entrance or exit of blood from the aorta into the sac, to blood waves initiated by angles in the direction of the current, by thrombi, or more rarely by pressure of the sac upon the aorta itself.

The less frequent *diastolic murmur* is most probably caused by (relative) aortic insufficiency, when the aneurysm lies in the first part of the aorta. It may be due to folds in the aorta, vibrations of clots, dilatation of the left ventricle, or to dissection of the aneurysm into the sinus Valsalvæ (Stokes, Marey). It is usually combined with a systolic murmur. A continuous murmur is rare. Of 132 cases (Sansom) in which the diagnosis was made, only 12 presented a systolic murmur, 25 gave systolic and diastolic murmurs, and in six, diastolic murmurs alone were found. Douglas-Powell detected murmurs in about half of his cases of sacculated aneurysms.

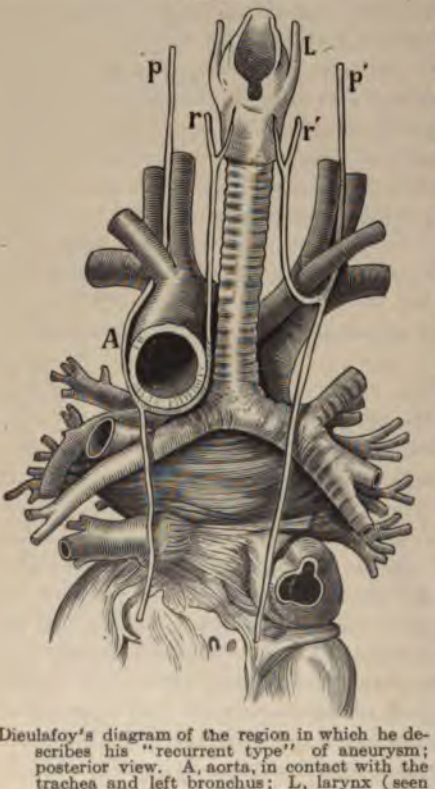
The Heart.—Its position varies with the size and location of the aneurysm. (*a*) Large aneurysms of the ascending aorta not only depress it, but cause such axial rotation that the left lies lower than the right ventricle; aneurysm in this location may cause atrophy of the right ventricle from pressure. (*b*) Aneurysm of the under surface of the arch may dislocate the heart to the median line. (*c*) Aneurysm of the descending segment may flatten the heart against the chest wall (Neusser), thus simulating mitral regurgitation. (*d*) Secondary aneurysmatic outgrowths from the chief aneurysm complicate the physical findings; these growths sometimes attain incredible proportions and produce confusing symptoms. (*e*) In uncomplicated cases, myocardial alteration is entirely accidental (Stokes, Key, and Skoda), which contradicts the usual statement that dilatation of the vascular system induces hypertrophy. Failure of the heart to hypertrophy is explained by anæmia, arteriosclerosis, coronary disease, or compression of the pulmonary artery, thereby decreasing the flow of blood to the left heart and coronary arteries. *Hypertrophy* of the left ventricle bears an intimate relation to aortic leakage; it may in rare cases result from aneurysm involving the entire periphery of the aorta. Stokes states that the most violent aneurysmal pulsation he ever witnessed occurred with marked myocardial atrophy. Compression of the pulmonary artery (early observed by Rindfleisch and Obermeier) may cause relative tricuspid regurgitation or pulmonary insufficiency (v. Schroetter).

The symptoms of aneurysm are given in their entirety in Stokes's early observations.

The Vessels.—The *pulsus differens* is very frequently physiological, due to slight irregularities in the size or branches of the radial vessels. When one radial is small, its fellow ulnar is often vicariously larger. Stokes explicitly stated that the right radial is normally larger than the left, whence if the right radial pulse is smaller, it means more than if the left were slightly smaller. A pulse may grow smaller and later return to its normal size; this is due to change in the size and the direction of the aneurysm. When the radials differ essentially in volume it is advisable to compare the brachials. Asymmetrical sphygmographic tracings may be due to other causes than aneurysm as atheroma, coagula, embolism, or reduction of the lumen of a vessel to a mere slit, perhaps by the dragging of the aneurysm or compression of the aorta or its branches by the sac itself; v. Schroetter instances a case in which the aorta was so compressed by a sac lying between it and the spine that abdominal pulsation and the femoral pulses were obliterated. Changes are observed most often in the carotid and subclavian vessels. The *pulsus paradoxus* and capillary pulsation may occur in large aneurysmatic sacs (Quincke). *Retardation* of the pulse occurs especially in fusiform aneurysms. The location of an aneurysm may sometimes be determined by regarding the vessels altered. If pressure is exerted upon the *superior cava*, dropsy of the upper portion of the body, of one or both arms, swelling of the face and of the tongue, sometimes exophthalmos and a "brawny" or "collar-like" distention of the neck occur. Obstruction above the azygos vein causes lividity and œdema which are confined to the head and arms; when it occurs below that vessel the chest is also congested. Obliteration of the innominate and azygos veins or the venæ cavæ may occur. *Rupture into the superior cava* is attended by cyanosis, and a continuous bruit like the *bruit de diable* is often present; in Ewart's case (not suspected during life) phlebotomy was performed, and bright-colored blood escaped in jets. The continuous murmur may also occur in cases of vascular sarcomata.

The Respiratory Tract.—*Tracheal tugging,* described by Oliver and Cardarelli simultaneously, is elicited by lifting the larynx with the fingers; a tugging sensation in the trachea is then felt with each systole; this symptom occurs in cases of cardiac overaction, left ventricle hypertrophy, aneurysmata of the arch and of the innominate and carotid vessels, and in adhesions to the trachea.

FIG. 30.



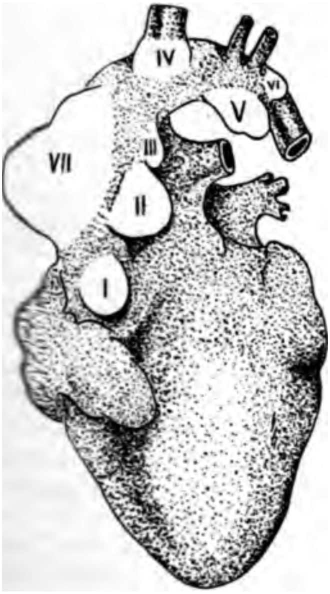
Dieulafoy's diagram of the region in which he describes his "recurrent type" of aneurysm; posterior view. A, aorta, in contact with the trachea and left bronchus; L, larynx (seen from behind); p, left and p′, right vagus; r, left recurrent laryngeal nerve, passing under the aorta, and r′, right recurrent, in relation to the subclavian artery.

Tracheal pulsation is present in many cases of aneurysm and may be demonstrated by the laryngoscope. The examination should be made by direct sunlight, and the observer should sit before the standing patient. Tracheal pulsation also occurs in tumors which compress the trachea; it may also be physiological. Drummond has described a systolic murmur over the trachea. A short murmur, synchronous with the systole, can sometimes be heard when the patient breathes quietly with the mouth open. Schroetter, Sansom, and others, describe it as "xschek," and refer it to propagation from the trachea.

Recurrent laryngeal paralysis was first described by Morgagni or by even earlier observers. Todd was the first to note laryngeal muscular atrophy on the affected side; paralysis may exist when the larynx is anatomically intact. Unilateral paralysis with the cadaveric position of the vocal cords, in the absence of obvious cause, is quite indicative of intrathoracic aneurysm or neoplasm, though it is also seen in cases of mitral stenosis. The cough is hoarse, and makes a clanging or brazen sound. It is a paralytic cough, *i. e.*, an imperfect expiration which lacks forcible closure of the glottis observed in ordinary coughing efforts; it was called the bovine cough by Wyllie from the fact that cattle have no false cords whose closure is an important mechanism in coughing. The right recurrent laryngeal nerve, curving around the subclavian artery, does not enter the thorax, and is therefore seldom involved; whereas the left recurrent nerve passes into the chest and curves between the aorta and left bronchus; Dieulafoy described a "recurrent type of aneurysm," which is usually small syphilitic in origin, often multiple, involves the left recurrent nerve, and causes spasm of the larynx. The spasm is due to recurrent laryngeal irritation which contracts the posterior transverse arytenoid muscle and induces dyspnœa and inspiratory stridor. Irritation of other vagus branches induces spasmodic dysphagia, anginal pain, etc.

LUNG SYMPTOMS.—Stenosis of the right bronchus is less frequent than that of the left, for the latter lies nearer the aortic arch. The lung moves less freely than normal; it suffers inspiratory retraction, and the breath sounds and fremitus are diminished. When a short note is elicited over an upper lobe (especially the left) it may cause suspicion of an aneurysmal compression with relaxation of the lung. It is common in the "recurrent type" of Dieulafoy.

FIG. 31.



Dieulafoy—Multiple syphilitic aneurysms.

Rokitansky concluded that large aneurysms are almost never associated with *pulmonary tuberculosis*, a view disproven by Stokes, Fuller, Jaccoud, Herard, Cornil, Hanot, and Kartz. Tuberculosis may result from (*a*) trophic change, (*b*) compression of the pulmonary artery, or (*c*) decreased lung excursion. It is found in 13 per cent. of the cases of aneurysm.

Bronchiectasis, *bronchorrhœa*, and suppuration of the lung, is well called "aneurysmal phthisis" by Osler. *Pulmonary retraction* is observed from compression, adhesive pleurisy or interstitial pneumonia. *Pleurisy with effusion* may complicate and entirely obscure an aneurysm. *Pulmonary gangrene* occurs from compression of the aortic nutrient artery of the lung or stagnation of mucus. Gangrene of the bronchial mucous membrane also occurs. Gangrene may produce subcutaneous emphysema. *Pulmonary hemorrhage* may occur from gross rupture into a large tube, from mere granulations in a compressed tube, or from an aneurysm slowly leaking into the parenchyma of the lung. The hemorrhage may be fatal, premonitory, or continuous; it may be seen as flecks in the sputum, as rusty sputum (but more abundant, more frothy, and less viscid than in pneumonia), as prune-juice expectoration with alveolar epithelium (as seen in valvular disease), and as discharges of pure blood or more frequently of imperfectly coagulated blood.

Sympathetic Symptoms.—The pupils are often involved; myosis results from sympathetic paralysis and mydriasis from sympathetic stimulation. It must be remembered that 7 per cent. of all persons have irregular pupils. Other sympathetic symptoms, as pallor or redness, and sweating, are infrequent.

Diagnosis.—The clinical signs and symptoms are most variable (Stokes). The presence of an aneurysm should be suspected when marked pain or paroxysmal dyspnœa occur. Variability of physical signs is suggestive of aneurysm.

Diagnosis of Location.—1. Aneurysm of the *ascending portion* is called by Broadbent the "aneurysm of physical signs." The usual signs are most often observed in the first interspace to the right of the sternum, which is often eroded. Luxation of the heart to the left, slowing of both radials, right recurrent paralysis, compression of the superior vena cava and of the pulmonary artery with hypertrophy of the right ventricle, dyspnœa and phthisis occur. Anginal or other cardiac symptoms frequently occur (Gairdner), and the aneurysm, though small, is usually rapidly fatal from rupture into the pericardium, pleura, upper cava, or heart-chambers. Aneurysms just above the valves tend to grow downward because of the reflux of blood from the aorta whereas those located higher grow upward, because of impact of blood from the left ventricle. Sympathetic involvement is common.

2. *In the arch,* the "aneurysm of symptoms" is observed with left recurrent paralysis or spasm, stridor, pulsation in the jugulum, unusual prominence or luxation of the sternal ends of the clavicles, prominence of the subclavian arteries, dulness to the left of the sternum, myosis, slowing or lessening of the left radial pulse, compression of veins particularly the left innominate, of the trachea, thoracic duct, sympathetic ganglia, bronchi and left upper lobe, and disturbance in the brain circulation (hemiplegia) when the carotid artery is occluded. These aneurysms usually grow backward.

3. In the *descending segment,* the "aneurysm of latency" is most often encountered. If there are symptoms, pulsation in the left interscapular region, slowing of the femoral pulse, pressure on the azygos or hemiazygos, pressure on the spine with erosion and tenderness to touch, intercostal neuralgia (sometimes with recurrent attacks of herpes zoster), stenosis of the left bronchus or œsophagus, pressure upon the heart forcing it against the sternum, and increased heart-rate from plexus pressure, are present. The x-rays alone may be able to detect aneurysms lodged in the lung substance.

Differentiation.—Differentiation from *solid tumors* is determined by their propagated non-expansile pulsation, their more rapid development, leukocytosis, and cachexia. In aneurysm the lymph glands are rarely enlarged either from lymph stasis or from inflammation; adenopathy is frequent in mediastinal tumor, and continued increase is directly indicative of neoplasm. Pain, recurrent paralysis and dysphagia are common to all tumors, both aneurysmatic and neoplastic. Venous ectasia is more common in tumor. A few rare instances of coincidence of tumor and aneurysm are observed. The pulsation in carcinoma is rarely associated with a diastolic "back stroke," which is frequent in aneurysm. An unequal pulse indicates aneurysm or atheroma. The bruit in tumor is usually systolic, but murmurs are frequently absent in aneurysm. Tugging is present in both but more common in the latter. Signs of aortic atheroma argue in favor of aneurysm. Aneurysm runs a much more protracted course than tumors. Exploratory puncture and diagnosis *ex juvantibus* with iodide are of differential importance.

Other causes for pulsation, such as a dynamic throbbing (aortic regurgitation), throbbing in acute aortitis, pulsation in an aorta dislocated from kyphosis, and pulsation in pneumonia (Graves) or in tumor of the lung or pleura, are usually differentiated when all symptoms and signs are carefully considered. In *pulsating empyema* the dulness is usually low in the chest and largely left-sided; puncture reveals the presence of pus and stops the pulsation.

Prognosis.—Recovery is rare, though spontaneous cure is possible by organization and calcification of thrombi in the sac or by its inflammation. Sometimes one aneurysm obliterates another. The course usually lasts more than a year. Patients have been known to live and work hard ten or twenty years. The author has watched for twelve years a blacksmith who has worked though he had an aneurysm of the ascending aorta, which was as large as his fist, and which protruded beyond the ribs. The size of the sac is of no prognostic value. Those of the small "recurrent type" are most dangerous, and large ones may be protected by connective tissue. Arteriosclerosis is prognostically unfavorable.

Mechanism of Death.—(*a*) *Rupture* occurs in 75 per cent. of cases: 20 per cent. into the left and 13 per cent. into the right pleura; 16 per cent. into the left bronchus; 12 per cent. into the pericardium; 9 per cent. into the left lung; 6 per cent. externally; 5 per cent. into the œsophagus; 4 per cent. into the trachea; (*b*) *suffocation;* (*c*) *marasmus,* especially in large aneurysms; (*d*) intercurrent disease; (*e*) embolism; and (*f*) brain lesions, are also immediate causes of death. Hemorrhage usually shortens the course, but in one case rupture into the bronchus occurred five years before death. Another patient suffered external rupture of the sac; he collected the blood in a basin and though he fainted from the hemorrhage he lived four months, only to die of typhus fever. Stokes described an instance of external rupture in which the presence of mind of the nurse, in stuffing

ing some cloth into the sac, kept the patient alive for a considerable time.

Treatment.—1. The *Tufnell treatment* of rest, low diet, and limited fluid is the result of the methods used by Morgagni, Albertini, Valsalva and Bellingham, without the venesection recommended by Valsalva. Absolute mental and physical rest is required for several months, and food should be reduced to the minimum. The heart action and blood pressure are greatly lessened. The treatment is of value only in cases of sacs with small necks. Active individuals will not tolerate this method. Relative quietness, and moderation in every phase of life must then be insisted upon. Ergotin, hypodermatically, has little effect. Tr. aconite ♏ i to iv may relieve the throbbing.

2. The *iodides* have been given prominence by Balfour, though they were used first by Bouillaud and later by Graves, Nélaton, and others. It is not clear how they operate. They are of equal value in syphilitic and non-syphilitic cases. They increase the secretions but do not inspissate the blood. It is questionable from the experimental standpoint whether arterial tension and dilatation of the sac are lessened. Possibly the sac wall is thickened or clotting is facilitated. However, it is certain that pain is alleviated. Ten to twenty grains are given at a dose as in arteriosclerosis. The dose is lessened if the pulse rate increases. Aside from relief of pain, the iodides are useless in large aneurysms with wide communications or in fusiform varieties.

3. *Cold* applications may relieve pain when the sac is near the surface. Supports may relieve the pain and protect against rupture. *Calcium chloride* in doses up to ten or fifteen grains has been used to promote coagulation, but apparently without effect.

4. *Gelatin injections* (1 or 2 per cent. solution) are recommended by Lancereaux to produce coagulation in the sac. It cannot be said that the results are convincing. Gelatin has been used for centuries in China and Japan to aid coagulation. There is danger of embolism and 23 deaths from tetanus are reported by Dieulafoy, 1903.

5. Electrolysis, *introduction of fine wire,* compression and surgical intervention have given discouraging results. A few cases of recovery are reported but one cannot help comparing their small number with the probably equal number of spontaneous recoveries (Boerger, in 1906, compiled 19 cases of dissecting aneurysms of the aorta which recovered). Fatal thrombosis may follow what in a lesser degree of thrombosis might have been beneficial. Total embolism of the aorta may also occur.

6. *Dyspnœa* is treated as in cardiac disease. Venesection may afford temporary relief, and morphine is finally indicated. Tracheotomy may be necessary in laryngeal spasm (reflex recurrent irritation) but the actual compression by the sac is usually too low for operative interference. Digitalis is at all times contra-indicated.

Abdominal Aneurysm.

This type constitutes 5 per cent. of aneurysms of the aorta. Over two-thirds occur in the upper part of the abdomen and 40 per cent. either at or above the cœliac axis. The average size is that of an orange and the tumor develops from the anterior surface of the vessel, less frequently from its lateral or posterior wall, and grows downward. Most of them are false aneurysms.

Symptoms and Signs.—Four per cent. of abdominal aneurysms are *latent* clinically; a good sized sac may be hidden under the arch of the diaphragm. *Pain* is usual, and is constant and boring, or paroxysmal. It may become less severe after hemorrhage from rupture. Relief is sometimes obtained by lying on the stomach. Indefinite *symptoms* are common in the *stomach* though they are less frequent than in the *duodenum,* where obstruction and hemorrhage may occur. *Inspection* does not show *expansile pulsation* and *tumor* in all cases. *Palpation* brings out the expansile pulsation and in some cases a *thrill. Percussion* may reveal *dulness* but the close relations to the liver, spine, kidneys and heart are prone to give indefinite findings. The diaphragm bounds the aneurysm above; the kidney to the left; the liver, pancreas and kidney to the right; and the stomach, pancreas and colon in front. The tissues about the sac thicken and produce adhesions. On *auscultation,* a *systolic murmur* (or two tones) is common and may reach the spine or loins; a *diastolic murmur* is sometimes heard. Rupture, diffusion by dissection, great thickening of the sac, and thrombi formation may easily obscure all auscultatory signs.

Diagnosis.—1. Compression Symptoms.—Compression symptoms, gastric and duodenal, have been mentioned. Abdominal aneurysm may be mistaken for gastric ulcer or cancer. The spine is eroded in 10 per cent. of cases; lumbar neuralgia, lumbago muscular rheumatism, myelitis, Pott's disease, and spinal tumor may be simulated. Pulsation should be searched for along the spine. Symptoms in the renal region may suggest renal colic, kidney tumor, perinephritis or psoas abscess (fluctuating blood from rupture). The ureter may be compressed. Pressure on the splenic vein may produce much splenic enlargement. The heart is rarely compressed and dyspnœa is uncommon. Retardation or absence of the femoral pulse may be of value in differentiation.

2. Abdominal Pulsation.—"Instead of being your first, it should be your last idea that abdominal pulsation is due to aneurysm" (Jenner). *Epigastric pulsation* may be confused with hypertrophy of the right heart or pulsation of the liver. The greatest danger of error lies in diagnosticating as aneurysm the pulsation which is so often seen in acute hemorrhage, or especially in neurasthenic subjects, who complain of pain and throbbing and in whom thrills, systolic and, according to Rosenbach, even diastolic murmurs may be felt and heard, especiallv when atheroma is developing. Rosenbach especially emphasizes the diagnostic importance of "intermittent dilatation of the aorta" which is frequently diagnosticated as aneurysm. The pulsation must be *distinctly expansile* (even then occasional errors are made) and a definite sac must be felt. Otherwise constant errors will be made. The *dynamic* pulsation of aortic regurgitation concerns the same points.

3. Communicated Throbbing.—Communicated throbbing, lent to a tumor of the stomach lying on the aorta, may also be attended by systolic murmur but the differentiation is usually made with ease, except in certain soft neoplasms. Tumors are often felt to better advantage when the subject is in the genupectoral attitude; they are more movable, more often produce dilated veins, ascites, and peritoneal friction. Aneurysm of the abdominal aorta or of its branches may, in exceptional cases, be movable. Stokes held that tumors developed upward and aneurysms downward.

4. Aneurysm of the Branches of the Abdominal Aorta.—(*a*) Aneurysms of the *cœliac axis* are difficult or impossible to distinguish and may occur with aneurysm of the main trunk. More than 20 cases are recorded. They are often traumatic or embolic (mycotic). (*b*) *Superior* (20 cases, Baccelli, 1904) *or inferior* mesenteric aneurysms are very uncommon; they occur most frequently in children, from sepsis. (*c*) Over 20 cases of *splenic* aneurysm are recorded. The spleen is often enlarged. (*d*) In the *hepatic artery* (35 cases, Grunert 1903) it is induced by acute infections (73 per cent.), trauma, embolism, or gallstones; 41 per cent. develop in the main trunk and 25 per cent. in its right branch; there is but one case in which it developed in the artery of the cystic duct. Pain, vomiting, hemorrhage, icterus, and large spleen have been noted: in 79 per cent. rupture occurred (in 10 cases into the peritoneum); they never pulsate, and in only one case was there a systolic murmur; Kehr operated on one case successfully. (*e*) Over 10 cases of aneurysm of the coronary artery of the *stomach* are recorded. (*f*) In the *renal* artery (26 cases, Skillern, 1906) aneurysm is rare. Three cases were successfully operated on. Von Schroetter found only 1 case in nearly 20,000 autopsies.

Prognosis.—Death is usual and the clinical course averages one to three years. *Rupture* occurs in 66 to 75 per cent. of cases: 33 per cent. into the peritoneum; 16 per cent. into the pleura, though the crura of the diaphragm offers considerable resistance; 7 per cent. into the duodenum; and 7 per cent. into the retroperitoneal tissue. Death does not necessarily nor at once follow rupture even into the peritoneum.

Treatment.—Treatment is as in the thoracic type. From its accessibility, operative procedures are more common and somewhat more successful. Continued compression of the aorta has benefited cases of aneurysm located low in the abdomen

ACUTE AORTITIS AND ARTERITIS.

These are rare lesions, occurring in the aorta as part of a sepsis, benignant or malignant endocarditis, or in the peripheral vessels after influenza or other infections. *Acute aortitis* was often confused with chronic atheroma, acute arteritis, and with organizing thrombi in the arteries. Syphilis (*q. v.*) is a factor in aortitis, of which the symptoms are retrosternal burning, actual anginal pain, dyspnœa, anxiety, and acute dilatation of the arch without violent throbbing of its branches. The diagnosis is uncertain and the treatment symptomatic.

Periarteritis nodosa was first described by Kussmaul and Maier and is called "arteritis proliferans nodosa" by Fletcher. This is a rare affection of which Versé in 1905 could find but 20 published cases; it consists of localized proliferation and round cell infiltration of the intima with its rupture through the other coats, and leads to thrombosis or aneurysm. It is probably an infection. It occurs largely in men between twenty and thirty-five years of age; one case occurred in an infant three months old. The nodes are seen in the vessels of the muscles, heart, kidney, intestines, and skin; they produce hydrops, nephritis, dyspepsia, alimentary hemorrhage, epigastric pain, low temperature, and a rapid pulse. The affection is fatal within six to twelve weeks, and treatment is purely symptomatic. In one-quarter of the cases there were syphilitic antecedents and Schmorl saw one cure with the use of mercury.

RUPTURE OF THE AORTA.

Rupture of the aorta may be traumatic or the result of severe strain, endarteritis, or narrowing of the vessel. Tolot and Sarvonat (1904) collected but six cases of rupture of a sound aorta. All coats may be ruptured and this usually occurs in a transverse direction. If the rupture is partial, a dissecting aneurysm results which may burrow even to the popliteal arteries. It most often occurs in the first segment whence hæmopericardium results. Rupture into the lung, mediastinum or retroperitoneal connective tissue may occur. The symptoms are sudden pain, a sense of "something ruptured," and the signs of hemorrhage; sudden death usually occurs, though prolongation of life for from one to eleven years has been recorded in cases of dissecting aneurysm.

Embolism of the aorta has a close relation to thrombosis, being due to impaction of atheromatous plaques, tumor tissue, echinococcus cysts, endocarditic plugs or ordinary thrombi. It is very uncommon. Sudden death results if the site is in the first part of the vessel. If it occurs near the division of the iliacs, collateral circulation is possible. There are pain and stiffness in the legs, sensory disturbances, paresis and contractures from anæmia of the spinal cord. The femoral pulsation is absent. The clot may back up toward the heart resorb. The bladder and rectum are disturbed. Hemorr the stomach, bowels and kidneys has been observed. The is bad, and the treatment is symptomatic.

Thrombosis usually results from several coincident cause infections, weak heart, aneurysm, atheroma, or compressi cases occur in the transverse arch between the left carotic clavian vessels or in the abdominal aorta at its point of divi pain, paraplegia, or anæsthesia produced are sometimes con spinal cord disease. It may be sudden; if gradual, coll culation may be established.

SECTION III.

DISEASES OF THE RESPIRATORY TRACT.

DISEASES OF THE NOSE.

(A). ACUTE RHINITIS, CORYZA.

Definition.—An acute, probably infective nasal inflammation.

Etiology.—(*a*) It is an *independent infection*, sometimes epidemic and popularly held as contagious; it occurs oftenest in the spring and fall and is promoted by variations in temperature. Cold is probably only a predisposing factor; various bacteria are held causative, but Thompson and Hewlitt have shown that 84 per cent. of cultures are negative. (*b*) It is a *concomitant* of various *infections*, notably of influenza, measles, pertussis, glanders, syphilis, etc. (*c*) *Toxic factors*, as iodides, bromides, ipecac dust, digitalin, dust, chemicals, etc., may cause coryza. (*d*) *Local disease*, as chronic catarrh, polyps, ulceration, etc., and possibly gout, are predisposing factors.

Symptoms.—The initial symptoms are malaise, depression, anorexia chilliness, fever of 101° to 102°, pains in the limbs, and sometimes, in the young, delirium and convulsions. The mucosa is dry, reddened, burning, painful, swollen and obstructed. There is repeated sneezing and anosmia. Herpes is common. There follows a clear, watery, nasal discharge (*coryza*) for one or two days, which subsides at night and recurs in the morning; within two days it becomes thickened and purulent. Extension may occur to the eyes, frontal sinus, antrum of Highmore, ear by the Eustachian tube, or to the pharynx or larynx. In infants nasal occlusion prevents nursing and may cause dyspnœa or suffocation, because the tongue lies high in the mouth near the palate. Acute rhinitis lasts one-half to one week and is seldom confused except with incipient measles.

Treatment.—At the very onset lavage with simple hot water is often sufficient; Dobell's solution (sod. borat and sod. bicarb. āā ʒij. ac. carbolici ʒss to water ʒx), diluted freely to avoid irritation, is beneficial; adrenalin solution 1 to 1,000 is excellent (excessive application of adrenalin may induce œdema of the throat and epiglottis). A cocaine solution (the plain alkaloid gr. j to albolene ℥j) relieves turgescence and frontal pain, but is a dangerous remedy to give into the patient's hands. Dover's powder gr. x, a saline aperient and a hot bath at the onset, usually give relief, specially when followed by:

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Extr. belladonnæ	gr. iiss.
Fluidextr. aconiti	♏x.
Phenacetini	gr. xv.
Camphoræ monobrom.	gr. xv.

M. et ft. capsulæ x.
S.—One capsule every hour for five or six doses.

Drugs are often of little use unless the patient remains in bed to relieve internal congestion and to avoid extension or recurrence. Salicylates mitigate the frontal pain. Cool baths or rubs aid in averting recurrent attacks.

(B). HAY FEVER.

Catarrhus æstivus, or summer catarrh, was first described by Bostock (1819). Morill Wyman first described the "June" or "rose cold."

Etiology.—Its etiology is not known, but the disease has long been associated with the pollen of various plants and grains. Dunbar believes that it is toxic, resulting from albumoses in the pollen. It prevails especially in England and the United States, develops in persons between fifteen and thirty years of age, and occurs twice as frequently in males as in females. It is more common in the upper educated classes, and perhaps in gouty individuals; it is most prevalent in the warm months, and in cities. Three special factors are noted: (*a*) A *predisposition*, either in the family in the form of nervous instability or other systemic tendency. (*b*) An *irritability* or other nasal abnormality, as undue nasal turgescence, or irritability to dust, smells, etc., hyperæsthesia of the inferior turbinates, polyps, or septal irregularities; Austin Flint became asthmatic from sleeping on feather pillows. (*c*) The *exciting cause*, or irritant, as pollen, dust, sudden change of temperature, and apparently in some cases mental excitement. Streptococci have been sometimes found in the nasal secretion.

Symptoms.—The onset is that of the vulgar coryza, but the disease is attended by more constitutional reaction and greater *depression*. *Injection* of the *eyes* with lachrymation and photophobia usually accompanies or precedes the coryza. The throat, larynx, and bronchi are dry and burning. Moderate fever, 100° to 101°, is fairly common. There is pain over the frontal sinuses and sometimes over the temporal and occipital regions. The nasal vessels are congested, and the *nose* itself becomes *swollen* and injected, resembling closely the alcoholic facies. In many cases *cough* develops; this is frequently followed by *asthmatic* seizures which simulate bronchial asthma. Recurrences are usual and attacks of coryza may alternate with asthmatic paroxysms. With the first frost the affection stops, as if by magic. The outlook is good as to life, for only isolated instances of death from asthma are recorded; as to recovery, the outlook is much less favorable.

Treatment.—(*a*) Prophylactic treatment; cauterization of irritable, congested or hypertrophic areas, and correction of polypoid or other abnormalities cure some cases and alleviate suffering in others. (*b*) *Nasal* treatment; much relief may be given, when curative measures fail, by nasal douches, by painting or irrigating the nose with 1 to 1,000 adrenalin and astringent solutions; suprarenal extract in doses of gr. ij to v has been recently used internally, as has nearly every drug in the Pharmacopœia. (*c*) The *general physiological resistance* is increased by fresh air, by cool rubs, and by tonics and nervines. (*d*) *Change of climate* is advisable before the date on which, with certain-regularity, hay fever appears; a sojourn at the sea-side, in Northern Michigan, in Canada and especially in a high altitude, often greatly relieves or sometimes wholly checks the symptoms. (*e*) *Asthma* (*v.* BRONCHIAL ASTHMA). (*f*) *Dunbar's serum*, patented as pollantin, is used in the nose (not hypodermatically) every morning, as a powder. Lubbert (1904) in 505 cases claims that 59 per cent. recovered, 28 per cent. were benefited, and 13 per cent. were unaffected.

(C). CHRONIC RHINITIS.

Chronic nasal catarrh properly comes under the care of the specialist, but minor cases are treated by the practitioner; some are due to scrofula, syphilis, anæmia or to other constitutional causes. Chronic catarrh may develop from repeated acute attacks, or may be chronic from the onset.

The Hypertrophic Form.—The nasal mucosa is red, swollen, spongy, and is covered with thick, gray-yellow secretion which may dessicate and sometimes form the basis of rhinoliths. Microscopically, the bloodvessels are wide and are surrounded by round cells, and the submucosa is thickened. The nasal obstruction necessitates oral breathing, partially nasal speech, obtunded taste and smell, and produces an elevation of the hard palate and a peculiar facial expression. The secretion is usually thick, yellow and purulent, rarely thin and abundant (rhinorrhœa). This type is often associated with adenoids and follicular hyperplasia in the pharynx. The lachrymal duct may be obstructed, and Eustachian occlusion may induce permanent deafness; laryngitis, bronchitis, bronchial asthma, polyps, and sinus disease are not infrequent complications. Nasal disease may be the cause of blepharospasm or other facial spasms, limitation of the field of vision, vertigo, and mental depression; and even psychoses are attributed to nasal reflexes. Fliess attributes many dysmenorrhœas to chronic rhinitis. In its treatment possible constitutional factors, as anæmia, syphilis or scrofula, are to be considered. Douches of carbolic acid, insufflations of equal parts of alum and calomel, and cauterization by the galvanic cautery or by chromic acid, are indicated. Douches should be given under low pressure, lest fluid reach the middle ear.

The Atrophic Form.—It is disputed whether this form begins as an atrophic process or results from hypertrophic disease. It is more frequent in females and usually begins early in life. The mucosa is pale and dry, its epithelium is lost or has suffered marked metaplasia, the erectile tissue wastes, the nasal passages are unduly roomy and there is but little secretion, which dries in the nose and emits a most offensive odor (*ozæna*). Its actual cause is not determined. Various of the common microörganisms have been found, but the Bacillus fœtidus, Bacillus mucosus and Bacillus fluorescens liquefaciens are believed to be causative factors. Grosheintz and Meiser have noted its association with a widening of the face in 33 per cent. of cases. Its sequences are similar to those of the hypertrophic variety.

Treatment.—The results of treatment are not satisfactory. Douches of a 1 per cent. permanganate of potash solution, iodoform insufflations, massage with iodine one part, potas. iodide 10, and glycerine 100 parts, and packing with gauze soaked in balsam of Peru are usually employed. Paraffin injections beneath the mucous membrane have lately been recommended to restore the normal lumen of the nasal passages.

(D). RHINITIS FIBRINOSA.

Rhinitis fibrinosa has been spoken of in connection with nasal diphtheria, to which it is most often due. It may result from streptococcus and staphylococcus infection; it is observed in measles, scarlatina, pneumonia, and typhoid, and it also occurs as an independent lesion. Irrigation with 1 per cent. carbolic acid or 1 to 1,000 sublimate solution is usually efficacious. Antitoxin is indicated in infection by the Klebs-Loeffler bacillus.

(E). EPISTAXIS.

Epistaxis is a symptom only. Its causes may be grouped as follows (*a*) *Local nasal* lesions, trauma, chronic and acute catarrh, picking of the nose, foreign bodies, ulcerations (simple, tuberculous, syphilitic carcinomatous), and angioma. Osler reports cases of epistaxis associated with telangiectasis of the skin and nasal mucosa. (*b*) *Hyperæmia*, either venous as in cardiac, respiratory and other obstructive lesions, or arterial as in hypertrophy of the left ventricle caused by aortic leakage, and as in interstitial nephritis, atheroma, apoplectic habitus, and gout. Some cases are seemingly vicarious to suppressed menstruation or hemorrhoidal bleeding. (*c*) *Acute infections*, principally typhoid, less frequently the others. (*d*) *Hemorrhagic diseases*, as pernicious anæmia, chlorosis, leukæmia, pseudoleukæmia, scurvy, hæmophilia, Werlhof's disease and cachexiæ. (*e*) Other conditions, as liver cirrhosis, pregnancy, icterus, diabetes, weakly constitution, and puberty; baloon ascensions or high altitudes are also causative factors.

The most frequent point of hemorrhage is the lower anterior part of the cartilaginous septum. The symptoms are chiefly the local hemorrhage from the anterior nares, but occasionally bleeding may occur further back in the nose, and, trickling into the pharynx, may simulate hemorrhage from the stomach when vomited, from the intestine when passed with the stool, or from the lung when coughed up.

Occasionally urgent acute anæmia or even death may occur, especially when there is "blood dissolution" as in blood diseases.

Treatment.—The patient should be kept quiet, in the semi-erect posture. After douching the nose with hot or ice-cold water to which adrenalin 1 to 1,000 or some tr. ferri chloridi has been added, the nose should be tamponed by Bellocq's catheter, or in less serious cases the patient should be enjoined not to blow the nose, for this only dislodges the forming clot. Gelatin may be injected into the nose, besides being injected hypodermatically. Internal remedies, as ergotin, are very unsatisfactory. Erosions or ulcers may later be cauterized.

DISEASES OF THE LARYNX.

(A). ACUTE CATARRHAL LARYNGITIS.

Etiology.—Though acute catarrhal laryngitis is observed at every age and in all climates, most cases occur in males between twenty and forty, and in cold, windy, changeable weather. (*a*) *Cold* is frequently an apparent factor, but probably operates only by lessening the resistance, and this occurs to a greater extent in delicate than in robust subjects; the bacteriology of these "rheumatic" cases is unknown but its occasionally epidemic prevalence is suggestive of an infection. (*b*) Acute laryngitis may complicate *acute* (or chronic) *infections* such as measles, scarlatina, pertussis, typhoid, hay fever or syphilis, tuberculosis, and lupus. (*c*) *Extension* may be caused *per contiguitatem* from the nose, pharynx or bronchi; oral breathing is a somewhat predisposing factor. (*d*) *Chemical,* thermal, or mechanical agents, as dust of all kinds, foreign bodies, trauma, overuse or abuse of the voice, alcoholism, inhalation of tobacco smoke, chlorine or bromine, and internal use of iodides are etiological factors. (*e*) *Stasis* from thoracic lesions or enlarged thyroid, local ulcerations in the larynx, nephritis, liver disease, chlorosis, and rhachitis are also causes.

Symptoms.—A sudden onset, with fever, chill, rapid pulse or dyspnœa is seldom observed. The local symptoms are a sense of *rawness* or tickling, dry, even spasmodic cough, and moderate pain on breathing, as the larynx is hypersensitive; sometimes external tenderness or some dysphagia may occur, usually from coincident pharyngitis. After a day or so a thin, transparent, and slightly cellular sputum appears, which later becomes purulent, yellow or in very rare cases, hemorrhagic. Some cases void no sputum (laryngitis sicca). The voice becomes *hoarse* or is wholly lost. The laryngoscope reveals diffuse or circumscribed redness, viscid mucus, some swelling, and occasionally epithelial erosions. The false cords are often swollen. The true cords lose their smooth, glistening appearance and become injected and uneven, so that their even coaptation is hindered. The swollen mucosa may get between the cords, and their infiltrated muscles are often paretic. The acute symptoms last for a day or two and the catarrh remains about a week longer.

Diagnosis.—Diagnosis is usually easy; nervous aphonia and cordal paralysis are distinguished by means of the mirror (see VAGUS DISEASE, under NERVOUS SYSTEM).

Treatment.—Prophylaxis covers the etiological factors; alcohol and tobacco should be forbidden. The general resistance is increased by cold rubbings, douches or baths, and exposure, locally, of the neck; mufflers should be avoided and cold water should be applied over the larynx. The voice must be used carefully and the nose, uvula and pharynx should be examined for predisposing lesions.

The therapy *of the established lesion* is as follows: At the onset sweating should be induced by hot drinks and by a hot bath; Dover's powder and quinine, each gr. x, or aconite, are indicated as in coryza; rest in bed is necessary. The voice must not be used. If the symptoms persist, an ice-bag should be applied locally; codeine or morphine with bromides, and inhalations of moist air or steam should be given, as described under ACUTE BRONCHITIS.

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Heroin hydrochloratis	gr. j.
Terpene hydratis	gr. xl.
Ammonii chloridi	ʒss.
Extr. glycyrrhizæ	ʒj.

M. et ft. capsulæ xv.
S.—One after meals.

Painting the larynx with a 3 per cent. silver nitrate solution is recommended by some physicians and is condemned by others as promoting submucous extension.

In children acute laryngitis is often attended by great swelling above and below the cords and by stenotic manifestations in the small, infantile larynx. The symptoms closely approach those of genuine croup (laryngeal diphtheria), from which it is distinguished as false croup, *faux croup* (Guersant) or *laryngite striduleuse* (Bretonneau). After an attack of mild laryngitis, coryza or sore throat, the child becomes restless and anxious, usually during the night when the secretion accumulates. Inspiratory dyspnœa develops, there is inspiratory retraction of the interspaces and epigastrium, the accessory muscles of respiration come into play, cyanosis appears and asphyxia seems to be imminent. The paroxysm subsides, spontaneously or after an emetic has been given, but it occurs again on the next two or three nights. The catarrhal symptoms may last from three days to a week. Trousseau remarked that there is rarely the complete aphonia which is observed in laryngeal diphtheria. The *diagnosis* is made from the history of coryza; the absence of diphtheritic patches in the pharynx, for diphtheria is eminently a descending infection; the absence of diphtheria bacilli, found in diphtheria even when the throat seems normal; and the absence of severe constitutional reaction. *Laryngismus stridulus* (*v.* VAGUS NERVE) is not attended by fever, laryngitis, or aphonia. The laryngoscope may reveal the simple œdematous laryngitis, but examination is notoriously difficult in easily frightened children. Treatment is the same as in acute laryngitis of adults; hot drinks, inhalation of steaming water and ipecac should be given to induce vomiting and evacuation of laryngeal secretion; von Niemeyer awakened the patient at intervals and gave a hot drink to evacuate the mucus. Widerhofer also noted excellent results from washing away any pharyngeal mucus, which he considers causative of the attacks. The attendant should be prepared, in doubtful cases, for intubation or tracheotomy.

Other acute forms are (*a*) *laryngitis acuta nodosa*, which is observed in acute rheumatism and which yields to salicylates. (*b*) *Laryngitis fibrinosa*, which is usually diphtheria, but which may be caused by a streptococcus infection in measles, scarlatina or other infections, and which sometimes results from inhalations of steam, or chlorine. (*c*) *Laryngitis submucosa acuta* invades the submucous tissues and is seropurulent. This rare affection may develop as an independent lesion, may follow a similar process in the pharynx, or may represent a septic localization. During invasion the symptoms are severe, with fever, rigor, delirium and other septic phenomena. The laryngoscope shows injection and yellow-white bulging areas of submucous suppuration which, without operation, almost invariably cause death by œdema of the larynx or by sepsis, for spontaneous rupture is exceptional. For treatment (*a*) an ice-bag should be placed over the larynx, morphine should be given for pain, and whisky for the sepsis; (*b*) incision of the foci is indicated; and (*c*) if necessary, intubation or tracheotomy should be performed.

(B). CHRONIC CATARRHAL LARYNGITIS.

Etiology.—The etiology of this type is practically identical with at of the acute form, of which it frequently follows repeated attacks; t is often associated with or is dependent on pharyngitis or rhinitis.

Symptoms.—The symptoms are those of the acute form, though less intense. *Hoarseness* alternates with fairly normal phonation. By the mirror there may be seen *injection*, darker than in the acute form, *swelling*, secretion, and sometimes stains from small hemorrhages. There are forms with no secretion (*laryngitis sicca chronica*); the secretion is rarely like that of ozæna, with glandular atrophy. In some forms granules appear, less from glandular hyperplasia than from proliferation of connective tissue (*laryngitis granulosa*). Erosions and rhagades may develop on the edges of the cords, or arytenoid cartilages. Ulcers are most often syphilitic or tuberculous.

Treatment.—Treatment of the cause and prophylactic measures are the same as in the acute form. Inhalations of 1 per cent. tannic acid or of 1 per cent. turpentine and sod. bicarbonate in solution; insufflations of tannic acid, lead acetate and alum āā gr. i; and according to some physicians, painting the larynx every third day with a 2 to 10 per cent. solution of silver nitrate with the aid of the laryngoscope, are valuable. Terpine hydrate gr. iij t. i. d. is often helpful. Cold water applied over the larynx, faradization, compressed air, and sea air may be beneficial.

Other Chronic Forms.—*Chorditis tuberosa* occurs as small grayish nodes on the cordal edges at the junction of their anterior and middle thirds, especially in singers and in alcoholics (Stoerk); they are composed of fibrous and epithelial hypertrophy. Scarification and lacti acid are indicated (*v.* TUBERCULOSIS).

Pachydermia laryngis (Virchow) is an increase of the submuco tissue and transformed epithelium into which connective tissue grow it is circumscribed or diffuse. *Warty excrescences* are common betwe the arytenoids, and the thick mucosa is sometimes umbilicated a may resemble cancer. Topical application of salicylic or lactic ac iodine in glycerine, electrolysis and the cautery are indicated. *Pol* may result from chronic laryngitis.

Laryngitis hypertrophica inferior is a hyperplasia of the muc and submucous tissue below the cords, and is identified by s clinicians with nasal rhinoscleroma, which is due to the extensio the rhinoscleroma bacillus from above downward. It may ca stenosis of the larynx, which the author saw in one of Kolisko's au sies. It is treated by scarification, dilitation, prolonged intubati or, when extreme, by tracheotomy.

(C). ŒDEMA OF THE LARYNX.

Œdema laryngis is a more proper designation than œdema glottidis, as the vocal cords are very seldom involved. It is most marked where the submucous tissue abounds and is greatest therefore about the epiglottis and ary-epiglottidean folds; it may develop under the cords. It varies with its cause, and may be bilateral, unilateral, diffuse or circumscribed. According to Sestier, 70 per cent. of cases occur in males and 89 per cent. in those over fifteen years.

Etiology.—There are *three etiological forms* besides some obscure cases. (*a*) *Inflammatory œdema* results from various inflammatory diseases of the larynx, submucous suppuration, or perichondritis; from typhoid, syphilitic, tuberculous or cancerous ulceration; from iodides; from trauma, surgical measures, thermic, chemical and allied causes; from extension from pharyngitis, parotitis, or cervical cellulitis; and from infections, as typhoid or measles. (*b*) *Congestive and hydræmic forms* result from chronic heart and lung disease, from tumors and nephritis, in which it may antedate œdema elsewhere, or from cachectic conditions as carcinosis or amyloidosis. (*c*) *Angioneurotic œdema*, as in R. S. Morris's fatal case, is the last type.

Symptoms.—Symptoms appear with varying acuity, sometimes most suddenly, as in Bright's disease, at other times more gradually but progressively, and again quite slowly. *Hoarseness* or aphonia, and a *metallic cough* are common. The chief sign is *inspiratory dyspnœa*, which characterizes nearly all forms of laryngeal stenosis. It is usually stridulous or wheezing; and is attended by retraction of the interspaces, cyanosis, up-and-down excursion of the larynx and ultimate suffocation. The mirror shows a pallid swelling in the parts enumerated, incision of which allows the escape of a clear serum. Œdema of the epiglottis and ary-epiglottidean folds may be felt with the finger if the mouth is held open and the tongue drawn forward, or it may be seen even without the mirror if the larynx is pushed up and the tongue deeply depressed.

Treatment.—Treatment is urgent, irrespective of the cause, for the large mortality (75 per cent.) is due to delay until the patient's strength is exhausted. The placing of ice over the larynx, the swallowing of ice, drastic purgatives and painting the larynx with 10 per cent. silver solution are at best palliative. The œdematous tissue should be fully incised, and, in case this should fail, intubation or tracheotomy should be performed.

(D). PERICHONDRITIS LARYNGIS.

Purulent inflammation beneath the perichondrium of the cartilages of the larynx is very seldom primary, but nearly always results *secondarily* to various inflammations or ulcerations. Pus accumulates between the perichondrium and cartilage, most frequently the arytenoid or cricoid, or less frequently the thyroid or epiglottic. It may burrow; the cartilage may become secondarily necrosed. Perichondritis laryngis most often develops in males between twenty and forty years of age.

Symptoms.—Its symptoms are commonly obscured by the causal disease. The rare primary cases are the most acute. Well localized pain and tenderness are peculiarly significant. *Dysphagia*, especially from arytenoid involvement, is frequent, and even aphagia may result. *Phonation is impaired* by the fundamental disease, by cord paresis and muscular infiltration. The cervical glands sometimes tumefy. *Stenosis* and *dyspnœa* are present. The *mirror* discloses focal accumulation of pus, swelling, redness, œdema and perhaps fistulæ which signify intralaryngeal rupture. The probe may elicit tenderness or may scrape on eroded cartilage. The *diagnosis* from laryngitis and œdema is often impossible, or at best but probable unless definite pus pockets are seen. The *outcome* may be suddenly fatal from rapid œdema; pus and cartilage fragments may rupture, thus giving relief or perhaps occluding the larynx; fistulæ, internal or external, may remain, and may burrow to the mediastinum, or sepsis may follow; aspiration pneumonia is not uncommon, and if immediate recovery ensues, a distorted or stenosed larynx may remain.

Treatment.—Treatment is similar to that of acute laryngitis; the pain should be relieved by opiates, but early incision under cocaine is necessary, and the physician should be prepared to perform tracheotomy or intubation.

(E). ULCERATIONS AND NEOPLASMS OF THE LARYNX.

The tuberculous and syphilitic ulcers have been considered. Superficial erosions may follow catarrh; deep ulcers result from submucous suppuration or from perichondritis, pressure ulcers from foreign bodies or laryngeal "bed sores" (*v.* TYPHOID); some clinicians consider the latter to be an ulceration analogous to intestinal ulceration. There are also the specific ulcerations of leprosy, glanders, etc.

Tumors include benign single or multiple papillomata located on the true cords (singer's nodes *v.s.*), fibromata, cysts, amyloid accretions, enchondromata, lipomata, and the malignant tumors, of which carcinoma is far more frequent than sarcoma. Primary *cancer* develops slowly on the posterior third of the cords or in other locations, as an excrescence which gradually assumes a cauliflower appearance; the basis of the later ulceration is nodular and there is no scar formation (*v.* SYPHILIS, LARYNX); hoarseness is an early symptom; all kinds of incipient tumors are frequently mistaken for laryngitis. Pain which may radiate to the ears or temples, cough, and dysphagia are constant in the advanced stage. Early operation, either intralaryngeal or by splitting the thyroid, will alone afford relief; inoperable cases must necessarily be narcotized.

Nervous aphonia, the *paralyses* and *spasms* of the larynx are considered under affections of the vagus nerve; syphilis and tuberculosis have already received consideration.

DISEASES OF THE TRACHEA AND BRONCHI.

The *trachea* is seldom involved alone. Tracheitis is frequent, with inflammation of the larynx and bronchi. Hemorrhage may result from diapedesis or from ruptured varices. Tuberculosis, syphilis and tumors are very infrequent.

(A). ACUTE BRONCHITIS.

Acute bronchitis is a most frequent primary or secondary disease, in some localities constituting 10 to 15 per cent. of all diseases. It occurs most frequently in cold, dusty or variable weather; three-fourths of all cases occur between December and April. Its *forms* are (*a*) inflammation of the *larger air tubes*, bronchitis, tracheobronchitis or macrobronchitis, and (*b*) inflammation of the *smaller tubules*, known as bronchiolitis, capillary bronchitis or microbronchitis, which in children is identical with bronchopneumonia (*q. v.*).

Etiology.—(*a*) It is a *symptom of various infections*, as *measles*, *pertussis*, *influenza* and *typhoid;* it also occurs frequently in scarlatina, variola, malaria, pneumonia, diphtheria, erysipelas, syphilis, tuberculosis, sepsis, glanders, actinomycosis, and anthrax. In these maladies bronchitis is secondary and results from the germs or their toxins. (*b*) It may result from the *toxic effects* of potassium iodide or bromide, tobacco, less often carbolic acid, cantharides, mercury and alcohol, which according to Hoffman, acts locally on the air passages. (*c*) *Mechanical irritants* are dust, and other foreign particles, as flour dust, particles of wool, metals, stone, etc., which exist in many factories; overuse or abuse of the voice is also a mechanical irritant. (*d*) *Constitutional conditions*, as obesity, nephritis, rhachitis, gout, scrofula, anæmia, diabetes, malnutrition and cachexia of various kinds, such as cancer, syphilis, or scurvy, are important etiological conditions. (*e*) *Venous stasis* in the lesser circuit favors bronchitis, by congesting the pulmonary veins, which empty chiefly into the vena cava superior and vena azygos; *mitral lesions* are the most important, but other valvular disease, arteriosclerosis, scoliosis, abdominal tumors, ascites or pleurisy, all of which affect the diaphragmatic excursion, are possible factors. (*f*) *Respiratory diseases*, as pneumonia, tuberculosis, abscess or gangrene of the lung, asthma, bronchial stenosis, and bronchiectasis ar usually associated with bronchitis. (*g*) *Cold and bacteria* are als etiological factors. It is disputed whether cold alone is an adequat factor without the coöperation of bacteria, but it at least plays potent predisposing part. Even in cases of apparently primary bronchitis, the pneumococcus, pyogenic cocci, Friedländer's pneumobacillus and other bacteria are found (*v. s.*). Pfeiffer's *micrococcu catarrhalis* is found in 59 per cent. of cases (Ghon, Pfeiffer); i is larger than the staphylococcus and often occurs in pairs. (*h*) *Ag* is a factor. Acute bronchitis may develop at any time of life, but i peculiarly frequent in the *aged* and in *children* between six months an three years old.

Symptoms.—1. Acute bronchitis of the larger tubes (*macrobronchitis* or *tracheobronchitis*) begins (*a*) with some constitutional *depression*, sometimes with fever and chilliness or in children even wit marked toxæmic symptoms. It often follows acute coryza or pharyngitis. (*b*) The *local symptoms* are *substernal rawness* or tickling; actua pain or tenderness over the sternum is mostly tracheal or muscular, fo the bronchi have apparently no nerves for perception of pain. On inspection of the trachea there is visible inflammation, redness, diffuse o circumscribed injection, submucous ecchymosis, swelling and laxnes of the mucosa, and dryness, which is followed by the appearance o a viscid transparent glairy mucous secretion; the mucous glands ar often distended by globules of mucin which slightly resemble youn tubercles; most of these findings are also noted in the bronchi in th rare cases which come to autopsy, but injection and redness largel disappear after death. Microscopically the cardinal signs of inflammation are found, such as paresis of the vessels, round cell emigration, desquamation of the ciliated epithelium, and compensatory effort at restoration by multiplication. (*c*) The *cough* is at first dry and unproductive and may remain so. Later a viscid, transparent sputum is raised, consisting almost wholly of mucin and very few white cell (*sputum crudum*). A day or two later the coughing efforts become les severe as the sputum grows more abundant and mucopurulent (*sputum coctum*); it contains polymorphonuclear and sometimes abundan eosinophilic leukocytes, epithelial cells showing myeloid degeneration and rarely ciliary epithelium or hemorrhagic spots. Localized inflammation at the tracheal bifurcation causes special irritation (Nothnagel). (*d*) The *symptoms resulting from cough* are disturbed sleep hoarseness from forcible closure of the glottis in coughing efforts pain in the side or at the diaphragmatic insertion, from unusua strain on the expiratory muscles during coughing; venous stasis whic induces headache, vertigo, swollen cervical veins, cyanosis and epistaxis; mechanical sequences, as vomiting, involuntary urination, especially in weak and old subjects; and in women, even abortion uterine prolapse, or hernia may occur. (*e*) *Physical findings* ar usually *bilateral;* unilateral signs, though possible, suggest other lesions especially tuberculosis. Auscultation reveals *rhonchi*, which resul from swelling of the bronchial mucosa, from slight stenosis by bronchial secretion and from air passing through or dislodging the secretion. The rhonchi at first are dry (*r. sicci*), and later moist (*r. humidi*). Over the large tubes these rales are sonorous, in the smaller tubules they are sibilant, and in both they vary acoustically. They may be heard simultaneously over the large and small passages, or successively, over the large and then over the small tubules. These rales, large and small, may be felt with the hand (mucous fremitus) or may be heard without the stethoscope. The breathing may be "cog-wheel," lengthened, slowed or sometimes increased by fever, but in quality it is only exaggerated or puerile, never bronchial. Percussion is wholly negative. Macrobronchitis lasts but a few days, possibly a little longer than a week.

2. Microbronchitis or capillary bronchitis is essentially the same pathological process as macrobronchitis but will be considered under bronchopneumonia, because of its great tendency, especially in children, toward pulmonary inflammation.

Treatment.—In the early stage a *full hot bath* may be given, followed by a *hot alcoholic drink*. A hot Turkish bath should never be taken, for exposure afterward is dangerous. *Hot fomentations* over the sternum are beneficial; a hot water bag or a large poultice may be applied over the sternum, but cold packs over the neck and chest are even more efficacious. Patients should always be kept in bed, for but few cases recover promptly without this treatment. A mild laxative should be given early. A drink or two of well diluted whisky is valuable. When the initial symptoms appear gr. x of *Dover's powder* should be given; it not only relieves pain and coughing but also appears to inhibit or modify the inflammation; in children it should be given with great caution because of the frequently unequal mixture of its constituents, and the peculiar susceptibility of the youthful nervous system to narcotics; in the aged it should likewise be administered with care, because the kidneys in old persons excrete slowly, and cerebral atheroma may induce sudden urgent symptoms, as psychical disturbance, Cheyne-Stokes breathing, and other basilar disturbances. The administration of opiates should be followed by some saline aperient. *Muscular pain* necessitates massage and administration of opiates. Other treatment is frequently superfluous, but if the symptoms and cough persist or if the initial symptoms are rather severe we administer:

℞

Vini antimonii	ʒiv.
Heroin hydrochloratis	gr. iss.
Vini ipecacuanhæ	ʒv.
Syr. tolutani . . . . .q.s. ad	℥ij.

M. et S.—One teaspoonful every three or four hours. The patient should be watched lest gastric symptoms appear.

℞

Potassii citratis	ʒiiss.	ʒj.
Codeinæ sulphatis	gr. iv.	gr. j.
Syrupi ipecacuanhæ	ʒiv.	ʒiss.
Succi limonis	℥j.	℥ss.
Aquæ q.s. ad	℥ij.	℥ij.

M. et S.—One teaspoonful every two hours. The first column is the dosage for adults, the second for children.

The fever rarely requires treatment; if high, aconite and belladonna may be given, as in coryza, but tepid sponging is more beneficial. A kettle should be kept boiling in the room to moisten the atmosphere, or a steam tent may be advisable as in croup (see DIPHTHERIA). If the bronchitis lingers longer, the formulæ given under chronic bronchitis will be useful.

(B). CHRONIC BRONCHITIS.

Etiology.—The etiology is essentially that of the acute type. Chronic bronchitis is more common in advanced life, and may develop slowly as a chronic process, or, more often, may follow repeated acute attacks. It may be manifest during the entire year, but most often develops on change of weather or in the cold season, as the "winter cough."

Pathology.—The larger and medium sized bronchi are most involved. Their color is a gray red or brown; the bloodvessels are thickened, and the bronchi become thicker from extravasation of leukocytes and proliferation of the peribronchial connective tissue; the mucous membrane is sometimes hypertrophied, and even protrudes in polypoid and papillary excrescences, or is atrophied, and appears thin and pale like a serous membrane. Erosions are not common but are sometimes seen at the tracheal bifurcation, especially in the aged. Stagnation of secretion is common. The inner surface of the bronchi is at times reticular, due to the prominence and hyperplasia of some elastic fibers and the wasting and sinking of other elastic or muscular fibers. Emphysema and some interstitial fibrous overgrowth are frequently found at autopsy.

Symptoms.—Fever and pain are absent, except in the frequent acute exacerbations. The usual intrinsic physical findings in *chronic macrobronchitis* are *large sonorous rales,* and sibilant rales when acute exacerbations cause invasion of the finer tubules. The cough varies with the intensity of the inflammation, with the amount and character of the secretion, or with changes in the weather. The sputum varies greatly, and upon its character is based the division into the following forms: (*a*) *Dry bronchitis,* the *bronchitis sicca* of Laennec, which is seen in the aged and is almost always associated with emphysema; in this type the sputum is either wholly absent or is very scant and viscid. (*b*) *Bronchorrhœa,* in which the excessive bronchial secretion may be mucous, serous, or purulent; in the serous form the secretion is thin and transparent, as in Laennec's case, in which two quarts were raised daily for twelve years; Struempell observed a serous bronchorrhœa resulting from enlarged peribronchial glands; the purulent form may be confused, on account of its abundant sputum, with bronchiectasis, lung abscess, or rupture of an empyema into the bronchi. (c) *Putrid bronchitis*, which has been described by Laennec, Andral and Traube. It may complicate chronic bronchitis but occurs more often with bronchiectasis, foreign intrabronchial bodies and tuberculosis; in this form the sputum stinks and is acidulous particularly when first voided. It consists of three layers; the upper one is foamy, the middle layer is serous, and the lower one consists of granular gray-green or bluish sediment, in which are also the *mycotic plugs* of Dittrich (1850); these plugs contain various bacteria, leptothrix staining violet with iodine, strepto- and staphylococci, Bacillus coli and Bacillus putridus and sometimes monads. Bacilli which retain fuchsin after treatment with mineral acids ("acid-fast" bacilli) are found in putrid bronchitis and in gangrene of the lung. They closely resemble tubercle bacilli, though they are narrower, more pointed at their ends and do not give inoculation results. Fat and myelin droplets, fatty needles, leucin, and tyrosin are also present. The amount of sputum may even reach a pint. Fever is common, "drum stick" fingers may develop, and complications may follow, as lung induration, diphtheroid ulceration, gangrene, pleurisy, hæmoptysis, cerebrospinal abscess, meningitis or pseudorheumatism. The fever frequently recurs. (*d*) *Blood-stained sputum* occurs particularly in the brown induration of the lungs, which is due to stasis (*v.* CARDIAC STASIS).

Chronic emphysema (*v. i.*) is an almost regular complication and is marked by its usual signs, such as a tympanitic percussion note, voluminous lungs, prolonged and, on exertion, difficult expiration, weakened vesicular murmur, hypertrophy of the cervical muscles, swollen neck veins, signs of right heart hypertrophy and dilatation, and eventually tricuspid insufficiency, liver congestion, and œdema.

Diagnosis.—The diagnosis is generally easy. Determination of its relation to tuberculosis and other causal features is most important. In every case chronic nephritis should be considered.

Prognosis.—The prognosis depends on the etiological factors. Absolute recovery is most unlikely.

Treatment.—1. *Prophylaxis.* Causal affections require treatment. Woolen underwear should be worn to protect against sudden changes of temperature.

2. *Climate.*—When feasible, a dry, warm, even climate is beneficial, as that of Florida, Southern California, New Mexico, the Carolinas, Egypt, and the Riviera.

3. *Cough.*—Codein, morphine, creosote, cod-liver oil and other remedies are employed as in tuberculosis (*q. v.*). Ammon. carb. (*v.* PNEUMONIA) gr. iij. can be given with syr. senegæ ʒj; syr. scillæ ʒj is incompatible with ammon. carb. but may be combined with ammon. chlor. gr. v to x.

℞

Ammonii chloridi	ʒiss.
Misturæ glycyrrhizæ comp.	℥iv.

M. et S.—One teaspoonful after meals.

The mistura glycyrrhizæ comp. (Brown's mixture) contains twelve parts of paregoric, six parts of antimonial wine and three parts of sweet spirits of nitre, which enhance its sedative action. It may be given in doses of from one to four drams. If the heart is weak, it is contraindicated because of the antimony it contains. In all cough preparations, the stomach may become deranged by ammonium, syrups, etc. Chloroform is particularly valuable in paroxysmal unproductive coughs, which opiates may not alleviate because they dry up the secretion.

℞

Ammonii carbonatis	ʒj.
Spiritus chloroformi	℥iss.
Syrupi senegæ	q.s. ad ℥iij.

M. et S.—One teaspoonful in hot water as indicated.

℞

Apomorphinæ hydrochloridi	gr. ss.
Heroin hydrochlorat.	gr. j.
Spiritus chloroformi	℥j.
Aquæ	q.s. ad ℥ij.

M. et S.—One teaspoonful two or three times a day.

Syr. scillæ comp. also contains senega and tartar emetic and is given in doses of gtt. xx to xl. The antimony it contains may depress the heart. Terpin hydrate is excellent, gr. iii to v (x). t. i. d., given in capsules with equal amounts of benzoic acid. The *balsams* are even more efficacious, as ol. terebinthinæ ♏ij. in milk (see formula for emulsion under TYPHOID, TREATMENT OF TYMPANY); ol. santali ♏ x to xx in emulsion is less likely to disturb the kidneys than is turpentine; balsamum Peruvianum ʒss or bals. tolutanum ʒj to iv. The balsams and creosote are directly indicated in fœtid bronchitis. Retention of secretion which does not yield to the action of drugs, may be treated by rhythmical compression of the chest during expiration. Potas. iodide gr. v to x t. i. d. is often excellent.

4. *Cardiac Weakness.*—Cardiac weakness is relieved most efficaciously by strychnine, which is more valuable than digitalis.

5. *Coincident Emphysema.*—Coincident emphysema and asthma (*q. v.*) necessitate the prolonged use of iodides and belladonna.

(C). FIBRINOUS BRONCHITIS.

Etiology.—There are three types of fibrinous or croupous bronchitis. (*a*) The *primary* form is an infrequent disease, of which but few more than 100 cases are recorded; it occurs in males (66 per cent. of cases) principally in those between ten and thirty years, and in weakly and anæmic subjects; it results from bacteria, as the Bacillus diphtheriæ, pneumococcus, strepto- and staphylococcus, aspergillus fungus, and protozoa, or from their toxins. (*b*) The *secondary* type develops by extension from laryngeal and pulmonary disease, or in infections, as pneumonia, typhoid, measles, and scarlatina; tuberculosis causes 50 per cent. of this group; some cases occur in skin diseases. (*c*) *Mechanical, thermal,* and chemical agents are causative factors in a few cases, and some have occurred in connection with valvular heart disease and exophthalmic goitre. Fibrinous bronchitis was first described by Clark, in 1697.

Symptoms.—The early symptoms are those of bronchitis, sometimes with fever, chills and hæmoptysis, but they are ambiguous until bronchial casts of fibrin or mucin are expectorated; this usually happens after a severe coughing paroxysm. The casts may measure even eight inches long, and may be as thick as a lead pencil or a finger; their upper and larger parts are circular or somewhat flattened, solid or hollow, but the lower small portions are branched, solid, spiral and often clubbed at their tips. Though usually termed fibrinous or croupous, many casts do not take Weigert's fibrin stain but respond to tests for mucin; these are then cases of bronchitis mucinosa or pseudomembranacea. Liebermeister (1904) holds that fibrinous is more frequent than mucous formation. The microscopic appearance of the casts is hyaline, their texture is fibrillar, and their surface is often covered with blood disks, hæmatoidin, or Charcot-Leyden crystals. Smaller casts may not be seen until the sputum is agitated in water, which washes off the adherent blood, pus or mucus; this may explain their frequency as an accidental finding. Pending their evacuation, there are *signs of unilateral bronchial stenosis,* dyspnœa, cyanosis, and inspiratory retraction, and there are no breath sounds in the corresponding lung area. Slipping or moving of the casts in the bronchus may cause peculiar rales. After their evacuation new casts form in one or more days. Niemeyer observed the daily shedding of a complete cast of a bronchus and its divisions.

Diagnosis.—In some cases casts are not voided during life, so that the autopsy alone determines the diagnosis. Asthma (*q. v.*) and bronchitis fibrinosa have certain common symptoms, as paroxysmal occurrence, eosinophilia and the crystals and spirals, but are usually differentiated with ease.

Course and Prognosis.—In *the acute form,* which lasts two weeks or less, there is great risk of sudden asphyxia during the evacuation of the casts; there is a death rate of 75 per cent. in the young and 50 per cent. in adults. In *chronic forms* the process lasts for months or even years, (cases of twenty-three and twenty-five years' duration have been observed). The prognosis is uncertain. Complications include splenic tumor, pneumonia, pleurisy, emphysema, and albuminuria which results from dyspnœa.

Treatment.—Treatment is unsatisfactory if not wholly futile. Inhalation of steam and alkaline vapors and pilocarpin should be given in order to loosen the casts. Emetics may be employed to facilitate their evacuation, and iodides and mercurial inunctions to inhibit the reformation. The iodides are the most effective.

(D). BRONCHIAL DILATATION, BRONCHIECTASIS.

Etiology and Pathology.—Laennec first described bronchiectasis, 1819. It is found in 2 per cent. of autopsies and is *always seconda* (*a*) to *bronchial disease*, stenosis, catarrh, inhalation of dust, forei bodies, or pressure from aortic aneurysm; (*b*) to *lung disease*, tube culosis, interstitial pneumonia (Corrigan), hypostasis, or atelectasi (*c*) to *pleural disease*, such as adhesions pulling on the bronchi. Near all cases are *acquired*, and occur mostly in middle aged males (77 p cent.). Rare congenital cases are recorded. These may be divid into (i) the unilateral, universal or diffuse form (Grawitz), (ii) tho due to congenital atelectasis (Heller), and (iii) those resulting fro prenatal bronchial (syphilitic) stenosis.

There are *two forms* of bronchiectasis: (*a*) the cylindrical or fusifor multiple or *pater noster* form, which develops from the atrophy of t muscular and elastic fibers induced by protracted severe coughing a perhaps by stagnation of secretion; it may follow measles, catarrh pneumonia, whooping cough or emphysema. (*b*) The rarer *sacculat form*, in which the bronchus may reach a diameter of even 3 inche and which might be called a bronchial aneurysm. The connecti tissue usually disappears, the ciliated epithelia are replaced by flat cel and the mucosa is thin, red, and swollen, and sometimes presen villosities and varicosities. The sacs may become closed at their neck particularly when caused by aneurysm, syphilis, or tuberculosis; thi converts them into sacs filled with gelatinous, cheesy or calcareou contents. They are known as lung stones, and consist of calcium carbonate and phosphate with traces of magnesia; they have an organi nucleus and sometimes cause "bronchial colic," or symptoms like thos of phthisis.

Bronchiectasis is seldom single; in multiple bronchiectasis the lun sometimes resembles a sponge or porous cheese. It occurs principall in the lower lobes and in bronchi of the third and fourth order. It ma be bilateral (60 per cent.) but the congenital pleuritic and pneumoni forms are always unilateral. Adhesions are very common when the cavity reaches the pleura.

Symptoms.—Most cases are not discovered clinically, and Wei therefore concluded that "most cavities escape detection and most diagnoses of cavities are incorrect." Sacculated forms produce more symptoms and signs than do the cylindrical forms, which are almost always impossible to recognize.

1. **Sputum and Cough.**—The sputum and cough are paroxysmal. When secretion fills the sac and flows over onto the normal sensitive mucous membrane beyond it, cough develops and quantities of mucopus are voided in the "*mouth full*" *fashion* described by Wintrich.

Change of posture produces similar effects. The largest amount is raised in the morning and this is frequently followed by vomiting. The daily quantity may amount to a quart. Its odor is acid, sweat-like, and offensive, but it does not attain the fœtor of putrid bronchitis nor the mature foulness of gangrene. When collected it forms in three layers; the upper one is foamy, the middle one is serous, and the lower one is purulent. A nummular or globular sputum may be noted as in tuberculosis, but if put in water it is more flocculent, more granular, and airless; and microscopically it contains altered leukocytes and fatty needles, occasionally red cells, hæmatoidin crystals, and very rarely, when the bronchial wall is ulcerated, elastic fibers.

2. Physical Examination.—(*a*) *Inspection* may reveal diminished respiratory movement, inspiratory retraction over the cavity, or flattening of the chest wall. The patient leans toward the diseased side, and rarely inclines toward the sound side or sits erect, in order to lessen the trickling out of the secretion. In the rare cases of bronchiectasis in an upper lobe the constant escape of the secretion results in exhausting and most unfavorable coughing (Skoda). The *x*-rays may show the location of the cavity. (*b*) *Palpation* and (*c*) *percussion* give variable results depending on whether the cavity is full or empty. Dulness prevails when it is full; when it is empty a tympanitic note results. A higher note on opening the mouth, on inspiration, or on change of posture has the same value as in cases of tuberculous cavity (*q. v.*). (*d*) On *auscultation*, *bronchial* or *metallic breathing* may be heard when the cavities are superficial but it is absent in dilatations which are small, deep, or full of secretion. *Large* and *small* rales, if heard persistently in the same locality, are suggestive of a deep bronchiectasis. The sound tissue over the cavity imparts a metallic consonance. *Metamorphosing breathing* is a certain sign of a cavity.

3. Complications.—The general nutrition sometimes remains normal; slight cyanosis together with anæmia is frequent. Hectic fever is often absent, but death may result, with symptoms resembling those of phthisis. Putrid bronchitis is frequent. Hæmoptysis from ulceration of ruptured varices occurs in 40 per cent. of cases; pneumothorax, pleurisy, empyema, lung induration or gangrene, emphysema, lobular pneumonia, amyloidosis and hypertrophy and dilatation of the right ventricle are fairly common. Marie's *ostéo-arthropathie hypertrophiante pneumonique*, rheumatoid affections (Senator), suppurative meningitis or abscesses in the brain and cord are occasional. In rare cases the cavity may bulge through the interspaces.

Diagnosis.—Diagnosis depends upon the character of the sputum, its paroxysmal voidance, and the signs of cavity formation. *Differentiation* is required (*a*) from *tuberculous cavities*; this is made by means of family history, habitus, the presence of elastic fibers and tubercle bacilli in the sputum, which are definitely determined by inoculation; the tuberculous cavities are most often apical, but frequently bilateral; they void less sputum and suffer less rapid variation in their physical signs. Bronchiectatic cavities occur mostly in the lower lobes, void more sputum, less often induce hemorrhage and more frequen cause right heart changes. (b) From *perforating empyema*, in whi cholestearin and hæmatoidin crystals are far more frequent than bronchiectasis; (c) from *abscess;* (d) from *gangrene* of the lung (*v.* (e) from *putrid bronchitis*, in which there is no sudden volume evacuated sputum nor signs of cavity; (f) from *encapsulated pyopn mothorax*, which may be very difficult of differentiation.

Prognosis.—The immediate prognosis is rather favorable, but u mate recovery is exceptional.

Treatment.—The therapeutic indications are (a) *evacuation of secretion*, for which expectorants and expiratory compression of chest, as in bronchitis, are somewhat useful; sedatives should ne be given, lest gangrene develop; (b) modification of *putrescence* balsamic remedies, as in fœtid bronchitis; inhalations of creosote m be given, though they are most irritating at first; a dram of creos is slowly vaporized over a flame, and the patient, in a closed room, hales the fumes with the eyes closed and the anterior nares plugg the procedure may be gradually lengthened from a few minutes to h an hour; (c) *surgical drainage*. An accessible cavity may be incis and drained, though accurate diagnosis of its location is difficult. Garré's collection of 57 cases (1902), 63 per cent. recovered and 37 p cent. died; (d) *change of climate* (*v.* TUBERCULOSIS).

(E). TRACHEAL AND BRONCHIAL STENOSIS.

1. Tracheal Stenosis.

Etiology.—The cause is less often within than without the trach *Tracheal causes* are rhinoscleroma (Stoerk, 1874), syphilis which 40 per cent. of cases also implicates the bronchi, tumors (cancer, poly fibroma, enchondroma, sarcoma, cancer or aberrant thyroid), forei bodies, tracheotomy granulations, and in rare cases perichondrit Causes *outside the trachea* are more frequent, as thyroid enlargeme particularly the retrosternal, cystic and circular forms, or struma which hemorrhage or inflammation has occurred; in some cases sudd death results, notably when the cartilages have been absorbed. Ane rysm, adenopathies, mediastinal tumors, vertebral tumor or cari and thymus hyperplasia are other factors.

Symptoms.—The symptoms may be ranged in *three series or stag* (a) when there are no symptoms or when they occur only on exertio (b) when there is constant dyspnœa, and (c) when suffocative attac terminate in final asphyxia. The *dyspnœa* is of a mixed type, *i. e.*, is both inspiratory and expiratory; the number of respirations lessened, there is inspiratory stridor and intercostal retraction, all t accessory muscles come into play, the head is held well forward a there is little or no laryngeal excursion. The pulse is more tense, vari in rate, and is sometimes of the paradoxical type. Tracheoscopy may reveal the location of the lesion. This is done most advantageously in the strong sunlight; the patient should stand with his back to the light and with the head bent forward, and the observer should be seated on a low seat before him. Fraenkel affirms that in 25 per cent. of tracheal stenoses due to tumor or aneurysm, there is coincident paralysis of the vocal cords.

2. Bronchial Stenosis.

Etiology.—*Intrabronchial* causes are most common and consist of pus, mucus, blood, fibrin and foreign bodies; obstruction due to the latter is laryngeal in 33 per cent., tracheal in 26 per cent., and bronchial in 41 per cent., of the cases. *Extrabronchial* factors are next in frequency; aneurysm, mediastinal lymphosarcoma and perilymphadenitis leading to fibrosis occur most often; less common are pericarditis with effusion, dilatation of the left auricle, cancer of the œsophagus, tumor of the lung, and thyroid or thymic strumas. Twenty cases have been reported in which lymph glands ruptured into the trachea or bronchi, causing suffocation; only three were saved by tracheotomy. *Interbronchial causes* (*i.e.*, in the bronchial wall) are the least common, as syphilis, glanders, rhinoscleroma and tumors. Thirty-six cases of primary cancer are recorded. The right is stenosed nearly twice as often as the left bronchus.

Symptoms.—Occlusion of a bronchus lessens the amount of air entering the lung which it supplies, whence the *vocal fremitus* and *breath sounds* are immediately *weakened* or suspended and the respiratory excursion and Litten's diaphragmatic sign are decreased. There is inspiratory retraction of the intercostal spaces on the affected side. *Inspiratory dyspnœa* occurs, particularly in acute cases. The head is thrown forward and the larynx moves up and down but little. The percussion note is not essentially altered, and this excludes many causes of dyspnœa, as pneumonia, etc.; after a time a somewhat tympanitic note may result from relaxation of the lung or acute emphysema. A local stenotic murmur over the bronchus is present in a few cases. Paroxysmal dyspnœa may result from stagnating secretion. The pulse is more tense and often slower. The *x*-rays may locate the obstruction. Bronchoscopy is sometimes helpful, but it is difficult to perform even for the experienced specialist. Death results from (*a*) the original disease, as aneurysm or tumor; (*b*) failure of the right heart, (*c*) asphyxia, with delirium, cyanosis, convulsions or Cheyne-Stokes breathing, or (*d*) pulmonary œdema, abscess, gangrene, tuberculosis, lobular pneumonia, lung induration or hemorrhage.

Diagnosis.—(*a*) *Of the location* of stenosis: in laryngeal obstruction, there are local findings and free up-and-down excursion; the subject holds the head well back, and the ingress of air to both lungs is equally impaired; tracheoscopy, bilateral lung involvement, the absence of laryngeal excursion, and the holding of the head forward are sufficient proof of tracheal obstruction; in bronchial stenosis but one lung suffe for air, the larynx is nearly immobile, and the head is thrown forwar (*b*) *Of the nature* of stenosis, exact diagnosis is more difficult, and impossible without distinct physical signs, as those of aneurysm, without history of the presence of foreign bodies.

The *prognosis* depends on the cause.

Treatment.—Treatment is also largely that of the cause, as syphili tuberculosis, etc. Thyroid extract and iodine are efficacious in ov 50 per cent. of cases of thyroid swellings. When the obstruction aneurysmatic, rest, mercury, iodides, venesection and gelatin injectio often afford considerable relief. When there are foreign bodies, t patient should be inverted, sharply struck on the back, and shake the physician should have long forceps and tracheotomy instrumen in readiness. The mortality from the presence of foreign bodies in t lower air passages is 52 per cent. with expectant treatment, and 23 p cent. with operation.

Bronchial and lung stones (*v.s.*) result from tuberculosis of th bronchial glands, from bronchiectasis, petrification of the bronch calcification and ossification of the lungs, and from gout and th presence of foreign bodies. Symptoms are frequently absent or a those of the primary disease; purulent bronchitis, asthma ("bronchi colic"), hectic fever and hæmoptysis mark some cases.

Stenosis of the bronchioles may result from *bronchiolitis fibrino obliterans* (Lange and Fraenkel).

(F). BRONCHIAL ASTHMA.

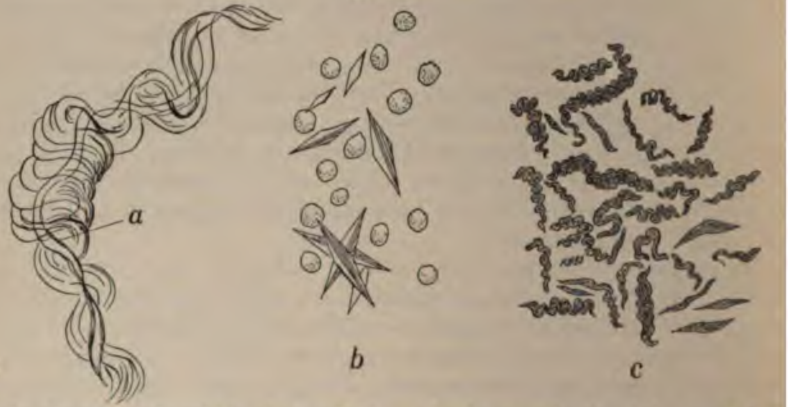
Definition.—A paroxysmal dyspnœa, due to stenosis or spasm of t smaller bronchioles, which is nervous or reflex in origin and is chara terized by expiratory dyspnœa, slowing of respiration, congestion of t bronchioles, a peculiar mucous exudation and acute emphysema. is also known as asthma spasmodicum or nervosum, to distinguish from other so-called asthmas, as the cardiac or renal types.

Etiology.—When due to no obvious cause, asthma is termed essent or *primary*, and when due to a clear cause it is called *symptomatic* reflex asthma (which occurs through the vagus). (*a*) *Nasal* and na pharyngeal disease is the source of the reflex asthma in numbers cases, as proven by Voltolini, Hack, Haenisch, Porter, Daly, Roe a others; it may be caused by rhinitis, tonsillar hypertrophy, adenoi or pharyngitis granulosa; the nose may be so hypersensitive that t odor of flowers, dust, hot air or smoke precipitate an attack. Asthma may be initiated by *pressure on the vagus* trunk, resulti from thyroid tumors or from enlargement of the bronchial glan following tuberculosis, measles, pertussis, or rickets. (*c*) *Abdomi diseases* are possible though not frequent causes; the asthma dyspep cum of Henoch, Meyer and Pribrâm has been especially emphasiz of late by Hayem who describes asthma resulting from gastric dil tation. Ovarian and uterine disease and pregnancy have been considered causal factors. (*d*) *Asthma toxicum* has been reported in lead and mercurial poisoning; *uræmic* and gouty asthma are also due to toxins. (*e*) It occurs especially in weakly, anæmic, nervous, scrofulous and rhachitic subjects, in males (66 per cent.) between the age of twenty and forty (though it has recently been claimed that 33 per cent. of the asthma cases develop under ten years), in the upper classes, and in professional men.

There are various theories as to the *mechanism* of the asthmatic attack; these are as follows: (*a*) it is a *spasm* of the *bronchial muscles* (Trousseau, Rhomberg and Biermer). Trousseau spoke of asthma as "an epilepsy of the lungs"; (*b*) it is a very acute catarrh (Louis and Traube); Stoerk found the larynx and trachea deeply injected; (*c*) it is a *vasomotor* disturbance (Weber); or (*d*) an acute exudative bronchitis, causing bronchial spasm (Curschmann, Lebert). Its pathology is uncertain, as but half a dozen autopsies are recorded; the ciliated epithelium is desquamated, the bronchioles are congested and there is eosinophilic exudation.

Symptoms.—The attack *begins very suddenly*, often at night and with or without such causes as fright, cold, heat, change of residence, etc. Sometimes there are prodromes, as conjunctivitis, coryza or flatulence and sometimes the attack coincides with the menses. (*a*) *Dyspnœa* is the first and the essential symptom; though at first slight, it soon becomes urgent; the patient opens the windows, sits upright, grasps the arms of the chair to brace the accessory respiratory muscles, and exhibits extreme distress, anxiousness and pallor, which is followed by cyanosis; the dyspnœa is expiratory, and the abdominal muscles are board-like during expiration; inspiration is somewhat lengthened, and *expiration is greatly prolonged*, wheezing, *and whistling;* respiration is not usually increased, but may reach 30 or 40. (*b*) There is marked *cyanosis* in the face and extremities, the cervical veins bulge out like cords, the skin is clammy, and the pulse indicates poor oxygenation by its tenseness, smallness and frequency; carbon dioxide intoxication causes the occasional headache, delirium, tinnitus, twitchings, convulsions and coma. (*c*) The *cough is dry* and unproductive. (*d*) The voice is faint and hoarse and the trachea is red. (*e*) *Other physical signs* are as follows: On *palpation*, the vocal fremitus is found to be decreased by the bronchial spasm or secretion; rales are often felt. The normal *percussion* note is replaced by a *tympanitic* note, due to acute pulmonary emphysema; the lungs are voluminous, and distend the thorax, cover the heart and push down the diaphragm, whose excursion is thus reduced to a minimum. On *auscultation*, the vesicular murmur is usually found to be faint or absent (Laennec) because of secretion in the bronchioles, and loud whistling, piping, or *sibilant rales* are heard often without the stethoscope and largely during expiration. They are due to bronchial stenosis. (*f*) *Sputum* is voided for the first time toward the end of the attack; this occurs as a species of crisis to the threatening, but very seldom fatal, asphyxia. A few drams of tenacious mucus, resembling egg albumin or gelatin, are voided Minute gray balls are seen; these are the "*perles*" of Laennec; whe unrolled the "pearls" are seen to contain the *spirals* of Leyden, Unga and Curschmann; they are best detected with the naked eye, and on black background; they measure 2 or 3 to 10 x ½ to 1 mm.; the are mucin casts of the bronchioles, to which may cling leukocyte epithelia, fat or myelin droplets; some show a central band of mucin it is thought that they occur from the passage of mucin through th

Fig. 32.



Spirals and crystals in bronchial asthma. *B*, Charcot-Leyden crystals; *A*, spirals magnified, *C*, their natural size.

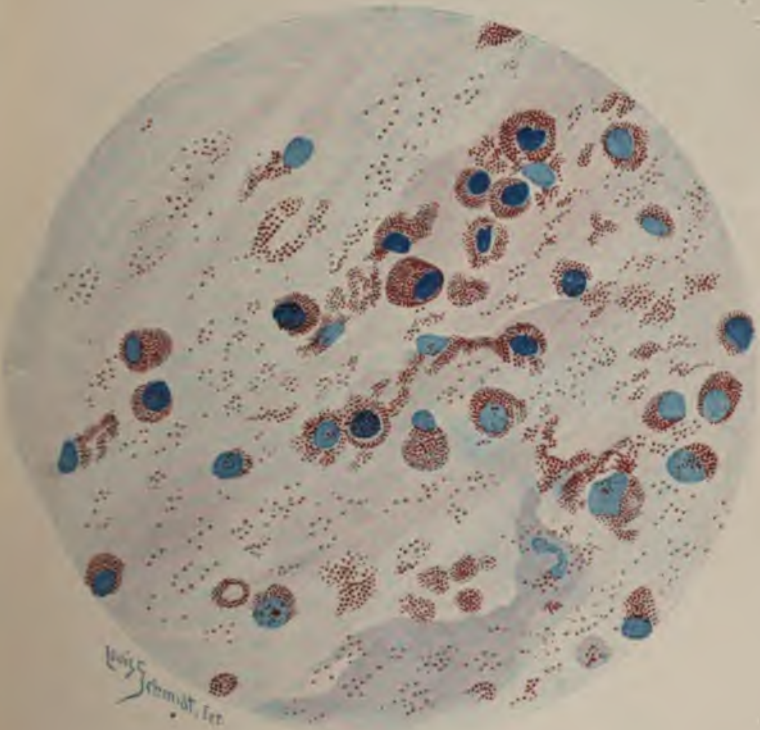
contracted bronchioles; they disappear within a day or two; spirals also present in catarrhal or fibrinous bronchitis and in pneumo The *Charcot-Leyden crystals* may be seen in yellow dots in the sput their origin is still disputed; they are pointed and octahedral, consis an organic basis with phosphoric acid, and are also seen in leukæ tissues, in normal bone marrow, and in semen. Hæmosiderin may found in the sputum. The eosinophile leukocytes are increased in sputum and blood, constituting a quarter to more than half of white cells. The crystals, spirals, and the occasional fibrinous casts a result rather than a cause of the asthma. (Plate X.)

The attacks last from a few minutes to several hours and may *repeated* at long or short intervals. In the hebdomadal form, atta recur every Sunday or Monday; they are probably due to change routine or to digestive excesses. The urine after the attack conta increased uric acid; constipation and flatulence may result fr fatigue of the diaphragm and expiratory muscles.

Diagnosis.—The diagnosis is made by (*a*) the paroxysmal occurre of asthma, (*b*) the expiratory dyspnœa, and (*c*) the acute transi emphysema.

Differentiation.—(*a*) All diseases causing *inspiratory dyspnœa* can at once excluded, such as œdema or spasm of the glottis, paralysis of post. crico-arytenoid muscles, and tracheal and bronchial stenosis fr

PLATE X.



putum from a Case of Bronchial Asthma, showing large numbers of Eosinophilic Leukocytes and Free Granules. (Simon).

It will be noted that the leukocytes are all mononuclear. (Eye-piece 1, objective 1-8, Bausch & Lomb.)

aneurysm. In *spasm of the glottis*, the spasm is inspiratory and of short duration, the larynx moves up and down, and the epigastrium retracts during inspiration; there is no emphysema, and no spirals are found. Spasm of the *diaphragm* is very rare (*v.* PHRENIC NERVE); it lasts a much shorter time, the epigastrium bulges during inspiration, there is spasm of all the inspiratory muscles, hysterical stigmata are often observed, and the lungs are normal. (*b*) *Expiratory dyspnœa* may also occur in (i) *chronic bronchitis with emphysema.* In these cases the emphysema is constant, the lungs show constant rales and fewer eosinophiles are present in the sputum. (ii) It also occurs in *immovable tumors* below the vocal cords which may turn upward during expiration (this is shown by the laryngoscope). (*c*) *Asthma cardiale* (so-called) is of a mixed type, *i. e.*, inspiratory and expiratory, and is therefore readily differentiated. The "lungenstarr" or lung distention (*v.* SIGNS OF VALVULAR HEART DISEASE), due to its engorged vessels, may simulate the emphysema of bronchial asthma. Cardiac asthma may occur either with high or low arterial tension, whence the condition of the pulse is no certain criterion. The eosinophiles are not increased to the same degree as they are in bronchial asthma.

Prognosis.—The prognosis is uncertain and depends on the etiological accessibility of the asthma. It is more favorable in young individuals than in those of advanced years. Cases which recurred for 64 years are recorded. Chronic bronchitis and emphysema are likely to develop.

Associated affections may include epilepsy, migraine, angina pectoris or vasomotor disturbances.

Treatment.—1. Of the Attack.—A hypodermic injection of *morphine*, gr. $\frac{1}{4}$, with *atropine* gr. $\frac{1}{60}$ and *nitroglycerine* gr. $\frac{1}{60}$, gives the most prompt and reliable results. Other measures are purely accessory. Chloral is almost useless, for the condition usually requires single doses of gr. xxx to xl, which are too large for safety. Fresh air, strong coffee, whisky, a cigar or cigarette, the application of cocaine to the nose, fluid extract of grindelia ♏. x to xx especially in bronchitic types, nitrite of amyl pearls, or a few whiffs of chloroform, sometimes give relief. The vexing question arises as to leaving chloroform or morphine in the hands of the patient whose attacks often come and go without medical aid; as a rule they should never be left with the patient. Inhalation powders, which are burned like incense, are very generally used; they all contain some such palliative as lobelia, stramonium, belladonna, hyoscine, etc. (Lobelia, powdered black tea and powdered stramonium leaves, each one ounce; soak well in two ounces of a saturated solution of potassium nitrate; dry.) Excellent cigarettes are made, containing arsenic, potas. nitrate, belladonna, and stramonium; their antispasmodic effects on deep inhalation of the smoke is sometimes remarkable.

According to Burney Yeo, the celebrated *Cigarettes d'Espic* are said to be made of the following ingredients:

Belladonna leaves	5½ parts.
Hyoscyamus leaves	2¾ parts.
Stramonium leaves	2¾ parts.
Phellandrium aquaticum	1 part.
Extract of opium	⅕ part.
Cherry-laurel water	a sufficiency.

Oxygen is occasionally efficacious and strong coffee may sometimes abort the attack.

2. Of the Tendency.—(*v.* ETIOLOGY.)—The nose requires especial attention; cauterization of sensitive areas, even when there are no gross lesions, is imperative. The diet should be regulated; the evening meal should be light, and carbohydrates and tea should be restricted; an emetic may abort an attack of the gastric type. Nervines (valerian, arsenic, bromides) may be used when necessary. The best remedy between attacks is potassium iodide; this treatment should be continued for months; potassium iodide is combined to advantage with Fowler's solution and belladonna, not only to correct the action of the iodides on the skin and secretory glands, but also for their direct nervous and antispasmodic effects:

℞

Liq. potassii arsenitis	ʒj.
Tr. belladonnæ	ʒj.
Potassii iodidi	ʒij.
Fluidextr. grindeliæ robustæ	ʒvj.
Aquæ	q.s. ad ℥iv.

M. et S.—One teaspoonful after meals, in water.

Alkalies may be vaporized and inhaled, as sodium bicarbonate or sodium chloride. Climatic treatment is as beneficial as in bronchitis; a low level near the sea and in a semitropical climate is an ideal location. General hygiene and the hardening of the patient by fresh air and cold rubs are important.

DISEASES OF THE LUNG.

(A). EMPHYSEMA.

Definition.—A permanent disease of the lungs, characterized pathologically by atrophy of the alveolar walls and great distention of the alveoli, and clinically by a large chest of the inspiratory type, distended lungs with poor excursion, and hypertrophy and dilatation of the right ventricle. Emphysema means a "blowing up" or distention of the lungs, of which the various types will be considered under DIFFERENTIATION. It was first described by Laennec (1826). It constitutes 7/10 per cent. of all autopsies (Virchow).

Etiology.—Substantive or alveolar emphysema occurs mostly in individuals over 30 or 40 years of age, in men more than in women, and in cold climates. The immediate cause is chronic bronchitis and bronchiolitis (Laennec), *catarrhe sec*; it may follow pertussis or bronchial asthma; its comparative infrequency is not generally understood, whence many writers assume a congenital or hereditary weakness or hypoplasia of the elastic tissue of the lung. Coughing, blowing, as in the use of wind instruments, straining during parturition or physical toil, artificial respiration in the new-born, and alcoholism are also considered to be causes.

Pathology.—On opening the chest at autopsy, the lungs are found distended (*volumen pulmonum auctum*, Traube); they overlap the heart, depress the diaphragm, and narrow the mediastinum. Their surface is pale rose-colored, is somewhat anæmic, and presents numerous small vesicles, ½ to 3 or 4 mm. in diameter, representing the fusion of several alveoli. Some large bullous areas are found under the pleura. The lungs crepitate slightly to the fingers, under which they feel, as Laennec put it, "like a pillow of down." The lungs collapse but little on section. In some places the pigment deposit is less than usual; this is called *albinism* by Virchow. Emphysema is most marked on the surface of the lung, particularly over the upper lobes, on their convex anterior aspect, and behind near the spine. The normally thin edges of the lung are greatly rounded. *Histologically*, two findings are characteristic: (*a*) There is *atrophy* of the *interalveolar elastic tissue*, so that the *alveoli fuse*, corresponding to the vesicles seen on the surface; slight intercommunication of the alveoli is normal, but the coalescence of several alveoli into one is extreme in emphysema; these changes account for nearly all the clinical findings in the lung. (*b*) The *capillaries* are *obliterated* by *thrombosis*, and become fatty, which explains the clinical hypertrophy, dilatation and ultimate failure of the right heart. The bronchi are frequently inflamed, thickened, shortened, and sometimes dilated.

Pathogenesis.—It is not wholly clear whether the elastic wasting or the vascular occlusion is the primary change; or, if the elastic atrophy is primary, whether it is acquired or congenital. The changes may be brought about (*a*) *mechanically* by forced inspiration (Laennec, Rokitansky, Gairdner) or by forced expiration (Jenner, Mendelsohn). This theory is most favored by clinicians, though pathologists think that emphysema is due to (*b*) *nutritive alterations*, as vascular changes, or atrophy of elastic or muscular tissue; calcification of the costal cartilages is probably more sequential than causal.

Symptoms.—Symptoms may be considered under two general headings: (1) Those of expiratory insufficiency of the lungs, due to their distention which results in turn from the loss of elastic tissue; and (2) those of cardiac insufficiency, which follow the above and result from obliteration of the vessels in the lesser circuit.

1. **Expiratory Pulmonary Insufficiency.**—Normally expiration is wholly passive, resulting from the elasticity of the lungs and collapse of

the chest. Expiration in emphysema is incomplete, as shown by Waldenburg's pneumotometry and Riegel's stethography; in the latter the curve resembles one resulting from division of the vagus. The *cyrtometer* shows that the chest is more circular; the *spirometer* shows a decrease of 20 to 60 per cent. in the normal vital capacity (2–4000 c.c.) and *mensuration* shows a decided reduction in the normal expansion of 2 to 3 inches. Upon *inspection*, dyspnœa is the most conspicuous finding; it is expiratory and is due to the loss of lung elasticity and consequent impairment of lung circulation and oxygenation; it is increased by exertion, by bronchial catarrh, by accessions of true asthma, and by cardiac insufficiency. If emphysema develops gradually it may cause no dyspnœa (Traube). The frequency of respiration is increased to twenty-five or thirty and the type is costal. The *facies* and *habitus* are most characteristic; the eyes are somewhat prominent, the nose is bluish and slightly thickened, the head is thrown back and the chest forward to give play to the accessory muscles of respiration and the skin is cyanotic even during rest and when dyspnœa is not urgent. A network of dilated veins is often seen over the lower chest resulting merely from venous obstruction. The jugular veins fill enormously on expiration and often pulsate during the diastole. The neck is short, and the hypertrophied sternomastoids, scaleni and trapezii protrude rigidly. The back and abdominal muscles are rigid, and thus supplement the inadequate action of the diaphragm. The shoulders are elevated, though stooped; the chest is of the permanent inspiratory type and is usually *barrel-shaped* (from loss of the elastic tissue rarely of the paralytic type; the anteroposterior, oblique, and sometimes the transverse dimensions of the chest are increased. The raised clavicles accentuate the supraclavicular notches, in which the apices sometimes protrude, on coughing, as hernial tumors. The angle of Louis is prominent and the interspaces are wider in the upper and narrower in the lower parts of the chest, and retract with each inspiration. The lower thorax shows a groove indicating the attachment of the expiratory muscles. The play of the diaphragm is limited, as shown by the *x*-rays and by the absence of Litten's sign. *Palpation* detects the rigidity and dilatation of the thorax, its poor excursion, the hardness of the muscles, the thick, often calcified costal cartilages, and decreased vocal fremitus. The edge of the liver may be found depressed, though the spleen is seldom palpable. *Percussion*, absolutely essential to a diagnosis, gives a note which is loud and deep—hyperresonant—or on the sides and back it is actually "*box-like*" or tympanitic; it is also described as muffled or wooden and in extreme cases impresses one as dull (Skoda). The voluminous lungs narrow or wholly cover the cardiac dulness, depress the upper level of the splenic and hepatic dulness, invade Traube's space, and show either no respiratory excursion or movement not exceeding a finger's breadth. At the back the lungs may reach the twelfth dorsal or even the second lumbar vertebra. Upon *auscultation*, expiration is found greatly lengthened so that it is to inspiration as 1 to 2 or 4, the converse of the normal. Vesicular breathing is absent, or the breathing is indeterminate; even when the stethoscope rises and falls there may be no audible sound. A rubbing sensation may result from muscular contraction or possibly from the emphysema vesicles. Rales result from bronchitis or stasis; a pearly-colored mucin with few cells may be evacuated after much coughing.

2. Cardiac Insufficiency.—The work of the right ventricle is increased because the imperfect expiration allows neither free aspiration of venous blood into the auricles nor adequate filling of the aorta, and because the pulmonary arterioles are in part occluded. The right ventricle, therefore, hypertrophies, and this causes the accentuated second pulmonic tone; hypertrophy occurs mostly about the pulmonary conus arteriosus. The diaphragm is low, and therefore the hypertrophied and more horizontal heart beats in the epigastrium. Dilatation soon follows and is accompanied, in advanced cases, by tricuspid leakage, nutmeg liver, congested kidneys, ascites, œdema, and other signs of cardiac insufficiency. The alteration in the heart, so marked at autopsy, is often obscured during life, for the lungs cover the heart and muffle its tones. Functional heart murmurs are mostly systolic, very rarely diastolic, and result chiefly from degeneration or relaxation of the heart muscle.

The *clinical course* is long and often covers decades. Patients may acquire emphysema in childhood and live to be 60 or 70 years old.

Diagnosis.—Developed types are easily recognized by the barrel-chest, its hyperresonance, the low borders of the lung, their lack of excursion, the obscuration of cardiac dulness, the expiratory dyspnœa, and cardiac stasis.

Differentiation.—(*a*) *Acute distention of the lung* or acute emphysema, as after asthma or pertussis, cannot be distinguished by one examination. It is seen repeatedly in bronchial stenosis due to aortic aneurysm. It regresses rapidly even after months, when its cause is removed. (*b*) *Vicarious or compensatory emphysema* often develops in one lung when contralateral pleurisy or cirrhosis of the lung exists; it may occur on the same side with the lesion; marked apical emphysema almost certainly indicates some deeper induration; it may develop in the death agony; it may eventually cause wasting of the lung. (*c*) *Emphysema senile*, or Jenner's "small-lunged" emphysema, is merely atrophy; the connective tissue wastes and the alveoli fuse, but the lungs are not enlarged; they are indeed smaller, the diaphragm stands higher, the cardiac dulness is increased, the chest flattened, the ribs oblique, the respiratory muscles wasted, the right heart is not hypertrophied, and respiratory excursion is retained. (*d*) From *interalveolar interstitial emphysema* the first differential point is the etiology; (i) it may be caused by trauma, external or internal, which occurs in 57 per cent of tracheotomies (Money), and is promoted by raising the deep fascia from the trachea during the operation (Champneys); it is especially frequent with pneumothorax. (ii) It may result from foreign bodies. (iii) Ulceration of the air passages, gangrene, abscess,* or

tuberculosis and less often gastric ulcer or cancer may be its caus
Other etiological factors are (iv) forced entrance of air, as in resuscit
tion of the new-born; (v) severe coughing efforts, as in whoopir
cough; (vi) severe pressure efforts, as in parturition; and (vii) spas
of the glottis. The second point is a series of symptoms which are nc
however, always present, as creaking in the mediastinum and som
times with each systole; emphysema and crepitation of the cervic
cellular tissue, bulging of the interspaces, creaking and tympany at tl
edge of the ribs when the emphysema is under the costal pleura; a
sence of the vesicular murmur; sometimes pneumothorax; and oblite
ation of the heart dulness, dyspnœa and air embolism. It is usual
fatal. (*e*) In *congenital hypertrophy* of the lung (*pulmo excessivu*
the lungs are very voluminous, but their excursion, note, auscultatio
and percussion are normal. To the author it appears to be especial
common among Poles. (*f*) *Pneumothorax* (*q. v.*) is unilateral,
usually associated with fluid in the pleura, has a more tympanit
note, and is associated with succussion, tinkling rales and other pat
ognomonic signs.

Prognosis.—Recovery is unusual in the genuine substantive forn
which is permanent. Transient forms resulting from pertussis, foreig
bodies, or asthma, often or usually regress completely. Life, eve
relative comfort, may be sustained for years or decades. Much depen
on associated conditions, as frequent severe bronchitis, chronic nephr
tis, arteriosclerosis, or myocarditis. Extreme forms impede the flo
of chyle which, with dyspnœa and fatigue, impairs nutrition. Sever
complications are failure of the right heart upon which compensatio
devolves, cyanosis, gastric disturbance, diarrhœa, hemorrhage fron
the lungs or bowels (in arteriosclerosis, gout or infarction), scant
urine, and anæmia (in the aged).

Death usually results from cardiac failure or from œdema pu
monum. It may occur from apoplexy, pneumothorax, hæmoptysi
uræmia, and very rarely from pulmonary inflammation, to whic
subjects of emphysema show no predisposition. The teaching th
tuberculosis (Rokitansky) or valvular disease (Bouillaud) exclu
emphysema, is incorrect.

Treatment.—(*a*) *Prophylaxis* concerns chiefly the concomitant
causal bronchitis. Severe exercise is to be avoided. (*b*) *Coincide*
gastric, renal or gouty disease must be treated. (*c*) *Climate* is as im
portant a factor as it is in chronic bronchitis, an almost inseparab
associate of emphysema; altitudes are expressly to be avoided as the
directly promote emphysema. (*d*) *Pneumotherapy* is sometimes ben
ficial. It includes inhalation of thinned air, or, where there is marke
catarrh, of compressed air. Two or three treatments of ten minute
should be given daily, and the time should soon be extended to half a
hour. (*e*) *Chronic bronchitis* (*q. v.*), and (*f*) *asthma* (*q. v.*) are impor
tant considerations in treatment. (*g*) *Rhythmic compression* of th
chest, three or four times daily, should be practised during 20 to 3
expirations. (*h*) *Cardiac insufficiency* is treated as in valvular o

myocardial heart disease (*q. v.*), and strychnine, venesection, and other measures should be used. (The topic is discussed briefly, as no remedy nor measure can restore lost elastic tissue nor return resilience to damaged contractile fibers.)

BRONCHOPNEUMONIA.

This affection, partially outlined by Sydenham and Gebhart, was first well described in 1840, by Rilliet and Barthez, who separated it from lobar pneumonia and recognized it as a secondary disease. It might be well classed among the infections, such as fibrinous pneumonia, except that unlike the latter it is not due to a single specific microörganism.

Definition.—Bronchopneumonia is defined with difficulty; (*a*) *etiologically* it is due to various microbes, is generally secondary to the bronchiolitis of some specific infection, as measles or pertussis, and occurs chiefly in children; (*b*) *pathologically* it develops about the bronchioles (bronchopneumonia) and in a few lobules (p. lobularis, p. insularis or nodosa) rather than throughout a lobe, its exudate is rather catarrhal (p. catarrhalis) than fibrinous, and its distribution is usually multiple and often disseminated (p. disseminata); (*c*) *clinically* it is identical with capillary bronchitis, which, if it occurs in the first years of life, almost never spares the lung tissue; it is marked by fever, cough, dyspnœa and cyanosis, which may often co-exist without obvious signs of hepatization.

Etiology.—(*a*) Some cases, especially in children under two years, are *primary*; these are estimated by Holt at 35 per cent., by others at a much lower figure; (*b*) most cases are *secondary* to infections affecting the upper air passages. In 279 cases of infantile bronchopneumonia reported by Samuel West, 32 per cent. followed measles, 24 per cent. pertussis, 16 per cent. diphtheria, 15 per cent. macrobronchitis, 7 per cent. ileocolitis, 3 per cent. scarlatina, 2 per cent. influenza, ½ per cent. each varicella and erysipelas. (*c*) *Predisposing causes* in the young are poor sanitation, malnutrition, syphilis, tuberculosis (scrofula), and rickets. Bronchopneumonia causes 9 per cent. of deaths in foundling asylums (Müller). (*d*) *Age* is an etiological factor. Most cases are observed in children two or three years old. Seventy-five per cent. of cases of pneumonia in children under five years of age are lobular (Holt). It is less frequent in adults; in weak, cachectic, nephritic and aged persons it may cause death. (*e*) In some cases microbes may act directly on the lungs, as in the primary cases in children, in the influenza of adults, and in aspiration or inhalation pneumonia. "Schluck pneumonie" is prone to develop in diseases in which the laryngeal sensibility is decreased as in diphtheritic pareses or bulbar palsies; in ether or less often chloroform anæsthesia in which the germ-laden saliva is drawn into the lungs; in comatose states, as apoplexy, or uræmia; in mental diseases; in the new-born; in cases of partial drowning; in operations on the throat, as tracheotomy or those for foreign bodies or cancer the larynx or œsophagus; or when there is persistent vomiting. Smok vapors, gases and dust are exceptional causes. (*f*) It is probable th *infection* may sometimes occur by the *lymph or blood streams*, especial in septic affections.

Bacteriology.—Bronchopneumonia is no specific bacteriologic disease. According to Netter, *in adults* about one-third of the cas show more than one organism, among which the pneumococcus ran first and the streptococcus second; and about two-thirds show one ger of which the pneumococcus constitutes 39 per cent., streptococcus per cent., Friedländer's bacillus 23 per cent., and the staphylococcus per cent.; *in children* mixed infection is present in half the cases, t streptococcus predominating, while in the other half there is but o organism, the pneumococcus being the most common. Others, Deurck, maintain that most cases show mixed infection. Other ger found are the influenza, typhoid, tubercle and colon bacilli, Pfeiffer Micrococcus catarrhalis, Micrococcus tetragenus, and meningococcu The tubercle bacillus is often associated with the pneumococcus.

Pathology.—The foci of consolidation are diffused usually in t lower and sometimes in the upper lobes, posteriorly. They vary size from that of a pin head to that of a walnut, or may be larger; ma or most foci lie near the pleura, through which they may often be see and felt; the pleura is often granular or slightly fibrinous, sometime ecchymotic. Foci are usually bilateral. On section the consolidate nodes appear reddish-brown and later yellowish from fatty degeneration of the cells; they are firm, airless, whence they sink in water, and on section are generally smooth and glistening, though some cases of pneumococcus infection show the same granulation and viscid secretion which are present in genuine fibrinous pneumonia. The bronchioles supplying the consolidated nodes are plugged with muco-pus, so that air cannot be forced through them; the bronchioles are reddened, swollen, sometimes ecchymotic or dilated. Many parts are collapsed, which some consider is preliminary to consolidation, but though atelectasis is frequent its importance has been overestimated. In fortunately made sections a dendritic racemose or grape-like arrangement may be seen in which the bronchioles correspond to the stems and the alveoli to the leaves or grapes. *Microscopically* the bronchioles show dilated vessels and leukocyte emigration, the alveoli show epithelial desquamation and proliferation, serous exudation, leukocyte outpouring, a scattering of red cells and but little fibrin formation; the alveolar walls show much interstitial inflammation. It is usuall stated that no fibrin is found, but this is a mistake; though influenz pneumonia is strictly cellular (catarrhal), aspiration, pneumococcic an other forms not infrequently show a fibrin network, though decidedl less than is the case in lobar types. Near the foci and in the upp lobes compensatory emphysema often develops. The bronchial lymp glands are hyperæmic, swollen and succulent. *Extension* occu directly from alveolus to alveolus or along the peribronchial connecti issue. In some forms numerous lobular foci may increase in size and erhaps fuse so that the greater part of a lobe may be consolidated; his form, known as the pseudolobular, is often due to the pneumococus, is often seen in measles and diphtheria and may clinically present close resemblance to lobar pneumonia, though pathologically areas f collapsed, dark vascular, or sound tissue lie between the bronchoneumonic patches.

Symptoms.—The clinical picture varies considerably, accordingly as he bronchopneumonia is primary or secondary. *In the primary form,* which is most often due to the pneumococcus, the onset is brusque, with chill, convulsions, vomiting, or pain in the side. The fever rises suddenly, remains high, and often falls by crisis. Capillary bronchitis is absent and the consolidation is often marked. The course is short and the death rate is low. In adults this form is frequently unilateral. The *more common secondary form* begins insidiously, especially if it follows a microbronchitis; it is often obscured by the disease which it complicates. The fever gradually rises to 103° or 104° (if fever were already present), and is generally irregular or remittent, rising with the formation of each new focus; it may be low or absent in marantic subjects. There may be no physical findings, or if present they are frequently indeterminate. If recovery ensues, lysis is the usual solution. Von Ziemssen held that fever in bronchitis seldom exceeds 102° and that higher registrations indicate pneumonia.

Subjective and General Manifestations.—These are often *more obvious* than the objective or pulmonary symptoms. The pulse rises to 120 or even 180 and in convalescence is more tardy in reaching normal than is the fever. The respiration is often 60 or 80, the expiration is frequently catchy or grunting, and the respiration ratio, normally 1 to 4, becomes 1 to 3 or 2; dyspnœa develops early, and with it restlessness and irritability. Cyanosis develops in the face and then in other parts from impaired flow of the blood to the heart which is due to continued coughing and to stagnant secretion. The cervical veins are prominent. Pain is not as common as in lobar pneumonia. The cough becomes dryer and more distressing; the sputum is usually swallowed in patients under seven years of age, but if obtained shows nothing characteristic, though von Niemeyer found that it sank in water or floated under the surface. Meteorism is not uncommon in severe or moribund cases.

Lung Findings.—On *inspection,* irregular excursion is sometimes seen. The accessory muscles of the nose, neck, chest, and abdomen are in full play. The interspaces, ribs, sternum, and epigastrium in young plastic chests, retract greatly with each inspiration; this must not be confused with the slight physiological retraction observed in the beginning of inspiration. *Palpation* may show increased fremitus in a focus near the surface and measuring 2 x 5 cm. Stagnant secretion in the air tubes may temporarily suspend fremitus. *Percussion* is often negative for a few days or even throughout the course, as it is obvious that deep, isolated foci cannot produce dulness. In the thin, infantile thorax, percussion must be made lightly and special importance should be attached to palpatory percussion. Dulness obtains over a focus having an area of 2 x 5 cm. It is most often found along the spine over the lower lobes but sometimes also over the scapular spines. Dulness is most marked in the pseudolobular (confluent) forms. Atelectasis may cause dulness but not bronchial breathing. A hyperresonant note in front indicates emphysema; behind it shows relaxation. *Auscultation* reveals subcrepitant or crepitant rales; they are often heard over an area not exceeding the bell of the stethoscope. Bronchial breathing and bronchophony are fairly frequent, and occur especially when the child cries. In other cases the breathing may be rude or puerile.

Special types have been considered under measles, pertussis, etc. Abt (following Holt, Carmichael, Ashby and Wright), offers the following pathological classification, which "undoubtedly gives the clearest basis for an understanding of the physical findings: (*a*) In *capillary bronchitis* there is no sign of consolidation in any part of the lung. Sibilant rales, coarse and fine crepitant rales are distributed over one or both lungs. The sibilant rales are due to congestion and swelling of the mucous membrane of the larger and smaller tubes. A feeble respiratory murmur indicates areas of congestion. (*b*) In *disseminated bronchopneumonia*, which is characterized by small areas of consolidation, percussion usually gives negative results, though sometimes there is very slight dulness. The vocal fremitus is usually not altered. As a rule the consolidated areas are situated deeply in the lung, or are too small to cause any appreciable difference in the percussion note. Auscultation reveals crepitant rales and bronchovesicular breathing over the consolidated areas. Vocal and crying resonance is exaggerated. (*c*) In *acute generalized bronchopneumonia*, the percussion note is dul over a large area. Even an entire lobe may be involved. Palpatio shows an increase in vocal fremitus. Auscultation reveals bronchi breathing, and fine moist rales are heard over the dull areas, while th rest of the lung shows evidence of bronchitis."

Issues.—*Death* may occur from carbonic acid narcosis, irritabili passing into apathy, the harassing cough becoming less marked a causing stagnation of bronchial secretion in the large tubes ("suffoc tive catarrh"); delirium develops, the pupils narrow, and Cheyn Stokes breathing and heart failure result. *Complete resolution* is t rule in cases which recover. *Delayed resolution* with remissions a exacerbations may occur. *Complications* may develop; (*a*) pleurisy the most frequent; (*b*) gangrene; (*c*) abscess, especially in aspiratio and influenza forms; (*d*) pulmonary induration; (*e*) bronchiectasi (*f*) tuberculosis, which is less often a sequel than a previous disea awakened by the new infection; (*g*) pericarditis, endocarditis, otiti emphysema, and fibrinous bronchitis are other possible complication

Diagnosis.—Reliance on physical signs alone leads to many erro and a diagnosis is often determined by the development of sudden hig fever or mild fever suddenly rising during bronchitis, by dyspnœ irregular rapid respiration, cyanosis, and pain in the chest. In ver young or marantic children and in the aged the onset and sympto may be very atypical. *Lobar pneumonia* (*q.v.* variations in children) occurs mostly in sound children over three years of age, runs a shorter cyclical typical course, ends by crisis, is unilateral and involves the greater part of one or more lobes; while bronchopneumonia occurs more often in younger weakly children, is often a sequel of bronchiolitis, runs a longer, more atypical, unfavorable and relapsing course, resolves by lysis, is bilateral and is less marked by definite pulmonary findings. *Tuberculosis* is often distinguished only by the course; it invades the upper lobes more than does bronchopneumonia, and bacilli-laden sputum may be recovered by washing the stomach or by swabbing the throat when the child raises mucus after coughing. *Pleurisy* (*q.v.*) is diagnosticated by means of the aspirating needle.

Course and Prognosis.—The *course* may be (*a*) *acute*, lasting about a week, of which the measles-pneumonia is the prototype; (*b*) *subacute*, lasting from two to eight weeks, exemplified by the pertussis-pneumonia, or (*c*) *chronic*, covering months, as in tuberculous bronchopneumonia.

The *prognosis* depends on the patient's social situation, previous history, age, the disease of which the pneumonia is a complication, and the extent of the pneumonia. The outlook in general is serious, especially in secondary forms, among which measles and pertussis represent the more serious types; in the very young it is almost as fatal as infantile diarrhœa; aspiration forms are very serious, as are those in marantic and aged subjects. In private practice the death-rate ranges from 10 to 33 per cent., in hospitals 33 to 50 per cent., and in asylums even higher (50 to 100 per cent.). Early cyanosis, dyspnœa and rapid pulse are unfavorable signs.

Treatment.—1. **Prophylaxis.**—(*a*) In infections which produce nasal or pharyngeal inflammation the nose and throat should be frequently cleaned with mild alkaline antiseptics. (*b*) When there is laryngeal or bronchial inflammation, congestion and atelectasis should be avoided by frequent and regular change of posture. (*c*) Deep breathing should be encouraged in older subjects, or enforced in the young by affusions of cold water on the neck and upper thorax. (*d*) Exposure should be avoided, especially at night by pinning the bedclothes or firmly fastening the nightgown to the foot of the bed.

2. **Therapy.**—Actual treatment is symptomatic, as the varied etiology precludes specific therapy. (*a*) The patient should have plenty of *fresh air*. The temperature of the room should be maintained at 67° to 70° with constant moderated ventilation. (*b*) The diet should be fluid, diluted or predigested; egg albumin, milk and plenty of water are indicated. At the onset or during the course calomel gr. $\frac{1}{10}$ every hour for five or six doses should be given, followed by castor oil, particularly if there is indigestion or tympany. (*c*) *Expectorants*, as ammonium carbonate, tartar emetic or ipecac, should not be given generally, as they derange digestion, and good digestion is of great importance to weak as well as to strong subjects. Aromatic spirits of ammonia, ♏. v to xx, every three or four hours, is least deranging and is a diffusive

stimulant. (*d*) *Pain* rarely necessitates the use of opiates, which pr mote stagnation of secretion; dyspnœa, pain, restlessness, coug cyanosis, and rapid pulse may be relieved by paregoric, which is prefer ble to Dover's powder or morphine; but great care is imperative in t use of any opiate in young, old, or weak subjects. (*e*) *Local applic tions*, as poultices or pneumonia jackets and blisters, are useless. (In the treatment of *fever*, antipyretics, aconite, antimony and oth cardiac depressants should be wholly avoided. Cool applications a equally efficacious; they also lessen tympany and carbon dioxide na cosis, stimulate the heart and brain centres, and deepen the respiratio A cold pack should be applied over the neck, chest, and abdomen a should be changed frequently. Cold must be used with care in ve young and in marantic subjects; in such cases a warm sponge or ba may be given, during which cold water may be poured on the neck stimulate deep breathing. (*g*) For *cardiac stimulants*, whisky a brandy may be given in daily doses of ʒss to ʒiij, very well diluted water or in peptonized milk. Aromatic spirits of ammonia, digitali saline infusions or enemata, camphor or strychnine (gr. $\frac{1}{100}$ every thr or four hours for a child six months old) may be used. Hypodermi of ether may cause sloughing. (*h*) *Respiratory stimulants* inclu tr. belladonna (♏.i. to ii. every two to four hours), oxygen, thorac faradization and mechanical compression of the chest. (*i*) *Emeti* are unreliable; ipecac and similar remedies have no effect when the is pronounced carbon dioxide intoxication, whence it has been recom mended to combine them with coffee, camphor, or cognac. For ca bon-dioxide intoxication phlebotomy should seldom be practised. (*j* *Inhalations of steam* (*v.* DIPHTHERIA and CROUP) may be beneficial compound tincture of benzoin and sodium bicarbonate are ofte added to the vaporizing water. (*k*) *During convalescence* fresh air c a sojourn in the country or in a warm climate is advisable.

INDURATIVE PNEUMONIA, LUNG CIRRHOSIS, FIBROID PHTHISIS.

Definition.—An (almost invariably) secondary overgrowth of t pulmonary connective tissue.

Etiology.—**1. Diseases of the Lung.**—These may induce *local*, lob lar or lobar, unilateral or bilateral fibrous induration. Local indu tion frequently occurs about tumors, parasites, tubercles, gumma abscess, gangrene, trauma, or pneumokoniosis, and is of no essent clincial importance, except as it demarks dangerous foci of disea *Diffuse* induration is important, though decidedly subordinate to t causal disease. (*a*) *Fibrinous pneumonia* sometimes (Charcot) resu in lobar induration; Fraenkel observed it seven times in 1,000 case it occurs notably in marantic or aged subjects and results from delay resolution and lack of regeneration of the alveolar epithelium; t lung becomes airless and dense from development of fibrous tiss within the alveoli whence it involves contiguous structures; its surface on section is smooth, and the connective tissue is strikingly translucent; slight induration may be noted in a lobe from repeated attacks of pneumonia. Subacute induration may begin within two or three weeks and reach considerable development in one or two months. (*b*) *Bronchopneumonia* (Legendre and Bailly, 1844) in the course of measles, pertussis, or influenza, sometimes leads to subacute or chronic induration, which may begin from an alveolitis or peribronchitis. (*c*) *Foreign bodies* and *aspiration pneumonia*; (*d*) *chronic tuberculosis*; and (*e*) syphilis, gangrene, actinomycosis, and glanders are etiological factors.

2. Bronchial Affections.—(*a*) *Bronchiectasis,* which was noted by Corrigan, who was first to use the term lung cirrhosis; (*b*) *bronchial stenosis,* as by aneurysm, or gumma; and (*c*) *pneumokoniosis,* and putrid and chronic bronchitis, may cause indurative pneumonia.

3. Pleural Affections.—The relation of pleurisy to lung cirrhosis is still disputed; some consider it primary to lung induration and others hold it secondary. It seems established that primary pleurisy may sometimes invade and cirrhose the lung; this is the "pleurogenous interstitial pneumonia" of Charcot; strands of connective tissue run into the lung and approach and fuse toward the hilum.

Other causes are uncertain. English writers mention a "fibroid tendency," which is probably not a real cause; diabetes, alcoholism and malaria are also referred to as possible etiological factors.

Pathology.—The pathology of an affection with such numerous causes can scarcely be described. The early induration is reddish, and the older connective tissue is darker, slate colored or gray; to them Charcot gave the names of *induration rouge* and *induration grise,* respectively. The new connective tissue may be formed in the bronchi, septi, alveoli, bloodvessels or pleura. The alveoli nearly always show hyperplasia of the connective tissue. The lung is airless and dense; is cut with difficulty and on section presents either a granulated or a smooth surface, the latter in cases following lobar pneumonia and sometimes in those consecutive to gangrene or aspiration pneumonia. The volume of the lung is decreased. Its shrinking produces changes in the mediastinum, heart, diaphragm and sound lung, which will be considered under SYMPTOMS.

Symptoms.—Incipient or localized lesions are usually undetected unless they are observed in the course of pneumonia or tuberculosis. Symptoms may be *inseparable from the causal affection* (tuberculosis, bronchiectasis) or may follow closely upon it (pneumonia, pleurisy). There may be a low fever, cough, dyspnœa on exertion, pain in the side, rapid pulse, cyanosis, anæmia, malnutrition, and night sweats. In cases where the original malady has ended, there may be only slight cough, or some shortness of breath on exertion.

Physical findings of the typical chronic case are as follows: (*a*) On *inspection* the interspaces are found narrowed on the affected side, the nipple and scapula are nearer to the median line, the shoulder is lower, the spine is convex toward the sound side, and the measurement

aterally decreased. In left-sided induration the
the heart, leaving its pulsation more widely exposed an
g the pulmonary artery, over which may be seen and felt its
lic filling and the strong diastolic closure of the pulmonary
ves; in right-sided induration the heart beat may appear to the
ht of the sternum or may be covered up by the compensatory
nphysema of the left lung. (b) *Palpation* corroborates these findings,
nd, as a rule, shows increased vocal fremitus. The second pulmonic
tone is much accentuated, corresponding to the hypertrophy of the
right ventricle and the occasional atheroma of the artery. (c) *Percussion* elicits dulness below and sometimes a slightly hyperresonant note above the indurated area; the apex of the upper lobe is on a lower level and the diaphragm stands higher (the liver rising or Traube's space increasing); there is no respiratory excursion. (d) *Auscultation* brings out the absence of breath sounds below, and bronchial breathing, and subcrepitant, crepitant, and bubbling rales above. Additional signs result from the right heart hypertrophy and dilatation and from coincident bronchiectasis, or cavity formation on the same side, and the well developed compensatory emphysema on the opposite and otherwise healthy side. There is nearly always some sputum.

Diagnosis.—Diagnosis is more easily made in the very chronic than in the subacute forms; subacute post-influenzal or post-pneumonic induration may easily suggest tuberculosis, or interlobular suppuration. Slow-growing tumors of the lung may cause confusion.

Prognosis.—The prognosis varies with the etiology. Ulcers may form from arterial occlusion or from infected bronchial secretion, whence the frequency of lung hemorrhage (in 50 per cent.) and its occasional role as a cause of death. Amyloidosis sometimes results, but death is usually caused by right heart failure, which may possibly occur during some acute intercurrent disease.

Treatment.—Therapy is unavailing, though life may be prolonged for decades under proper hygiene. Exercises with the arms may check the early shrinking, but nothing can remove an established induration. Warm climates at the sea level or at slight elevation are beneficial
Some writers hold that turpentine is of benefit in checking induratio

PNEUMOKONIOSIS.

Zenker employed the term to include various indurative disea
the lungs due to "dust" inhalation.

Etiology and Pathology.—1. *Anthracosis* is due to inhala
carbon, coal dust, soot, charcoal or graphite, and is known
miner's disease." (a) Moderate amounts of carbon inhaled
trachea and bronchi are absorbed by the leukocytes, are w
ward by the ciliated epithelial cells and are expectorated.
ance varies greatly. (b) Larger amounts penetrate th
lodge in the connective tissue, or enter the ly

whence they reach the small lymph nodes around the bloodvessels, the bronchi, the pleura, or the mediastinal lymph glands. A moderate amount of lung pigmentation occurs in all city inhabitants, while the lungs of countrymen are pink and devoid of soot deposit. It is possible that the lymph nodes may later throw out some of the pigment into the bronchial lumen. (*c*) Very large amounts may reach the alveoli. In these cases the lungs are ink-black. When they are, as it were, saturated, connective tissue proliferation develops (endoperialveolitis nodosa and endoperilymphangitis fibrosa) in *insular foci*; this is sometimes called fibrous bronchopneumonic induration because of its localization; it occasionally measures 5 x 15 cm.; on section the foci are hard and exude an inky fluid. *Diffuse induration* is common. The *mediastinal lymph glands* are *indurated*, and the process often extends outside the glands (perilymphadenitis); by this latter process the glands may become adherent to and rupture into the pulmonary veins, whence pigment-metastases into the liver, spleen, kidneys, and mesenteric glands are not infrequent. Bronchial perilymphadenitis may also lead to mediastinopericarditis, to stenosis of the trachea, bronchi, œsophagus, cava, vena azygos, and pulmonary artery, to œsophageal diverticulum, to fatal hemorrhage into the pericardium or into the bronchi when the aorta is also eroded, to aspiration pneumonia by intrabronchial rupture or to vagus or recurrent laryngeal paralysis by pressure.

2. *Siderosis pulmonum* (Zenker) is a very similar change, due to inhalation of mineral dusts, as those of iron, brass, or bronze; there are the same interstitial deposit, lung induration, bronchial gland cirrhosis, and metastases. The irritation is greater, the nodes are more frequent and the induration is more intense than in anthracosis. The iron deposits are yellow or red from the oxide, or black from the phosphate. The lungs may contain 1½ per cent. of iron.

3. *Chalicosis pulmonum* (Peacock) is due to inhalation of alumina, quartz or sandstone, and is known as "stone-cutter's or mill-stone-maker's phthisis," "grinder's rot" or "potter's asthma." Inhalation of calcium is less irritating than of silica or quartz. Of all forms, the most nodules and the maximal induration occur in chalicosis.

4. Similar changes are observed from inhalation of wool, flax, cotton, grain, tobacco, glass, porcelain, cocoa, pepper, cinnamon, bone, and horn. Claisse and Jossué place special stress on bacterial coöperation.

Symptoms.—These may appear only after many years, when the symptoms of *chronic bronchitis*, *asthma*, *emphysema*, and finally of *lung cirrhosis* appear. The *sputum* contains coal dust, soot, charcoal particles, as in Traube's celebrated case, and yellow oxide or dark phosphate of iron, quartz, and silica. *Cavities* may form from bronchiectasis, from softening of the nodose indurations or from tuberculous complications; in some cases lung stones, set free by ulceration, may be raised. The mediastinum may be exposed by retraction of the lungs, due to fibrous mediastinitis. Tuberculosis is common, occurring, according to Phillip, in 13 per cent. of anthracosis and in 44 per cent. of chalicosis.

Scissor-grinders rarely live beyond forty years of age and those employed in blasting mines usually succumb in four years.

Treatment.—Treatment is largely prophylactic and is that of the concomitant bronchitis and emphysema.

ATELECTASIS.

Atelectasis is a congenital or acquired "absence of air." It is also called apneumatosis.

Congenital Atelectasis.

1. The congenital atelectasis, first described by Joerg (1834), who differentiated it from pneumonia, is the persistence of the normal fœtal condition of the lungs; it develops in various forms of dystokia, and in weakly or premature infants.

Etiology and Pathology.—This variety is due to deficient inspiratory efforts from weak muscles, insufficient stimulation of the medulla centres, and aspiration of mucus or meconium. The base, lower anterior margin, and lingual lobe are most involved and the distribution is lobular. The color of the lung is a steel-blue, and if the subject lives, the atelectatic portions do not become pigmented, for no air enters them. The airless areas are superficial, sunken, lax, do not crepitate, and sink when put in water; in the early stage the part can be distended by inflation of the bronchus supplying it and on section exudes a serous or slightly blood-stained fluid. Cylindrical and cystic bronchiectasis may result; the ductus Botalli and foramen ovale are usually patent, the right heart is commonly dilated and thrombosis is frequent in the brain sinuses, kidney, and right heart.

Symptoms.—The new-born who have atelectasis, breathe superficially and cry but little. Inspiration is marked by retraction of the epigastrium, subclavicular and intercostal spaces, for the lungs cannot follow the inspiratory distention of the thorax. If the undistended area measures 2 by 5 cm. it may be dull and increased fremitus with bronchial breathing may be detected, but more often the breathing is weak or entirely absent. Carbon dioxide narcosis develops, the pulse becomes small, the sensorium becomes dulled and, without active intervention, asphyxia with spasms causes death. If the patient lives, the chest is peculiarly deformed, its lower parts being sunken, so as to show the ribs. Koestlin describes a total collapse of one lung in a man aged twenty-two.

Treatment.—Removal of meconium and mucus by swabbing or aspiration through a catheter, Silvester's artificial respiration, Schultz's swinging, faradization, and warm baths with cold affusions to the neck and upper chest are indicated.

2. Acquired Atelectasis.

Acquired atelectasis in children was first described by Legendre and Bailly (1844), and that in adults by Traube.

Etiology.—It may be due to marantic causes, to obstruction or compression; the lungs, already distended and ventilated, collapse and the air contained is absorbed. (*a*) The marantic form occurs in individuals reduced by typhoid, infantile diarrhœa or cachectic conditions, and by lying for a long while in the dorsal decubitus. (*b*) Obstructive collapse develops most frequently in capillary bronchitis (bronchopneumonia), as the bronchioles in the young are disproportionately narrow; it may develop in all conditions which promote laryngeal, tracheal, or bronchial stenosis. These forms affect the posterior parts particularly. (*c*) Compressive apneumatosis follows compression by cardiovascular conditions (aneurysm, dilated heart, and exudative pericarditis), by pulmonary tumors, pleural exudates or pneumothorax, by mediastinal tumors or adenopathies and by abdominal distention (tumors, ascites or meteorism). The part of the lung involved depends on the direction of the compression. In kyphoscoliosis permanent compression of the lung frequently leads to atelectasis, and if it occurs early in life, to hypoplasia.

Pathology.—Pathologically the same general findings obtain as in the congenital type. The lung is generally darker, red-brown or bluish. Bronchiectasis may develop after a time. In the congenital and acquired types, hyperæmia may cause a flesh-like appearance (carnification), or serous transudation may cause a spleen-like aspect (splenization). There may be coincident inflammation or, in the healthy parts, vicarious emphysema. Long-standing atelectasis results in fatty degeneration of the alveolar epithelium and hyperplasia of the interalveolar and interinfundibular connective tissue, known as collapse induration.

Symptoms.—Symptoms are easily obscured by pleural effusion, and by the causal bronchopneumonia, with whose foci it may be confused. Inspiratory retraction, lessened vesicular breathing, a slightly tympanitic note from relaxation of the collapsed lung, and crepitant rales are common. The vocal fremitus is seldom increased, and bronchial breathing is uncommon, unless the foci are both large and superficial. Dilatation of the right heart is common. In recent cases the *physical signs disappear after a few hours of change of posture, or after deep breathing* either voluntary or induced by cold affusions; this, with absence of fever, pain, and cough, serves as an effectual differentiation from infarct, pneumonia, or effusive pleurisy. After one has slept for some hours on the back, a few crepitant rales may be heard over the lower posterior parts for three or four inspirations; this is quite physiological.

Prognosis and Treatment.—The prognosis and treatment vary according to the cause. Sudden death is not uncommon in persons

having deformed spines. In all fevers, as typhoid, and in all respi
ratory inflammations, the posture must be changed; orders to thi
effect should be written, and the change should be recorded on th
history sheet. Cold affusions to the neck, Brand's hydrotherapy an
cardiac stimulants are indicated.

ABSCESS OF THE LUNG.

Suppuration is not a common lesion, but may occur in a sound o
diseased lung.

Bacteriology.—The staphylococcus is most frequently the causativ
microörganism; the streptococcus, pneumococcus, pneumobacillus
influenza bacillus, colon bacillus, and leptothrix are also found.

Etiology.—(*a*) *Lobar pneumonia* causes 62 per cent. of the case
(Tuffier); localization in an upper lobe, alcoholism, marasmus, pneu
monia complicating emphysema and lung induration, and hemorrhagi
types are the predisposing factors; abscess of the lung is somewha
analogous to the necrosing interstitial pneumonia in cattle and horses
(*b*) *Lobular pneumonia* may suppurate, especially in the influenzal type
(*c*) *Aspiration pneumonia* is causal in 18 per cent. (Tuffier), partic
ularly in insane or delirious persons; foreign bodies, food, saliva, pu
from lesions in the upper respiratory tract, severe vomiting, œsophagea
cancer or diverticulum rupturing into a bronchus, are its usual causes
(*d*) *Embolic suppuration* produces 17 per cent. of the cases. (*e*
Trauma is an etiological factor. (*f*) Abscess may complicate othe
pulmonary or bronchial affections, as tuberculosis, glanders, actino
mycosis, echinococcus, or bronchiectasis. (*g*) *Rupture into the lun*
of contiguous foci of suppuration, such as empyema, suppurating per
bronchial glands, subphrenic abscess, spinal caries, and gastric ulce
is the cause of 3 per cent. of lung abscesses. Most of the cases occu
in middle-aged males.

Pathology.—The cavity varies from the size of a hazelnut to th
of an orange; in rare instances it may occupy an entire lobe or lun
The abscess is usually single, except in the embolic form. Its sha
is irregular and it is sometimes multilocular. Its inner surface
irregular with floccular mural shreds, and is walled with pus-secreti
granulations, which in some cases may fill and obliterate the cavit
Fibrous tissue develops about the cavity, and in chronic abscess b
comes dense and scar-like. The cavity is usually near the lung
surface, whence the frequency of complicating pleurisy; 80 per cen
of lung abscesses are in a lower lobe (Tuffier).

Symptoms.—The development of symptoms varies according
the causal factor; in lobar pneumonia the crisis or lysis is delaye
and resolution is incomplete; in influenza the respiratory sympto
and fever persist; in sepsis, multiple lung involvement perpetuate
the septic process. (*a*) *The sputum* is purulent, creamy, is ofte
offensive or rancid without putridity, is alkaline and may measure

pint to a quart daily. It separates, on standing, into two layers, a granular sediment and a serous upper layer, sometimes with frothy surface if the coughing is intense. In metapneumonic cases the sputum is sometimes grass-green. The sediment almost invariably reveals *shreds of lung tissue* (Traube and Leyden), and yellowish-green or gray flocculi; these are absolutely essential to diagnosis, and consist of elastic fibers to which alveolar epithelium adheres. *Hæmatoidin* crystals or platelets, short sheaths of *fatty crystals, bacteria* (*v. s.*), and in chronic cases *cholestearin* and scar-like connective tissue are also found. The sputum is voided periodically, as from cavities of other causation; "mouthful expectoration" is the rule. (*b*) The *decubitus* varies with the location of the cavity; for instance, the patient assumes the erect posture if the cavity is in a lower lobe, to prevent leaking of the secretion into the contiguous air passages. (*c*) *Signs of cavity* are found if the exulceration is large and superficial; 80 per cent. of them occur in the lower lobes. They are essentially those of tuberculous or bronchiectatic cavities (*q. v.*) and vary with the stagnation or evacuation of the pus; thus a tympanitic note, bronchial or amphoric breathing, and increased vocal fremitus are found when the *vomica* is empty, and dulness, weakened breath sounds and decreased fremitus are present when it is full. The *x*-rays also serve to localize, but when their findings and the physical signs differ the latter are more trustworthy. (*d*) *Hectic fever,* sweats, rigors and leukocytosis occur from resorption.

Diagnosis.—The etiology and cavity formation are often, and the sputum is always, characteristic. (*a*) *Abscess breaking into the lung* from the spine, or from the subphrenic or pleural spaces, is sudden, sometimes suffocative in onset, and the hæmatoidin crystals and shreds of pulmonary tissue are absent. (*b*) *Gangrene* (*v. i.*) is a factor in differentiation. (*c*) *Tuberculosis* usually occurs in an upper lobe; abscess in a lower one; elastic fibers and hæmatoidin crystals are more abundant in cases of abscess, and tubercle bacilli are found in tuberculosis, at least after several examinations. (*d*) In cases of *bronchiectasis* elastic fibers are seldom voided.

Prognosis.—Spontaneous recovery may follow granulation and cicatrization, especially in lobar pneumonia, where recovery is the rule. In influenzal or aspiration pneumonia or in sepsis, the outlook is far less favorable; marasmus, hectic fever, amyloidosis, lung induration, empyema, pneumothorax and pericarditis are frequent issues.

Treatment.—(*a*) The *general strength* should be maintained by giving a full diet, tonics, and alcohol. (*b*) *Decomposition,* if present, should be modified as in bronchiectasis or gangrene, by administration of creosote or turpentine internally. (*c*) *Surgical intervention* is indicated, according to Fraenkel, (i) when the abscess is large, shows no tendency toward spontaneous cure and the contents decompose; (ii) when rupture into the pleura occurs; and (iii) when the abscess results from the breaking into the lung of a contiguous focus of suppuration. Of Korte's 12 operative cases, 83 per cent. recovered; Garré's 96 cases, 80 per cent.; in Tuffier's series, 77 per cent.; Freyhan's acute cases, 90 per cent.; and in his chronic cases but per cent.

(G). GANGRENE OF THE LUNG.

Definition.—A necrosis and mortification of lung tissue. Gangrene, though more common than abscess, is an infrequent affection; Eichhorst found gangrene in 0.1 per cent. of his clinical cases and Hensel in 1.6 per cent. of his autopsies.

Bacteriology.—Some physicians maintain that certain germs produce the necrosis and other germs the gangrene, but others hold that one microörganism may produce both changes. The staphylococcus is found in its various forms, also the streptococcus, colon bacillus, and pseudotubercle bacillus which retains its stain when treated with acids like the bacilli found in putrid bronchitis and bronchiectasis; the Bacillus pyocyaneus, and the Micrococcus tetragenus are also found; Hirschler and Terray have cultivated a micrococcus which liquefies gelatin and produces skatol and indol, and therefore causes a putrid odor in the cultures. Babes described bacilli resembling those of malignant œdema. The leptothrix, oïdium albicans, spirilla, sarcinæ and monads are probably of but secondary importance.

Etiology.—Decidedly reduced physiological resistance, *e. g.*, from diabetes, is apparently a necessary etiological factor. *Lobar pneumonia* may terminate in gangrene when, as in abscess, the patient is weakly, diabetic, addicted to alcohol or suffering from putrid bronchitis; its frequency is not great, for it occurs in but 0.4 per cent. (Fraenkel) or in less than 1 case in 1,500 pneumonias (Aufrecht); pneumonia causes 23 per cent. of lung gangrene (Coupland and Hensel). *Lobular pneumonia* is less frequently causal, though grippal pneumonias may terminate in gangrene (7 per cent. in Fraenkel's series). In Coupland and Hensel's combined cases, 14 per cent. were due to *pulmonary tuberculosis*, and 10 per cent. to *tumor of the lung.* Gangrene may complicate lung abscess or echinococcus. *Aspiration pneumonia* resulting in gangrene occurs particularly in insane, delirious, paralyzed, or emaciated subjects; foreign bodies, necrotic diseases of the throat or bronchi, laryngeal perichondritis, bronchiectasis, putrid bronchitis, and bronchial stenosis promote gangrene. *Embolic gangrene,* a cause of 19 per cent. of lung gangrene, may result from suppurative gangrenous lesions in peripheral parts, as in or about the appendix, uterus, or extremities. *Rupture into the lung,* as of œsophageal cancer or diverticulum, putrid pleurisy, subphrenic abscess, or gastric ulcer may cause gangrene. *Trauma* is an occasional promoting factor. Gangrene usually occurs within the *second to fourth decades* of life and chiefly *in males.*

Pathology.—Genuine gangrene must not be confused with softening of the lung, which is due to regurgitation of the gastric juice into the lung during the death agony or after death. In rare cases, too, a non-putrid pneumomalacia, analogous to myomalacia cordis, is observed. Lung gangrene, according to Hensel, occurs in the right lung in 45 per cent., in the left in 37 per cent., and in both lungs in 18 per cent. of the cases. Laennec, who first described pulmonary gangrene, distinguished *two types*, the circumscribed and the diffuse. (*a*) *Circumscribed* gangrene occurs chiefly in the lower lobes and toward the surface, rarely in the interior and oftener on the right than on the left side. The focus may attain the size of an orange. Laennec distinguishes three stages. (i) The formation of a yellow, green or brown slough, which may be expectorated entire; (ii) liquefaction, or moist gangrene; and (iii) formation of a cavity which is irregular in shape, has ragged walls, and contains a stinking liquid resembling the sputum of gangrene (*v. i.*). The cavity may be limited by granulations and later by connective tissue, or may gradually extend, corroding more and more lung tissue. Extension may also result from aspiration of the septic matter from the bronchi into other lung areas. Recovery may follow localization, organization of granulations, and contraction of connective tissue. Microscopically there is (i) an inner zone of offensive fluid or necrotic tissue, bacteria and emigrated leukocytes; (ii) outside of this is a zone of granulations, beyond which is (iii) congested or œdematous lung. (*b*) The *rarer diffuse gangrene* has no demarkation, and a large area, an entire lobe, or even the whole lung may be macerated, stinking and discolored. The diffuse form occurs more often in the upper lobes, and in the right more frequently than in the left side. In both forms the sputum irritates and injects the bronchial mucosa, the bronchial glands are usually tumefied and degeneration or metastasis may occur in various viscera.

Symptoms.—1. The *sputum is characteristic;* its presence is almost absolutely essential to a diagnosis, and it is absent only when the putrid focus has no bronchial connection. Its odor is extremely offensive, fæcal, stinking, garlicky, or sometimes sweetish. This odor is less marked in diabetic gangrene; it is most offensive at the moment of evacuation, and on standing may gradually lessen. The sputum, which is *voided periodically,* and sometimes in mouthfuls, is usually abundant (a pint to a quart), is alkaline, and on standing separates into three layers, an upper layer of foam, a middle one of green or gray serum, and a lower one of green or brownish granular sediment. In the sediment various important structures are found; there are *flocculi* of lung tissue, which are seen best against a dark background, and which vary in size from small particles to pieces as large as the thumb and show alveolar arrangement; elastic fibers are not nearly as common as they are in abscess of the lung, unless the process is very acute, for a trypsin-like ferment rapidly digests the fibers; *Dittrick's plugs* are usually found, consisting of detritus and bacteria, and emitting a most fœtid odor (described under PUTRID BRONCHITIS AND BRONCHIECTASIS); *long fatty needles* (Virchow), lung pigment, pus cells, red corpuscles, hæmatoidin, bacteria, infusoria, triple phosphates, leucin, tyrosin, formic and other allied acids, phenol, indol, skato ammonia and sulphuretted hydrogen are also found.

2. *Chest findings.*—Besides the findings of *bronchitis* caused b the offensive sputum, signs of infiltration or cavity formation are ol tained if the focus is superficial and measures two and a half inche In the *common circumscribed form* the signs of cavity are identic with those in tuberculosis, bronchiectasis or abscess, *i. e.*, a tympanit note, increased vocal fremitus, bronchial or amphoric breathing, an metallic rales are obtained when the cavity is empty, or dulness, d creased fremitus and weak breathing, when it is full. In the *ra* *diffuse form* there are signs of infiltration as in pneumonia, whic signs are also found in the initial stage of the circumscribed gangren Pulsation of the gangrenous lung is very rare. The x-rays are ofte valuable in detecting the localization of the gangrene. The patier lies so that the cavity is dependent, in order to avoid constant tricklin of the cavity secretion into the bronchi, *i. e.*, on his back when it posterior, or on his face when it is anterior or in an upper lobe.

3. *General symptoms* result from the causal disease or from sepsi Remittent fever, chills, sweats, prostration, emaciation, pallor, rapi bounding pulse, anorexia, vomiting, diarrhœa, leukocytosis, albumin uria and in severe cases a profound typhoid state mark the degre of intoxication.

4. *Complications* may dominate the clinical picture. (*a*) Pleuris (in 12 per cent.), usually purulent or putrid, may be one of the chi symptoms, and operation may evacuate most offensive fluid with larg fragments of necrotic lung. (*b*) Hæmoptysis is seldom fatal thoug frequently profuse, and the blood raised may be dark brown. (Pyopneumothorax, or (*d*) rupture into the thoracic wall, œsophagu aorta, mediastinum, pericardium, or peritoneum may occur. (*e*) The may be metastatic deposits in the brain, liver, joints, or muscles. (Clubbed fingers occasionally result from septic absorption. (*g*) Am loid degeneration of the liver, spleen and kidneys sometimes resul from chronic gangrene.

Course and Prognosis.—The course may be acute, subacute chronic; in general it is more rapid in diffuse (usually fatal within week), and more chronic in circumscribed forms (weeks to even thr months). The prognosis depends on the etiology, the physical streng of the patient, the degree of absorption, localization and surgic accessibility (*v.* THERAPY).

Diagnosis.—Exploratory puncture is dangerous, for it favors d fusion. (*a*) In *fœtor ex ore* the breath is fœtid but the sputur if any, shows no characteristic findings of gangrene. (*b*) In *putr* *bronchitis* the sputum shows Dittrich's plugs and fœtor, but no fat crystals, elastic fibers, nor pulmonary tissue. (*c*) In *abscess* elast fibers and pus are more abundant, the fatty crystals are oftener sheaths than in long needles, and hæmatoidin crystals are more con mon; bronchial plugs or fœtor are absent. (*d*) In *bronchiectasis* elasti fibers are very rare and lung tissue has never been found. (*e*) I *tuberculosis* coincidence of the two affections and the pseudotubercle bacillus (*v.* TUBERCULOSIS) may alone cause confusion.

Treatment.—1. Surgical.—Expectant treatment results in a mortality of 53 per cent. (Eichhorst) to 80 per cent. (Lenhartz). In 15 personal cases (a small number), 10 (66 per cent.) recovered under medical treatment. With operative treatment Tuffier gives the death-rate in 72 cases as 40 per cent., and Freyhans in 26 cases, 30 per cent.; Garré in 122 cases records 66 per cent. of recoveries and 34 per cent. of fatalities. The indications for treatment are (*a*) the vital indication when acute gangrene assumes the *foudroyant* type, or (*b*) when, in chronic forms, there is no tendency toward spontaneous healing. Bull reports recovery even in multiple gangrene. Accurate localization by physical findings or by the *x*-rays, is of course conditional and was possible in 65 per cent. of Tuffier's series of 74 cases.

2. Expectant Treatment.—This is indicated in deep-seated gangrene, in hopeless metastases as those of the brain, or in apparent demarkation. The patient should be kept on his back to avoid leakage into sound bronchi. The diet should be sufficient to support the strength and should include full alcoholic stimulation. Carbolic acid, balsam of Peru or creosote may be given internally. Turpentine is most efficacious when there is a complicating hemorrhage (Skoda).

TUMORS OF THE LUNG.

1. Carcinoma.

Cancer of the lung may be primary or more often secondary to that of the mamma, or of the digestive or genital organs. It occurs in 0.17 per cent. of autopsies or in 1.8 per cent. of cancer autopsies (Paessler, quoted by Fraenkel). Most primary cases develop between forty and sixty years of age. In primary forms the sexes are equally involved though Paessler found 73 per cent. in males; the secondary forms are frequent in women.

Etiology.—Trauma is an uncertain factor. Cancer usually develops in the bronchi, in which previous ulceration, syphilis or adherent and rupturing lymph glands are predisposing factors. Carcinoma probably may develop from the bronchial mucous glands or even from the alveolar epithelium. Histologically the cylindrical type is by far the most frequent, though medullary, scirrhous and other forms may be encountered. Cancer usually begins at the hilum, and may extend toward the pleura, either by contiguity along the bronchi or by the lymph spaces. The upper lobes are more often involved than the lower ones, and the right twice as frequently as the left. The disease is generally unilateral. In 85 per cent. of cases metastases occur in the liver, bones, brain, mediastinum, testes, and kidney. Secondary cancer is generally bilateral; the lungs are strewn with miliary nodules resembling miliary tuberculosis, or present numbers of larger nodules. Calcification and ossification may occur in tumors of the lung.

Symptoms.—When there is a primary accessible tumor in another part of the body, the appearance of cyanosis, dyspnœa, blood-stained sputum, and signs of lung infiltration or of cavity, facilitates a diagnosis of lung metastasis. When there is a hidden primary focus or a primary lung tumor, diagnosis is often for a long time difficult or impossible.

1. **Early General Symptoms.**—These are usually ambiguous; they are suggestive of tuberculosis, pleurisy, or bronchitis, all of which may indeed complicate neoplasm. Thoracic oppression, asthmatic attacks, stridor and *dyspnœa* result from pressure on the trachea and vagus or wide substitution of tumor for lung tissue. Cyanosis, dyspnœa, rapid breathing, fever, emaciation and sweats may suggest miliary tuberculosis; this resemblance may also be noted at autopsy in miliary carcinosis. Fever is, however, not common. Actual pain is usually pleuritic. Cough is common.

2. **Sputum.**—The sputum in over 50 per cent. of cases is rather characteristic, though it may show similar changes in tuberculosis and other affections. Altered blood gives it a reddish-brown color. It may resemble raspberry jelly in hue and in consistency; it is sometimes grass-green or prune-juice in color. Occasionally macroscopic and microscopic bits of cancer tissue, in characteristic nest-like arrangement, are seen.

3. **Physical Signs.**—(*a*) *Inspection:* the patient usually lies on the affected side to give freer play to the sound lung. Marked *ectasia* may result; sometimes the diseased side is shrunken from complicating lung cirrhosis or atelectasis. Unilateral retraction with other signs indicates bronchial stenosis. (*b*) *Palpation* elicits *increased fremitus* if the tumor grows into the bronchial lumen or compresses it from without. (*c*) *Percussion* gives an *irregular dulness*, increasing to absolute flatness, in outlining which the finger perceives greatly *increased resistance;* it most often occurs over an upper lobe or under the sternum. (*d*) *Auscultation* varies with the patency of the bronchus, giving *bronchial breathing* when it is open and weak or *absent breathing* when occluded.

4. **Compression Symptoms.**—Besides the early dyspnœa and cough (*v. s.*), (*a*) the heart is often luxated or its yielding auricles are crowded. (*b*) The venous trunks suffer compression with frequent cyanosis and œdema of the face, neck and arms and with dilatation of other veins of the thorax, due to formation of a collateral circulation. (*c*) There is compression of the vagus, with rapid or slow heart, and recurrent laryngeal compression with phonic changes. (*d*) Dysphagia, (*e*) intercostal neuralgia and brachial neuralgia or paralysis, and (*f*) tracheal or bronchial stenosis are other symptoms of compression.

5. **Metastasis.**—This occurs in 85 per cent. of cases; there may also be extension, by contiguity, to the axillary or cervical glands.

6. **Complications.**—These include hæmoptysis from necrosis and cavity formation; gangrene and bronchiectasis; and pleurisy, which is usually serous; hemorrhagic effusions occurred in 12 per cent. of Moutard Martin's 200 cases; the fluid may resemble venous blood; adipose or chyliform effusions are rare; hydropic, fatty and granular cells may be obtained by puncture, and Hellendall advises aspiration for flocculi of tumor tissue, with the syringe and needle partly filled, with salt solution, before aspiration. Carcinomatous nodes may develop at the site of puncture, though hard nodules at the site of tapping not infrequently occur after puncture of simple pleuritis and ascites.

2. Sarcoma and Lymphosarcoma.

Sarcoma and lymphosarcoma may be primary or secondary, and are more frequent than cancer. They probably begin chiefly in the lymph nodes of the bronchi, and oftener on the left than on the right side. Extension in all directions occurs along the peribronchial tissues from the hilum. A primary very malignant lymphosarcoma develops in the lungs of miners in the cobalt mines of Saxony; it causes 75 per cent. of the deaths among them; secondary sarcomata in the liver are common. Ulceration is less common than in cancer, but the symptoms are, in the main, identical. Sarcomatous tissue is occasionally voided in the sputum or aspirated by the exploring needle. The diagnosis of malignant lung tumors must be made chiefly from aneurysm (*q. v.*) and pleurisy (*q. v.*). The *x*-rays may be of some value. There are three main clinical groups, (*a*) in which a neoplasm, perhaps its exact type, can be diagnosticated; (*b*) in which some growth is present but differentiation from aneurysm is uncertain; and (*c*) in which the disease entirely escapes detection or is only suspected.

Prognosis.—Death generally occurs in less than a year with cachexia or suffocation, but it may result in a few weeks after the first symptoms appear.

Other Tumors of the Lung.

Other tumors, as fibroma, lipoma, osteoma, or enchondroma are of solely anatomical interest.

Treatment.—The treatment of tumors of the lung is only palliative. Morphine must be used when the dyspnœa and cough are extreme. The *x*-rays offer little encouragement. Arsenic seems to help when given in full doses.

CIRCULATORY AFFECTIONS OF THE LUNG.

1. Acute Congestion.

As a secondary lesion acute hyperæmia commonly occurs wi
acute tuberculosis, pneumonia, influenza, effusive pleurisy, perha
rheumatism, with inhalation of hot vapors or air, or with cardiac strai
when extensive lesions occur in the lungs the obstructed blood m
seek other parts of the lungs and possibly produce *collateral congestio*
though this is justly disputed. French writers describe an *ac*
primary congestion to which they give the name of Woillez (*Malac*
de Woillez), though it was previously described by Dechambre, Lege
dre, Bailly and Fournet. Its status as an independent lesion is dou
ful; some cases are apparently larval or abortive pneumonia whi
occurs in any large epidemic. According to Carriére it is due to atte
uated pneumococci, which he has demonstrated by puncture of t
lung. Cold, exposure, alcoholism, and violent exertion are alleg
causes. It develops like pneumonia, with chill, fever of 101° to 10
pain in the side, dyspnœa, cough, and a sputum which is at first scan
and later is more abundant and viscid but not blood-stained. Physic
examination is said to elicit somewhat impaired resonance, weak
occasionally bronchial breathing, rales which are coarser than in pne
monia and at times a pleural rub. All these symptoms and sig
strongly suggest atypical fibrinous pneumonia. Venesection and vas
dilator remedies, as aconite or veratrum, are said to be of benefit.

2. Passive Congestion; Hypostasis.

In *broken compensation*, particularly in mitral stenosis, the lungs a
distended, heavy, indurated, pigmented, and on section are brownis
red; this is brown induration (*induratio pulmonum fusca*). Histologi
ally the capillaries are turgid, the connective tissue is increased, t
lungs are tinged with altered blood pigment, and the sputum contai
the so-called heart cells (See SYMPTOMS OF VALVULAR DISEASE.) T
breathing is sometimes puerile and the lung voluminous.

Hypostasis is a species of passive congestion which results fro
weak heart, as in typhoid and other fevers, in old age, cachexiæ, ma
asmus, in opium and other narcoses, or in apoplectic and other coma
Even under these conditions it rarely occurs if the patient's positi
is changed every few hours. The lung is dark red, engorged wi
blood and serum, and may also show aspiration pneumonia a
atelectasis; the latter conditions are often incorrectly called hypostat
pneumonia. On physical examination, there is moderate dulness ov
the lower lobes posteriorly; in apoplexy this may occur only on t
paralyzed side; the breathing is more often weak than bronchi

and moist (sometimes crepitant) rales are heard. Fever is no part of hypostasis.

Treatment.—Treatment of passive congestion is that of the fundamental lesion; phlebotomy, cardiac stimulation or even aspiration of the right ventricle in broken compensation and in hypostasis, frequent change of posture, and cardiants, are indicated.

3. Infarct; Embolism.

Etiology.—Embolism was first observed by Laennec; Virchow, Cohnheim and Litten established its pathology. The most frequent source of lung embolism is (*a*) fibrinous coagula from the right heart, the frequency and mechanism of which have been discussed under ACUTE AND CHRONIC ENDOCARDITIS (mitral stenosis) and SEPTICÆMIA. Clots may also reach the lung from peripheral or peri-uterine thromboses, and are either simple (*i. e.*, non-suppurative, marantic) or inflammatory and septic. Rarer causes are (*b*) tumor or hepatic tissue, (*c*) fat embolism after fractures, and liver injury, (*d*) air embolism or (*e*) parasites (echinococcus cysts).

Pathology of Infarction.—The embolus may obstruct a main branch of the pulmonary artery, straddle one of its secondary bifurcations, or lodge in a smaller vessel. Not every embolism produces an infarct, for instantaneous death may occur, the occlusion may be incomplete, or collateral circulation may be possible. Occlusion of an end vessel results in the hemorrhagic infarct, which is often wedge-shaped with its base toward the pleura, is dark at first and later lighter, is airless, on section projects above the cut surface, often measures 2 to 6 cm., and is attended by extravasation of blood into the lung cells and sustentacular tissue, though the lung is not torn. This is known as a fibrinous or *simple infarct.* If the embolus contains pus or putrefactive microörganisms, it develops into *abscess* or *gangrene.* Infarcts occur oftener in the lower than the upper lobes and more frequently on the right than on the left side. They are often multiple. Small infarcts may be wholly absorbed, but the larger ones leave pigmented cicatrices.

Symptoms.—(*a*) Infarction of the main pulmonary trunk may result in immediate death, as sudden death from sitting up after childbirth. In other cases death occurs in a short time from cerebral anæmia attended by convulsions, dyspnœa, and cardiac failure. The infarct may cover the greater part of the lobe involved; if the patient lives for some time, a sudden systolic murmur may develop over the pulmonary artery (Litten). (*b*) Infarction of a smaller vessel is often ushered in with chill, vomiting, dyspnœa, cyanosis and fever, even with bland embolism. The sputum contains dark blood and there is a "stitch in the side" from pleural participation. Physical examination frequently elicits a pleural rub and later, in some cases, slight dulness, especially behind, between the spine and scapular line; crepitant rales and bronchial breathing are frequently present. Pyogenic or putric clots cause abscess, gangrene, empyema, or pneumothorax.

Diagnosis.—Diagnosis is often at best a probability diagnosis Sudden cardiac failure is very difficult to differentiate from an embolic occlusion of a main pulmonary trunk, especially when death occur without hæmoptysis. The etiology, the sudden onset and circumscribed chest signs are of diagnostic importance. Treatment lie solely in cardiac stimulation.

4. Œdema of the Lungs.

Etiology.—In the etiology of this secondary, common, frequentl terminal or agonal affection, *three important causes* stand in the foreground, *viz., heart, kidney* and *lung disease.* Œdema consists of th pouring out into the air cells and interstitial tissue of a serous transudate. A precise classification of the mechanism of pulmonar œdema is difficult:—(*a*) *Stasis* due to weakened heart is probably it most frequent mechanism. Cohnheim and Welch demonstrated experimentally that it may result from failure of the left ventricle wherea there is continued activity of the right ventricle; this may be common in the death agony, but the explanation is not entirely satisfactory as cases of pulmonary œdema occur in which the pulse (and therefor the action of the left ventricle) is strong; however, general cardia anasarca may occur when the radial pulse is apparently sufficientl strong, though it is known that the heart must be weak. Grossman advances the theory that there is a spasm of the left ventricle. Wea heart may result from any disease of the endo-, myo-, or pericardiu Sahli explains certain cases by (*b*) *paralysis of the pulmonary vessel* Under either (*a*) or (*b*) nephritic œdema or the toxic œdemas, such a those from the iodides, ether, pilocarpine or muscarin, may be i cluded. (*c*) *Inflammatory œdema,* such as that around a fibrinou pneumonia and numerous other pulmonary diseases, cannot be strict brought under œdema, for it is an exudation, not a transudatio However, in this form the pulse is usually strong, and there is ofte fever. (*d*) *Angioneurotic* œdema is sometimes described. It is al possible that (*e*) *occult changes in the blood and bloodvessels* are caus factors. These varied mechanisms, though generally stated to b most common in cardiac, renal, and pulmonary lesions, may also ope ate in cachexia, anæmia, cerebral disease, old age, and innumerab other conditions.

Pathology.—Pathologically œdema may be circumscribed or often diffuse. The lungs are large, heavy and spongy; they pit, and o section, especially of the bases, a frothy, serosanguineous fluid exude

Symptoms.—Symptoms usually come on *precipitately,* with hurrie breathing, dyspnœa, cyanosis and carbon dioxide intoxication. Th *sputum* is copious, foamy, serous, somewhat resembling soapsuds, o at times is tinged with blood; it is rich in albumin and poor in mucu and cells. The *bubbling breathing* indicates serum in the alveoli. The second pulmonic sound is accentuated, the pulse is weak, the lung note is slightly tympanitic from relaxation, and coarse and fine rales are heard everywhere. If the lower lobes are filled, the physical signs may closely resemble those of consolidation, *i. e.*, there may be increased fremitus, bronchial breathing and dulness. The *diagnosis* is determined from the sputum and lung findings.

Treatment.—Treatment may be beneficial in some cases, though too often œdema is the death agony itself. (*a*) *Prophylaxis* concerns the causal disease, for example, cardiac weakness, or anasarca, in cases of nephritis. (*b*) Rapidly acting, *diffusive stimulants* are urgently indicated hypodermatically, as camphor, ether, strophanthus and strychnine, and coffee or champagne by mouth. (*c*) *Morphine* with atropine is at times almost specific. (*d*) *Venesection* relieves the right heart and therefore the lungs. (*e*) *Stimulating expectorants,* as senega and *rapid purgation* by crotin oil, ♏iij, on the tongue, are sometimes of value. The author has obtained no results from the acetate of lead, which is recommended by Traube in doses of gr. ss to i every hour or two, and by Nothnagel in a single dose of gr. x to xx.

5. Hæmoptysis.

Hæmoptysis or hæmoptoë refers to "spitting of blood," while profuse hemorrhage is known as pneumorrhagia.

Etiology.—(*a*) Laryngeal ulceration or inflammation; (*b*) tracheal ulceration or aneurysm bursting into the trachea; (*c*) severe inflammation or ulceration of the bronchi, stasis from cardiac disease, bronchiectasis, putrid and fibrinous bronchitis, rupture into the air passages of aneurysm or anthracotic lymph glands, and thermal, chemical or mechanical irritation are etiological factors; hæmoptysis may also occur in the acute exanthemata, in such affections of the blood as hæmophilia, scurvy, purpura hæmorrhagica, and leukæmia, and in gout, renal disease and cholæmia; hæmoptysis occurring as vicarious menstruation was mentioned by Hippocrates, and though rare, is an established variety; vicarious hæmoptysis after ovariotomy or suppression of hemorrhoidal bleeding is still more uncommon and its existence has been regarded with just scepticism. (*d*) Lung disease is the most important etiological factor, and the chief lesion is (i) tuberculosis, (*q. v.*), which causes 80 to 90 per cent. of cases of blood-spitting. It is commonest between the fifteenth and thirtieth years, and very rarely occurs under six years of age, as children seldom develop cavities. As stated under tuberculosis, it occurs as capillary or profuse hemorrhage. It was noted long ago that hæmoptysis sometimes occurs rather epidemically among tuberculous cases; in such an outbreak, Flick, Ravanell and Erwin found the pneumococcus in the sputum. (ii) Infarcts are usually characterized by hæmoptysis. (iii) Pneumonia, abscess and gangrene; (iv) tumors; (v) parasites of the lung, echinococcus, filiaria, gregarina, distoma and actinomycosis; and ([illegible] syphilis and leprosy, are more uncommon causes. (*e*) Hæmoptys[illegible] nervosa is most rare, though authentic cases have occurred in cerebr[illegible] traumatism, hysteria (where it has caused death), epilepsy and i[illegible] some other neuroses. (*f*) A toxic hæmoptoë may occur from mercury or iodide, but it is very rare.

Symptoms.—Symptoms may follow those of the causal disease, as tuberculosis, mitral stenosis, or aneurysm, or they may develop in apparent health. Hæmoptysis *is in itself only a symptom.* Blood poured out into a cavity or into the lung substance is not always voided, especially in very weak or moribund persons. Prodromes are unusual; *without* other *warning*, a salty taste in the mouth, substernal warmth, a sense of something rising from the chest, and coughing. are followed at once by a few spoonfuls or more of blood. At times a previously mucous sputum may be accidentally noted to be blood-stained. The blood varies in amount from the merest stain to 5 quarts (Laennec); it may appear but once or may recur several times. It is generally bright red and well mixed with air; the formation of rouleaux is uncommon. If the blood has clotted in the lung or air passages, the clot, too, is mixed with air and perhaps with mucus. Casts of the bronchioles may be voided. If there is gangrene or abscess, the blood may be brown or buff-colored and the red cells may be disintegrated. Brown hæmosiderin granules, or, if the blood has remained in the clot two weeks, hæmatin crystals may occur. Asthma crystals are occasionally seen.

Physical findings, other than those of the fundamental condition, are frequently lacking; if the blood obstructs a fairly large bronchus, there may be bronchial breathing, bronchophony, increased fremitus, and dulness if the affected area measure 2 by 5 cm. In the bronchioles, blood gives rise to small rales, in the bronchi to coarser rales.

Complications.—Fever, aside from the causal disease, may result from resorption (ferment fever). Urobilin icterus, functional heart murmur, relaxation of the right heart, albuminuria and puffiness of the ankles are possible but not conspicuous complications.

Diagnosis.—Examination of the nose, gums, throat and larynx readily excludes hemorrhage which may simulate hæmoptoë. It is often difficult to distinguish at the time of bleeding between arterial and capillary or between bronchial and pulmonary bleeding, but the question may be decided by the history, by later physical examination and by the clinical evolution.

HÆMATEMESIS.——*vs.*	——HÆMOPTYSIS.
(*a*) The blood is acid (unless a very large amount is voided).	Is alkaline
(*b*) The patient nearly always recognizing	whether it is vomited or coughed up.
(Blood from the lung is very rarely swallowed and then vomited.)	(Hæmoptysis, very rarely, causes consensual vomiting.)

(*c*)	Is often dark (though large amounts of bright red blood may be vomited, as in ulcer).	Nearly always bright (though when stagnating in a cavity it may become dark or buff).
(*d*)	Is mixed with food, HCl, ferments, etc.	Mixed with air, which is also found in the clots. (Not infrequently tubercle bacilli, heart cells, etc., are found.)
(*e*)	In 10 to 15 per cent. of cases blood is in the stools.	Is very rarely in stools.

Prognosis and Course.—The duration is variable, but spontaneous cessation is the rule. The immediate prognosis is good, as early death from hemorrhage is rare, except in cases of profuse pneumorrhagia, resulting, for instance, from aneurysmal rupture, and even here the patient may live years after the aneurysm has broken into the lung substance. Drowning or suffocation of the patient in his own blood is rare. The final outcome depends on the etiology. In Ware's 386 cases, 16 per cent. recovered.

Treatment.—**Prevention.**—In tuberculosis (*q. v.*) the patient should be kept quiet, the cough should be suppressed and exertion and vocal efforts should be avoided; in heart disease and marantic thrombosis, rest is indicated to obviate detachment of the thrombi.

Therapy.—*Absolute physical and mental quiet* should be enforced when hæmoptysis occurs. No movement should be allowed; coughing should be suppressed by hypodermatic injections of morphine, which also allay the attendant mental excitement (opiates are not to be employed in cases of very large hemorrhages, for bronchial occlusion may result). The patient should be assured of a favorable outcome; no physical examination should be made except auscultation of the chest in front, and this should be done only if necessary, for immediate treatment takes precedence of diagnosis; if rales indicate which side the hemorrhage is on, the patient should lie on that side to prevent aspiration into the contralateral bronchus.

Arterial pressure should be reduced by administration of aconite or veratrum, which lessen tension in both the pulmonary and arterial circulation. A liquid, simple, spare diet should be given. Alcohol, coffee, tea, strychnine, and digitalis should be interdicted. Even if the patient collapses cardiants should be avoided, for syncope is nature's readiest hæmostatic; the old custom of venesection in this way promoted thrombosis. Cardiac excitement is lessened by placing a large ice-bag over the precordium. Active purgation by croton oil is very beneficial.

Hæmostatics are not proven to control the lesser blood circuit, which appears to be rather independent of drugs which operate on the arterial circuit. Ergot, if anything, rather raises the blood pressure in the lungs and should therefore seldom be given. Calcium chloride gr. v to x, and common salt on the tongue are of some value (salt acts reflexly, by way of the vagus, on the pulmonary vessels). Gelatin (2 per cent.) may be used in urgent cases and the beneficial effects of turpentine have long been known. Nature, as a rule, effects the cure;

drugs do not, though opiates reinforce nature. Hippocrates employed ligature of the extremities, *i. e.*, binding tightly for a few minutes the vessels of the groin and axilla, in order to lessen the return flow of blood toward the point of hemorrhage. Artificial pneumothorax, to compress the bleeding lung, has been advocated in cases of desperate, profuse and persistent hæmoptysis.

PARASITES OF THE LUNG.

Echinococcus of the Lung.

Man may become infected with the tinea echinococcus from dogs by their licking their master's hands or from fæces contaminating human food, especially vegetables. The unilocular form is much more common than the multilocular. Infection occurs either (*a*) primarily, the embryo skipping the liver, or (*b*) much more often secondarily, by metastasis or breaking through the diaphragm from the liver. According to Neisser's figures from 900 cases, 50 per cent. occurs in the liver, 9 per cent. in the kidneys, 8 per cent. in the cranium and 7 per cent. in the lungs. The small miliary white cysts may grow to the size of an adult's head. They may calcify, indurate, suppurate, putrify or rupture into contiguous parts (See ANIMAL PARASITES).

Symptoms.—Many cases produce no symptoms and are unexpected findings at the post mortem. Dieulafoy described three clinical stages:—(*a*) the initial, (*b*) the tumor stage and (*c*) the suppurative or rupture stage. (*a*) The *initial stage* often has no symptoms, but may be marked by scanty capillary hæmoptysis, cough, emaciation and possibly fever, all of which may closely simulate incipient phthisis. (*b*) In the *tumor stage*, there is bronchial (or weak) breathing, increased fremitus and circumscribed dulness, which occurs in the lower lobes and twice as frequently on the right side as on the left; it sometimes causes bulging of the chest wall. There is a sharp transition between it and the note of the sound contiguous lung. Dislocation of the heart, compression of the lung and other luxations may occur. The *x*-rays often confirm the physical signs. The dulness curves upward, but is quite different from the curve of pleuritic effusion which is concave upward. Tumor of the lung gives a larger, harder flatness. Puncture (*v. i.*) reveals a clear, aseptic fluid of 1,009–15 specific gravity with little or no albumin, very rich in sodium chloride and the characteristic succinic acid; the inosite is of no practical importance; the hooklets are pathognomonic. (*c*) In the *stage of suppuration or rupture*, as in liver echinococcus, rupture may occur into the pleura (in which primary disease is 25 times as rare). Pneumothorax may result. Far oftener rupture into a bronchus occurs, with pain, dyspnœa, collapse, and sudden "mouthful" expectoration of pus, small vesicles, lamellated cyst wall and hooklets. At the time of rupture a curious urticaria develops (Finsen) which is probably toxic as the fluid from echinococcus cysts produces it when inoculated into animals. Profuse hæmoptoë is common. If it comes from the liver, the sputum is ochre-yellow from undissolved bilirubin, even when there is no direct connection with the bile vessels; cholestearin is often present. The vesicles or cyst wall may cause suffocation. Rupture may also occur into the pericardium, stomach, or peritoneum. In this stage hectic fever is usual.

Diagnosis.—Diagnosis is determined only from the sputum, or from puncture which has decided dangers, as sudden, probably toxic death is a common sequel.

Prognosis.—The course varies much. A cyst in two or three months becomes as large as a hazelnut, and in five months as large as an orange (Dieulafoy). Death may result from suffocation, hemorrhage, gangrene, hectic fever or toxæmia from rupture or puncture.

Treatment.—If puncture is made, and is followed by severe symptoms, the case should be instantly operated. There is no medical treatment. Without surgical intervention 57 per cent. (Hearn) to 66 per cent. (Davaine) die. Tuffier and Garré report but 10 per cent. mortality with operation.

Distoma of the Lung.

This affection was first described by Baelz in Japan, Formosa and Corea. The worm is plump, wide, egg-shaped, measures 8 to 10 x 4 to 6 mm., is brownish red and is provided with two small suckers. Its eggs are oval, brown and measure 0.08 to 0.1 x 0.05 mm. Little is known of its origin. It is found in the bronchi and in the infarcts and cavities which it produces. The cavities contain red disks, a mucoid substance and shreds of lung tissue, and are surrounded by fibrous tissue. The parasite is also found in the liver, diaphragm, mesentery and brain. The course of the disease is chronic, though most cases terminate favorably; it is marked by (*a*) bronchitic symptoms, (*b*) hæmoptysis and (*c*) a viscid clear sputum in which minute brownish-red dots, the distoma eggs, are seen. In exceptional cases death results from emaciation, exhausting hæmoptysis or brain embolism.

Tuberculosis, *syphilis*, and *actinomycosis* of the lung are described under INFECTIONS.

DISEASES OF THE PLEURA.

(A). PLEURISY.

Definition.—Pleurisy is an inflammation of the pleura, rarely
mary but usually secondary to tuberculosis or other lung disease, gen
infections, septicæmic affections and various diseases in contigu
tissues or organs.

Pleurisy constitutes about 3 per cent. of internal maladies an
the most common of all post mortem findings (in over 50 per ce
It is susceptible of division into infinite varieties, acute, subacut
chronic according to its course, dry or effusive according to its
sistence, primary or secondary as to its cause, diffuse or circumscri
as to its distribution and serofibrinous, hemorrhagic or suppura
as to its nature. Any such classification is more or less artific
whence it seems preferable to describe first all its symptoms and gr
special types later.

Etiology.—**1. Tuberculosis.**—So-called primary, spontaneous
cryptogenetic pleurisies are often tuberculous (Barrs, Netter, Lemo
Vaillard, Coston and Dubville). Landouzy considers that 98 per c
of all pleurisies are tuberculous and that 60 per cent. of all case
phthisis pass through the so-called *pleuritis a frigore;* Netter h
that 70 to 80 per cent., and Ashoff that nearly all "primary" ca
are tuberculous. Thirty-two per cent. of Osler's cases were tube
lous, 32.6 per cent. of Bowditch's and 40 per cent. of Hedges ca
Le Damay in 55 "primary" pleurisies proved by inoculation that
per cent. were tuberculous. Trauma favors development of tu
culous pleurisy. The causal lesion may be recognized at autopsy
calcified bronchial glands or in small lung foci. Tuberculous pleu
is often bilateral. Non-tuberculous pleurisy, by causing adhesi
may sometimes favor the development of pulmonary tuberculo
Its special relations will be discussed later.

2. Diseases of the Lung Substance.—Such diseases as pneumo
abscess, gangrene, infarcts, tuberculosis, tumors, pneumothorax, ec
ococcus, tertiary syphilis, and actinomycosis are etiological fact
according to Koplik, 95 per cent. of pleurisies in children follow pn
monia. It is less frequent in diseases involving the bronchi, as
grippe or measles.

3. Acute Infections.—Rheumatism, sepsis, puerperal fever, en
and pericarditis, variola, scarlatina, measles, diphtheria and ot
acute infections may result in pleurisy. It develops in about 2
cent. of typhoid cases; it may develop in secondary syphilis with
lung lesions (Chantemesse and Widal), probably from toxæmia.

4. Diseases of Adjacent Organs.—(*a*) *Intrathoracic* affections,
volving the ribs, spine, œsophagus, mediastinum, lymph glands,
phragm, and pericardium; (*b*) *abdominal* affections, as of the stom

spleen, or liver (abscess, tumor, or cirrhosis [in 16 per cent.]), subphrenic abscess, suppurative or tuberculous peritonitis (in 25 per cent.), perinephritic abscess, abdominal malignancy, etc., may promote pleurisy; pleural infection may occur by direct local invasion or by the lymph and blood streams; in neglected appendicitis, hepatic and diaphragmatic invasion occurs, followed by pleural infection.

5. **Tumors.**—Echinococcus and endothelioma (Wagner and Fraenkel) are the most important types. According to Neelsen, endothelioma is an infectious process, a lymphangitis proliferans (Schwenniger) or according to Schottelius it is a lymphangitis carcinomatosa. The pleura swells and develops callosities or adhesions. Carcinoma, sarcoma, ecchondroma, and rarely lipoma are also factors; pleural neoplasms are far more frequently secondary than primary.

6. **Altered Conditions of the Blood (Dyscrasiæ).**—Gout, nephritis, scurvy, and tertiary syphilis are often causative of pleurisy; these conditions frequently produce bilateral pleurisy and result possibly from chemical products or from reduced physiological resistance, which predisposes toward various infections.

7. **Terminal Infections.**—Various forms of sepsis are often fatal to subjects of chronic disease of the heart, liver and kidneys; bilateral pleurisy is frequent.

8. **Age, Sex, Localization.**—*Age*: most cases occur between the twentieth and fiftieth year, but no person is exempt, from intra-uterine life to extreme old age. *Sex*: Eichhorst observed pleurisy in 3 per cent. of all his male and 2 per cent. of all his female cases, and found the following *localization*: right-sided in 54 per cent., left-sided in 42 per cent., and bilateral in 4 per cent. Cases following nervous disease, cold or exposure, venous thrombosis, or trauma can usually be brought within the above given classification.

Bacteriology.—The etiological classification above given does not correspond to the bacteriological division. Suppurative pleurisy may result from the streptococcus, pneumococcus, tubercle bacillus, or less often from the staphylococcus, or typhoid or colon bacilli; serous pleurisy may be due to the tubercle bacillus or to the pyogenic organisms. The frequently negative cultural results have favored the hypotheses that chemical products, such as retained urinary substances in uræmia or tuberculin in phthisis, may of themselves produce pleurisy, or that bacteria produce pleurisy and then die. It must be remembered that cultures are usually made, not from the pleura directly, but from the fluid effused from it. The organisms found are most frequently the pneumococcus, tubercle bacillus and streptococcus, less often the staphylococcus, pneumobacillus of Friedländer, typhoid, colon, influenza (in two cases only) and diphtheria bacillus, gonococcus, ray fungus, glanders bacillus, Micrococcus tetragenus, and Micrococcus pyogenes tenuis. (*a*) *Purulent pleurisy* (empyema) is most often due to the streptococcus in adults and to the pneumococcus in children. About 10 per cent. of empyema is tuberculous; empyema giving no results culturally or by inoculation is usually tuberculous and may be regarded as a cold abscess of the pleura (Kelsch and Vaillard). The staphylococcus, Micrococcus tetragenus and pyogenes tenuis, pneumobacillus, typhoid and colon bacilli are infrequent factors. (*b*) *Serous pleurisy* is usually tuberculous, as shown by the constantly increasing percentage of cases in which inoculations are successful. It was formerly stated that tubercle bacilli are not frequently found, and that, when found, their presence suggests a direct communication with a tuberculous focus in the lung or lymph glands; negative results are due either to an insufficient amount of fluid (less than 10 to 20 c.c.) being inoculated, or to the effusion being due to toxins rather than to the microörganisms themselves. Eichhorst found tubercle bacilli by direct examinations in but 15 per cent. of serous pleurisies, though his inoculations were positive in 62 per cent. Jousset recommended that the fluid be allowed to coagulate and the clot digested with pepsin, centrifuged and then stained; his method is termed inoscopy. (*c*) Most cases of so-called primary pleurisy which are non-tuberculous are due to Fraenkel's pneumococcus. (*d*) Meta- and para-pneumonic pleurisy is most frequently due to the pneumococcus, but also to the streptococcus and staphylococcus, even when no pus is formed, although mixed infections have a tendency to suppuration.

Symptomatology.—Physical findings are absolutely essential for diagnosis and all subjective symptoms are ambiguous or may be wholly absent. In the main, all types have much the same signs which differ more quantitatively than qualitatively; serous pleurisy nearly always shows friction, and fibrinous pleurisy is almost always accompanied by some exudation.

Inspection.—(*a*) Inspiratory retraction of the intercostal spaces may result from imperfect ingress of air, from pain or from adhesions. Epigastric retraction during inspiration is due to contraction of the diaphragm. (*b*) The patient often lies at first (during the dry stage) on the sound side to avoid pain from pressure on the inflamed surfaces; later (during the exudative stage) he lies on the diseased side in order to allow, for mechanical reasons, a freer expansion of the sound lung, especially when the exudate is large. Traube believed that continued lying on the diseased side aggravated pain by congesting the pleural and subpleural veins. The patient may sit up for breath, or assume the diagonal decubitus (Andral). (*c*) *Ectasia* (distention) of the affected side occurs especially in children, and is determined by mensuration and inspection. The interspaces are obliterated, the costal arch is immobile during respiration, the skin glistening, the spine curved toward the diseased side, the shoulder higher and the nipple and scapula are further from the median line. The right chest is normally one or two centimeters larger than the left; again, the sound side usually measures three centimeters more than its normal dimensions (or more than the diseased side) on account of compensatory emphysema (Gerhardt and Kerlias). In some cases of very large effusions the writer has observed an apparent retraction of the diseased side. (*d*) *Respiratory movement* of the affected side *is de-*

creased or absent in both dry and exudative pleurisy. Respiration is reflexly decreased on the diseased side, and at times occurs somewhat later than on the sound side, *i. e.*, the breathing lags on the diseased side. The frequency of respiration is increased from lung compression, pain, heart luxation, the low diaphragm, fever or œdema and paresis of the intercostal muscles. Pain alone may reflexly decrease the respiratory expansion, and variation on the same side may occur, there being more movement above and less below. (*e*) The *x*-rays often, but not always, show a dark shadow. (*f*) Inspection also reveals cyanosis, œdema of the chest wall (in suppurative pleurisy and occasionally in other forms), dislocation of the apex beat, substernal pulsation, venous pulsation; (*g*) *absence of Litten's phenomenon* and (*h*) rarely pulsation of the effusion. (*i*) C. L. Greene describes a rhythmic lateral displacement of the heart peculiar to pleurisy: "Such movement is most marked in medium-sized effusions. The heart approaches the affected side in inspiration and moves outward in expiration. The extent of movement is variable, but often amounts to two inches. It may be measured by fluoroscopic examination, auscultatory percussion, or, in the case of right-sided effusions, by simple deep percussion of the free cardiac border, or in some instances by mere inspection of the apex beat. Deep breathing, and especially forced expiration, are essential to the success of the maneuver, and to obtain this morphine must sometimes be administered. Marked rhythmic lateral movement has not been found by the writer in pneumonia, tuberculous infiltrations of the lung, malignant disease of the pleura or lung, or in subdiaphragmatic abscess."

Palpation.—Palpation confirms the findings of inspection. (*a*) An up and down (less often horizontal) *friction rub is felt;* it increases on pressure, which also increases the pain and tenderness, and it is the more readily distinguished the coarser the rub and the more capable the chest wall of vibration. It is usually absent in obese individuals. The friction develops before the effusion, is often present with it, and after it is absorbed. The rub is due to roughening of the pleura, which loses its glistening appearance and looks like glass upon which one has breathed (Klebs); the two raw pleural surfaces scrape on each other. It may be very transient, lasting a few hours or days, or even disappearing when the patient takes a deep breath; or it may last for months, or even years. (*b*) Pressure on a tender interspace during deep inspiration may cause a sudden contraction of the upper part of the rectus muscle on the side of lesion (Schmidt). (*c*) *Vocal fremitus*, normally greater on the right than on the left side, varies according to the level at which it is tested. (i) Above the effusion it is normal. (ii) Near the spine, over the compressed and relaxed lung it is increased. (iii) Below, it is weak or absent, from exudation of thick fibrin or fluid, though less change is observed in dry pleurisy than in the effusive form. Irregular findings are due to the presence of adhesions bringing the lung closer in some places than in others to the chest wall. The condition of the lung and the bronchial secretion are important in diagnosis, whence before testing, the patie should cough and take a few deep breaths. The results are irregu in cases of loculated effusion. (*d*) By tapping with the finger ti the *increased resistance* is clearly palpated. (*e*) Œdema of the ch wall on the diseased side may be inflammatory (in serous as well in purulent pleurisy), or mechanical and more intense from pressu on the azygos or hemiazygos veins. (*f*) Fluctuation is rarely palpab and only in cases of great effusion. It is elicited by pressing fingers of one hand upward toward the diaphragm with the fingers the other hand in the interspaces. (*g*) Palpation also determines *dislocated apex beat,* the dislocated liver or spleen, the bulging of diaphragm and sometimes a furrow between the diaphragm and li (Stokes).

Percussion.—Percussion is negative in dry pleurisy, except wh there is a very thick fibrinous exudate, which may cause duln and weak breathing; after an effusion has resorbed, the plastic lym which is left may cause dulness. When there is effusion (*a*) *duln* is found between the fifth and eighth ribs in the left axilla or on right side over the liver and posteriorly. Dulness may also result fr old adhesions, atelectasis of the lung, great fibrin formation in pleura, or a very thick chest wall. As in pneumothorax, distention the lungs and calcification of the ribs may somewhat obscure the d ness of an effusion. In doubtful cases the apparently sound si should be percussed first. According to Ferber's experiments on t cadaver, thirteen ounces (400 c.c.) of fluid are necessary in the adu for recognition and four ounces (120 c.c.) in children; post morte experiments, however, do not exactly fulfill the conditions obtainin during life. The effusion must be one inch (2 c.c.) thick to produc dulness. Sometimes, however, even six to seven ounces (200 c.c. which produce a dulness two fingers in height, may be recognized In some doubtful cases smaller amounts may be detected, as prove by puncture, especially in persons having thin thoracic walls, as in children and women. Light percussion is necessary when the effusions are small, lest other normal parts be brought into vibration. Befor percussion the patient should breathe deeply several times to eliminat partial collapse of the lung and impaired ingress of air from accumulated bronchial secretion. *Percussion is absolutely essential to diagnosis.* Not merely the audible dulness should be regarded, but also the *palpable sense of resistance,* on light percussion. The dulness is marked, and usually amounts to *distinct flatness.* In larger effusions, the *flatness* is at first located posteriorly, and is noticed to increase along the spine and also laterally in the axilla. In massive exudates, flatness may reach from the lower limit of the pleura to above the clavicles.

The dulness is usually highest posteriorly, but adhesions may cause an opposite condition. The line of dulness is not even, but describe a parabolic curve (courbe de Damoiseau), due according to Gerhardt to the uneven thickness of the chest wall or according to Eich horst to irregular adhesions. If the fluid should effuse while the patient is in bed, the highest level of dulness would be in the axillary line; if it should effuse when he is out of bed, the upper level would be horizontal. Dulness is found one to two centimeters higher than the upper level of the fluid, due to plastic exudate. (*b*) Right-sided effusions merge with the liver dulness, but the lower limit of left-sided effusions can usually be determined, because they *invade Traube's semilunar space.* This "half moon" space lies between the fifth and sixth costal cartilages above, and tenth rib below, representing the supplementary pleural space, and is designated as the "half-moon" space because of the curve at its upper portion, produced by the apex of the heart; its tympanitic note is decreased or replaced by flatness in left-sided effusions (without adhesions). Dulness here may not be confused with that of the spleen, for there is no respiratory excursion in pleurisy, and the dulness lies more toward the heart than toward the splenic region. Scybala, or food in the stomach or bowels, may also impinge on Traube's space. (*c*) *Respiratory excursion is absent* in effusions of any size. (*d*) *The change of the upper line of dulness with change of position* has been emphasized by Gerhardt, Rosenbach, Da Costa, and by most writers. It is usually stated that change of position causes variation in the level of the effusion, and that the level changes in 55 per cent., changes very little in 30 per cent., and none in 15 per cent. of cases. According to personal experience, it is difficult to detect on account of the adhesions which are usually present at the level of the fluid. The line of dulness does not change rapidly on mere change of posture; the patient must lie some hours in another posture before any alteration of the upper level occurs. (*e*) *A vertical strip of tympany* along the spine, often measuring three centimeters wide, may be detected when there is a large exudate; it is due to compression of the lung. Tympany in the lower part of the left chest may result from gaseous distention of the stomach and intestines. (*f*) Tympany is observed over an upper lobe from compression or relaxation of the lung. It was first observed by Avenbrugger and Skoda, and is usually called the *Skodaic note.* Rosenbach explains it by tension of the intercostal muscles during inspiration. (*g*) "*Cracked-pot*" *resonance* occasionally appears on short forcible percussion over the upper lobe, and results from sudden expulsion of air through the *rima glottidis.* (*h*) *William's tracheal note* is observed on the left more than on the right side, and chiefly when the entire side is dull. It is obtained most clearly in the first and second interspaces, and is due to transmission of percussion vibration from the fluid to the trachea, giving a tympanitic note. A change of pitch with opening and closing of the mouth, like that observed by Wintrich, in lung cavities, may sometimes occur. (*i*) *Grocco's sign* is a small triangular dulness on the sound side along the vertebræ, due to compression of the healthy side or to dislocation of the aorta and mediastinum by large exudates. (*j*) There may be *dislocation of other organs.* There is no direct parallelism between the amount of exudate and the amount of luxation,

which, of course, is absent if the organs are adherent. The heart
usually dislocated *in toto* and but seldom suffers torsion or change
the direction of its axis. The right ventricle may be seen to pulsate
the right of the sternum, even as far as the line of the right nipp
when the effusion is left-sided. In right-sided effusions the apex
pushed to the left and downward, for the diaphragm is lower. T
liver is less easily displaced than the heart, but in right-sided effusic
is pushed down and the left lobe tipped somewhat upward, though bc
lobes may be dislocated downward in very extensive pleurisies (Trau
and Fraentzel). In left-sided effusions the spleen is dislocated dow
ward and forward, undergoes some torsion, and may stand perpendi
lar to the costal arch.

Auscultation.—This is most important in dry or effusive pleuri
(*a*) The *friction rub* (see Palpation) may be hard or soft, is sup
ficial (*i.e.*, close to the ear), and resembles the sound made
scratching the finger upon the ear, or the crunching of snow un
the shoe; it often develops in the first twelve hours. It is most fi
quent during inspiration, but is also very often detected in expir
tion; it seldom occurs during expiration alone. Inflammation of o
leaf of the pleura is sufficient for its production. It may disappe
after several long breaths which smooth the pleural roughening. Th
interrupted rub is due to pleural irregularities. It may be increase
by pressure and is subject to no law as to its duration. Wintric
heard a pleural rub which lasted more than four years and Gerhard
heard one lasting six years. It is heard most frequently over the
anterolateral portion of the chest, and is detected more clearly with
the stethoscope than with the naked ear, but may sometimes be heard
at a distance of several feet as in a case seen by the writer with Dr. H.
M. Richter. If it is apical, it is probably due to tuberculosis. It may
also be present in miliary tuberculosis (v. Jürgensen) and in tumors
of the lungs or pleura without actual pleurisy. It usually becomes
less clear when exudation develops, and its reappearance may indicate the beginning of absorption, or the extension of the process at
the level of the fluid. (*b*) The *breath sounds* are modified not only
by the effusion, but also by the condition of the lung, the strength of
breathing and the conditions of conduction existing in the pleura and
thoracic walls. In general, and especially in men and children, the
respiratory sounds are weakened, even in fibrinous pleurisy, from
repressed breathing, and perhaps from pain alone; cog-wheel breathing is common. In moderate effusions three zones are observed: (i)
Absence of breathing in the lower parts from compression of the lung
by the exudate, which lessens the conduction of the vesicular sounds;
(ii) Bronchial breathing in the middle zone from moderate collapse of
the alveoli of the lung, with fair conduction through the bronchi.
(iii) An upper zone of sharp or exaggerated vesicular breathing. I
larger effusions the lower zone extends higher and bronchial breath
ing may be heard near the clavicle or between the scapula and spine
In the largest effusions the breathing is indistinct everywhere, wit

areas of metallic bronchial breathing when the lung is wholly collapsed. Amphoric breathing results in exceptional cases. (*c*) The *voice sounds are not heard* over the fluid or over very thick, plastic exudate. They may persist in various places, when islets of the lung are adherent to the chest wall. (*d*)*Bronchial breathing* and increased fremitus may be present, especially in children with empyema, under the following conditions: if the chest wall is not the seat of œdema, if the bronchi are patent, if there is no great fibrin deposit on the pleura, and if the lungs are retracted but not absolutely compressed. The bronchial breathing may sometimes be as intense as in pneumonia or tumor. In adults increased fremitus may be observed between the spine and scapula where the lung is compressed. (*e*) *Bronchophony* depends upon the same conditions as bronchial breathing, *viz.*, patent bronchi, moderate retraction of the lung and conditions favoring good conduction of sound. (*f*) *Ægophony*, which is an exaggerated bronchophony, was described by Laennec; it is found chiefly in middle-sized effusions, usually at the level of the fluid. It occurs less often in large cavities and in infiltrations of the lung. In large effusions it is rare, the compression being too great. It is caused by the voice sounds passing through the compressed smaller bronchi to the chest wall; it may be present when the exudate is increasing or decreasing at a given point. (*g*) *Baccelli's symptom* is the "whispering voice," which is heard on the diseased side; Baccelli thought that it was observed more often in serous effusions, but it also occurs in other forms of pleurisy, in chronic induration of the lungs, pneumonia, hydrothorax and cavities. (*h*) A wide propagation of the *heart tones* is indicative of consolidation, rather than of effusion, but if consolidation is excluded, the hearing of the heart tones more widely than normal suggests dislocation of the heart by an effusion. Murmurs, which result from bending of the large vessels by dislocation of the heart may be heard, as well as accidental murmurs resulting from a weakened heart, from toxæmia or from high temperature.

Diagnostic Puncture.—This determines: (*a*) the existence of pleurisy, excluding pneumonia, hypostasis, tumor, or atelectasis; (*b*) the character of the fluid; and (*c*) the prognosis. It is indicated in all doubtful cases. Puncture with aspiration is also an important therapeutic measure. *Negative results* from puncture may result from a thick fibrin deposit and thick exudate, from failure to reach an encapsulated effusion, as in interlobar pleurisy, and from the membrane being pushed in before the needle so that the fluid is not reached. In negative or dry taps after withdrawal of the needle, examination should be made to see that no plug of fibrin or cells has collected in the needle. If a plug is obtained it should be emptied upon a glass slide and examined under the microscope. Care should be taken that no carbolic acid or alcohol remain in the syringe or needle, which, by coagulating the serum, may cause some dry punctures. The puncture should be made with a large needle. *Positive results:* when the needle is introduced, resistance may be felt, which is due to a callous pleurisy or an indurated lung. Often after introducing the needle, the point may felt to be freely movable as in a cavity. Sometimes the results diff according to the level of the puncture; serum may in rare cases obtained from a high puncture and a cellular deposit from a low one; this results from the cells settling to the bottom. Again, pun ture in different localities may show serum in one, and hemorrhage pus in other sacs. Bacteriological examinations made with 10–20 c.c. the serum give frequent results, and 60 to 90 per cent. of the sero cases are proven tuberculous on inoculation. The special chemic bacteriological, cellular and other characteristics will be discuss under individual forms.

Symptoms.—Symptoms may be absent (latent pleurisy); if prese they are not distinctive and there is no fixed relation between the sym toms and physical findings. (*a*) The *temperature* is variable. Its r is usually gradual and a rigor, as in pneumonia, rarely occurs. E weeks there may be no temperature, even in purulent exudat especially in marantic, diabetic and anæmic subjects and in termi infections. When present it may be continuous, but more frequen subcontinuous or remittent. The average elevation is 101° to 10 Temperature is no positive index as to the character of the flu Exacerbations of temperature are important prognostically, especia when the appetite is poor and no change occurs in the physi findings. Recovery is not to be expected until the temperature h been normal for some time. A sudden rise and fall, like a crisis, exceptionally observed just before resorption. (*b*) The *pulse* is increase it is sometimes small and is occasionally of the paradoxical type unequal in the two radial vessels. (*c*) *Dyspnœa* is seldom a part pleurisy, for effusions may attain enormous proportions without respi atory difficulty, particularly when they are gradual in developmen Dyspnœa is usually referable to coincident bronchitis, pneumoni lung congestion (Potain), pericarditis, arteriosclerosis, nephritis or t bilateral pleuritis. (*d*) *Pain* is usually an initial symptom. It is in n way proportional to the physical findings. It may be absent when auscultation shows a marked rub. Its character is usually dull, tearing, stabbing and sometimes very severe; it may be crampy or colicky. The pain is usually pleural (Cruivelhier) but may be neuralgic or due to neurotic or muscular causes. Pain is present in the diseased side, well down over the ribs, over the liver, epigastrium or low in the back, and sometimes may radiate to the sound side (Laennec and Gerhardt), into the arm or spine, or into the abdomen, especially in children, in which case it may simulate appendicitis. (*e*) *Coughing*, an early and frequent symptom, results from accidental bronchitis or more often from pleural irritation. It may occur after change of posture or after tapping. It is usually unproductive. (*f*) Other symptoms are anorexia, vomiting, headache, difficult swallowing due to involvement of the *foramen œsophageum* (Ferber) and marked sweating (the sweats of pleurisy rank next in importance after those of sepsis and rheumatism.) The urine is scanty but increases during resorption, when peptonuria and transient albuminuria are often found (Courmont and Nicola). The *skin* may be pale and cachectic from purulent accumulations, red from high temperature, cyanotic from poor oxygenation or suddenly pallid from hemorrhage into the pleura when there is also a sudden increase of fluid. *Emaciation* is common in chronic pleurisy of the tuberculous or purulent type.

Special Forms.—I. Character of the Inflammation.—In 790 cases Eichhorst found serous pleurisy in 81 per cent., fibrinous in 12 per cent., purulent in 5 per cent., ichorous and hemorrhagic, each in 1 per cent.

1. *Serous pleurisy* is the prototype of pleural inflammation. As stated, most cases are tuberculous. The fluid is lemon-colored. Its specific gravity ranges between 1,015 to 1,023, which Méhu considers characteristic of inflammation. Albumin is present, and amounts to 3 to 7 per cent.; sugar is frequently present; glycogen, urea, uric acid, and bile are inconstant constituents. The fluid clots after puncture. All varieties of pleurisy, serous and otherwise, contain white blood cells, red blood cells and endothelial cells; the cells may show some fatty degeneration.

Cytodiagnosis.—Much emphasis has been placed on the character of the leukocytes found in tuberculous pleuritis; Widal and Renault (1900) especially insisted—and later writers confirmed the statement—that *lymphocytes* are characteristic of primary pleural tuberculosis and afford early diagnostic data, long before the results of inoculation are available; the fluid must be examined before it coagulates; in other pleurisies the polymorphonuclears prevail; in other words, the "lymphocytic formula" indicates tuberculosis, the "polymorphonuclear formula" indicates pyogenic infection or pleurisy secondary to well-developed phthisis, and the "endothelial formula" indicates irritation, transudation or malignancy. The tuberculous nature of pleurisy is established by inoculation tests, which are positive in 50 to 66 and 94 per cent., by apical involvement or that of other serous membranes, by the finding of tuberculosis in cases of sudden death in the course of pleurisy (Lendouzy, Dujardin, Beaumetz, Dieulafoy, Kelsch, Binet), and by the fact that 25 to 33 per cent. of cases later become obviously tuberculous. It is well to suspect, at least, the tuberculous nature of all so-called rheumatic pleurisies (*pleuritis a frigore*), though cold may sometimes produce pleurisy in both man and beast. The sputum and lungs should be carefully watched in such cases for some time (See PROGNOSIS). For the tuberculin test and Marmorek's modification of it see DIAGNOSIS OF TUBERCULOSIS. The leukocytes are not increased in the blood count.

2. *Fibrinous pleurisy* usually shows some fluid, serous or purulent, if systematic punctures are made.

3. *Purulent or suppurative pleurisy* (empyema) usually begins as a serous exudate which soon becomes opaque and purulent. The sterile tuberculous form has been discussed (*v. s.* BACTERIOLOGY). The most frequent types are (*a*) the streptococcic form, which is observed chiefly in adults, in which the fluid is thin and separates on standing into an

upper serous layer and a lower denser layer, in which fib
formation is slight; and (*b*) the pneumococcic form, which is m
common in children, in which the fluid is more homogeneous and vis
like the "laudable" pus of the early writers, in which the fibrin
membrane formation is abundant, and the odor peculiar; it
more often accompanied by endocarditis, pericarditis and meningi
and is more often febrile and more susceptible of spontaneous recov
than the streptococcic form. Over 80 per cent. of juvenile empye
are pneumococcic. The fact that the pneumococcus has far less p
genic properties in the lung than in the pleura, has excited frequ
comment. Microscopically, various bacteria are present (*v.* BACTE
OLOGY), polymorphonuclear and often fatty white cells, and someti
cholestearin and Charcot-Leyden crystals. The distention of the ch
may be enormous; Krause reported 22 pounds of pus in one ca
Leukocytosis and hectic fever are mentioned under symptoms.

Empyema pulsans (Walshe) is not frequent; 95 per cent. of
pulsating effusions are purulent, while but 5 per cent. are serothor
93 per cent. are left-sided. A pulsating tumor as large as the fist m
be noticed, which usually, but not always decreases during inspirati
or the effusion may simply pulsate behind the ribs; in either case
pulsation may be expansile. The pulsating tumor is usually betw
the second and fourth ribs, anteriorly, but may be seen lower on
side or even well below in the back. Pulsation is not, as some consi
it, a sign of necrosis of the pleura; it has been attributed, though wi
out satisfactory reason, to such causes as coincident pneumotho
(Féréol), atelectasis of the lung (Comby), or adhesions and paresis
the intercostal muscles (Traube, Keppler). The most apparent c
ditions for pulsation in an empyema, are considerable cardiac ener
massive exudation and paresis of the intercostal muscles. Empye
pulsans may be confused with aneurysm, which latter is much slo
in its evolution, is much oftener located above and to the right, and
its own signs, murmur, and thrill; it may be confused with pulsat
abscess, gangrene or tumor of the lung and pulsating pneumonia. T
pulsation ceases after aspiration. Aneurysm may co-exist with pleur
Expansile pulsation of the chest wall has been observed twice with
exudation, aneurysm or any organic change (Lafleur and Edwar

Empyema may rupture externally, *empyema necessitatis*. Rupt
is usually along the sternum or lower ribs. Gravitating abscesses m
result, as pulsating tumors in the groin and loin, which may be c
fused with aneurysm or spinal caries. Rupture into the lung m
cause suffocation during sleep; according to Netter, 25 per cent.
post-pneumonic empyemas rupture into the bronchi. Fistulæ, he
fever and amyloidosis are frequent sequelæ. Hippocrates knew of
"mouth full" expectoration and also the fact, later described
Traube, that pus may penetrate the lung like a sponge without p
foration or pneumothorax. Unverricht thinks, however, that perfo
tion into the lung without pneumothorax is probably caused by a sm
opening which allows the escape of fluid into the lung but does not all

the escape of air into the pleura, because of the higher tension in the exudate. Hallin (1905) found 114 cases of *bilateral empyema* recorded in the literature; 67 per cent. occurred in males and 90 per cent. in young persons under fifteen years old; the mortality was 30 per cent.

4. *Putrid pleurisy* is usually associated with gangrene of the lung, pneumothorax, œsophageal carcinoma, appendicular abscess and kindred lesions. The fluid is extremely fœtid; fat, leucin, tyrosin, cholestearin and sometimes hæmatoidin crystals are found; the colon bacillus and other gasogenic bacteria are frequently present.

5. *Hemorrhagic pleurisy* is usually serofibrinous, and the exudate is tinged with blood. Five to six thousand red cells per c.mm. are necessary to produce a reddish tinge. Its character is seldom suspected before puncture. (*a*) Most cases are *tuberculous*. Dieulafoy speaks of these cases as having "hæmoptysis into their pleuræ." After puncture, more fluid is frequently formed, but recovery is possible. (*b*) Some cases are *cancerous*; Moutard-Martin has found hemorrhagic pleurisy in but 12 per cent. of pulmonopleural cancer; pain, dyspnœa, luxation and rapid pulse are frequent symptoms; the fluid is dark red, contains little fibrin, and is usually formed rapidly; a case of Desnos was punctured 30 times in six months. (*c*) Some cases occur in *liver cirrhosis* (of which Laennec published the first case in 1826), in nephritis, and in hemorrhagic fevers or exanthemata; profuse hemorrhage into the pleura was called pleural hæmatoma by Wintrich and pachypleuritis hemorrhagica by Chouppe from its resemblance to hemorrhagic pachymeningitis.

6. *Chyliform pleurisy* occurs chiefly in pleural neoplasms or in tuberculosis; the fluid is milky, contains fatty droplets, fatty cells and albuminous detritus in suspension; cholestearin and lecithin may be found (*v. i.* CHYLOTHORAX). The exudate is sometimes colloid.

In multilocular exudation, in which one pocket contains pus, another serum, a third hemorrhagic fluid, etc., Galliard speaks of polymorphous pleurisy.

II. Location of the Exudate.—1. *Pleuritis diaphragmatica* produces much subjective disturbance. Radiating pain is often experienced, which may resemble that of angina pectoris (Andral). Pain and tenderness in the epigastrium and between the tenth rib and the xiphoid, is pathognomonic, according to Guéneau de Mussy.

Schmidt's respiratory abdominal reflex, an inspiratory spasm of the upper part of the rectus muscle, is rather indicative of phrenic involvement. Dysphagia from inflammation of the foramen œsophageum may result. If left-sided, vomiting and singultus may be noted, and if right-sided, icterus is not uncommon. Cough may occur spontaneously or on pressure. Pressure in the lower interspaces near the spine elicits tenderness; tenderness over the cervical portion of the phrenic nerve may be noted, together with reflected pain along the mammillary branches of the cervical nerves to the shoulder. Dyspnœa is usually great, the temperature is generally normal, the lower chest moves but little, the respiratory murmur is very weak, and friction may be heard over the liver or in the "half-moon" space of Traube. If there is no exudate, the diaphragm may remain high. Fluid may accumulate between the diaphragm and lung without reaching the chest wall, and therefore without causing dulness. Fibrinous is more common than serous effusion.

2. *Interlobar pleurisy* is difficult to diagnosticate, as there is little surface dulness. The amount of exudate may vary from a few c. c. up to 400. It occurs most frequently after lobar pneumonia or in tuberculosis. Previous pleurisy may favor its development by marked adhesions obliterating the general pleural space. As Laennec noted, it is almost always suppurative. Its early diagnosis is uncertain, for the fever, rales, cough and dyspnœa are suggestive of a lesion of the lung; later a zonular dulness is found conforming to an interlobar fissure; the dulness is bounded above and below by a normal or somewhat over-resonant note. The heart may be pushed to the right, when there is left-sided localization, but the liver is not luxated in right-sided localization. The symptoms may resemble those of lung abscess from which during life differentiation is sometimes impossible. Interlobar pleuritis may be shown clearly by the *x*-rays. Large effusions may break internally, and pus is then suddenly detected in the sputum. Hæmoptysis may occur. Puncture is attended with some danger, as the lung may be infected by the needle, during its withdrawal. Spontaneous recovery is infrequent. The outlines of the lobes are shown in Figs. 33, 34 and 35.

3. *Mediastinal pleurisy,* described by Laennec and Andral, is most often tuberculous or pneumococcic, and more frequently purulent than serous. The symptoms of lung compression are less distinctive than are those of compression of the mediastinal tissues. There may be wheezing, dyspnœa, dysphagia, fever, intrathoracic oppression or pain, a pertussis-like cough, enlarged thoracic veins from azygos compression, deviation of the larynx or trachea, and dulness between the scapula and spine; the *x*-rays picture may be suggestive. The sudden onset and febrile course differentiate it from aneurysm, tumor, and adenopathies. The outlook is unfavorable, without operation.

4. *Peri- (para-) pleuritis* (Wunderlich, Bartels, Billroth) is an inflammation outside of the parietal pleura, and is usually purulent. Some cases follow glanders, but most of them are due to *actinomycosis.* Fistulæ and the evacuation of the characteristic sulphur granules are common. The symptoms are more local and irregular than those of pleurisy; fluctuation is more frequent; heart dislocation and lung compression are rare; the respiratory excursion is free, and on heavy percussion lung resonance may be elicited beneath the dulness. Metastatic inflammation, perforation and burrowing are common.

III. Pleurisy in Children.—In children pleurisy is attended by more general symptoms, higher temperature, higher pulse and more rapid respiration rate. Bronchial breathing is more frequent, there is less dulness, less luxation, and less friction. It often follows pneumonia, where, in the majority of cases, the empyema is more benign. Tuberculosis is less frequently causative in children than in adults. According to Lemoine, metapneumonic pleurisy is distinguished from the parapneumonic type, by its slower course.

Fig. 33.

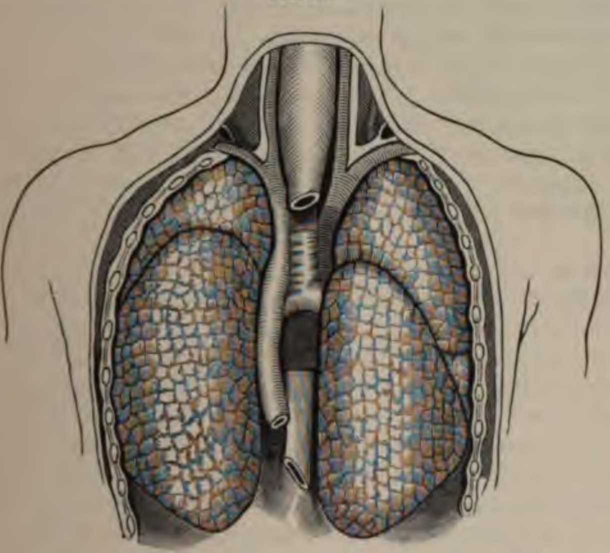


Fig. 34.

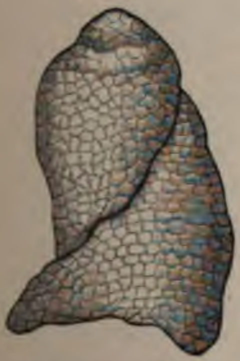
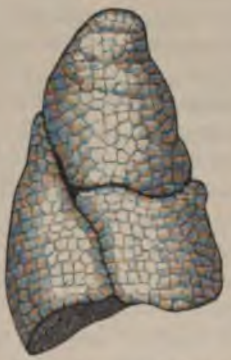


Fig. 35.



Cuts from Dieulafoy, showing: Fig. 33, the posterior surface of the lungs and their interlobar fissures; Fig. 34, the lateral aspect of the left lung; and Fig. 35, that of the right lung.

Course.—The course of uncomplicated pleurisy averages three to six weeks; acute cases last two, sub-acute three to eight, and chronic eight or more weeks. No general rule can be laid down, and the onset, whether mild or stormy, is no index of the issue. No cycle is seen in any form of pleurisy. Pleuritis acutissima, with high fever, typhoid symptoms and early death is fortunately rare. Friction along the lowest limits of the pleura and in front rarely attends great effusions and therefore (after the exclusion of tuberculosis, carcinoma or pyæmia) the prognosis is relatively good. In the average case dry pleurisy precedes and succeeds exudation. Recurrence and exacerbations rende the prognosis unfavorable. Death frequently follows from exhaustio and gradual cardiac failure. Partial absorption with persistence o temperature or with dyspnœa is indicative of tuberculous pleurisy Rheumatic pleurisies heal more readily than tuberculous. The autho has seen recovery of secondary carcinomatous pleurisy.

Issue.—1. Resorption.—Resorption may occur and even purule (pneumococcic) pleurisy may, in exceptional cases, be absorbed in pa without operation or rupture, leaving inspissated caseous accumulations. Serous pleurisies may be absorbed after three to six month during which time persistent friction is common over the lower part the chest.

2. Adhesions.—The thickness of pleural adhesions is more importa than their extent. Universal obliteration of the pleura by thin adhesi is often of no consequence. Retraction of the lung, compensat emphysema, pain, thoracic oppression, obliteration of the complementary pleural space, dulness, decreased vocal fremitus and breathing failure of Litten's phenomenon, decreased respiratory excursion and stagnation of bronchial secretion may occur when the adhesions are thick. If there is exudate around the heart, the apex beat may increase during the inspiration; this also occurs in bronchial catarrh (Eichhorst). Failure of the right heart and stasis may ensue, though most extensive adhesions sometimes exist without cardiac embarrassment. Peritonitis, mediastinitis, induration and obliterative pericarditis may complicate the case. In some instances the pleura calcifies or ossifies, leaving a deposit measuring three cm. or more in thickness.

3. Sudden Death.—Fatal syncope may result after exertion, after coughing or after the efforts of urination or defecation. Sudden death may occur from paralysis of the left ventricle, from cardiac thrombosis, pulmonary œdema, bending of the aorta, compression of the cava at the diaphragm, and rarely from pulmonary embolism caused by veno thrombosis. Rupture of fluid into the lung or bronchi, which m drown the patient during sleep, and death from hemorrhage into th effusion, from pneumothorax, or from brain embolism are even rarer.

4. Retractio Thoracis.—Retraction is characterized by increase resistance, decreased fremitus, decreased circumference of the affecte side and often by bronchiectasis. The percussion note is dull and th breathing is usually decreased, but sometimes is bronchial. The causative factors are atmospheric pressure, contraction of the pleural scars extension of the inflammation into the lungs, and pleurogenous (interstitial) pneumonia, which is also known as pleuritis deformans, an which sometimes divides the lung up into lobes or islets. Retractio is generally a slow process but the writer has seen marked deformit develop in three weeks.

5. Changes in Other Organs.—When retraction occurs, other orga may be dislocated. The liver may be higher when the right side involved, and in left-sided retraction the lung may shrink away from the heart, the spleen and the heart may lie higher and Traube's space may be increased. Organs displaced by exudate may in rare cases remain luxated after the exudate is absorbed. Pleural callosities may produce recurrent laryngeal paralysis which may also follow the pressure of a fluid exudate or enlargement of the glands. Other complications are acute and chronic infiltration in the lung; inflammation and degeneration in the chest muscles; extension to other serous surfaces; septicopyæmia; stasis in the liver, stomach, extremities, etc.; hemorrhages into the skin; chronic nephritis; amyloid degeneration and pulmonary, glandular, genito-urinary or miliary tuberculosis. Scagliari, quoted by Eichhorst, reports forty-seven cases of paralysis of the arm, probably due to brachial neuritis.

Diagnosis.—The physical findings only are final; symptoms are most unreliable. Diagnosis of the malady is the first step, and the second is the determination of its cause.

Differentiation.—1. The diagnosis of *pleuritis sicca* is established only by feeling or hearing the friction rub. (*a*) It must be distinguished from *rales*. The crepitant rale is largely inspiratory, and is heard as a number of small rales at the end of inspiration. Bronchitic rales or rhonchi are continuous. Coughing and deep inspiration alter or remove rales but the pleural rub is increased by pressure and deep inspiration, and is less extensive. It may disappear after a number of deep inspirations, because this makes the two surfaces temporarily smooth. (*b*) It must be differentiated from *sacculated breathing* and from the physiological, bilateral cracking in the chest muscles on deep breathing. (*c*) Distinction is necessary from *intercostal neuralgia*, which is intermittent, occurs in typical attacks and is limited to the interspaces; Valleix's points are usually but not always present; they exist at the angle of the ribs, in the middle of the axilla and anteriorly, and correspond to the points of exit of the posterior, lateral and anterior perforating branches of the intercostal nerves. Intercostal neuralgia is increased less on deep breathing than is pleuritic pain. (*d*) Pleuritis sicca must be differentiated from *caries of the ribs*, in which pain is localized directly over one rib and not between the ribs. (*e*) It must be distinguished from *rheumatism* of the thoracic muscles. This may be very difficult, for both affections may induce pain on breathing, tenderness and disturbed breathing; there may be a history of cold or exposure, or of repeated attacks. There is usually no fever or cough. Faradization decreases the pain, and movement increases it more than is the case in pleurisy. (*f*) The rub in *pericarditis* (*q. v.*) is more distinctly cardiac, and depends upon the cardiac movements. In pleurisy along the border of the heart, on holding the breath the pleuritic rub is heard near or over the heart, and decreases after five or six heart beats. On very deep inspiration the pleural rub ceases and the pericardial rub is increased. Differentiation is most difficult when there is pleurisy over the lingual lobe and left border of the heart, though pericarditis most commonly produces a rub at the base of the heart, especially over the conus pulmonalis art iosus.

2. *Pleuritis humida* (pleuritis effusiva) must be distinguished from *infiltration* of the lung substance (see table in DIAGNO OF PNEUMONIA). The fremitus is decreased in pleurisy, increa in consolidation; if a bronchial plug decreases the fremitus consolidation, it can usually be dislodged by coughing. In c solidation the dulness often corresponds to one lobe; the pleur dulness increases downward, though the upper border of duln in pneumonia is also sometimes irregular. Only when pneumo involves an entire lung is there any luxation of the heart, or volvement (in left-sided pneumonia) of Traube's space. In c solidation there is a close topographical parallelism between intensity of the bronchial breathing and the degree of dulness. Wh there is very slight bronchial breathing, pleurisy is probably not pres although it may exist in some cases. The sputum, the length of clinical course, dislocation of the heart, liver, and spleen, the in quency of chills, the ectasia shown by the cyrtometer and the solut by lysis, are indicative of pleurisy. Ægophony occurs more frequen in pleurisy along the upper border of the fluid, but is usually transito *Exploratory puncture* is the final test and also determines the natur the fluid. The presence of blood, carcinoma cells, streptococci, pn mococci, tubercle bacilli, etc., are of diagnostic value. (*b*) It must differentiated from *tumors of the lung*, which produce cachexia, n rowing of the chest behind on one side, and the characteristic exud this is often brown (Fraenkel) and sometimes chyliform, and cont fat globules (Quincke) and degenerated large cells which may sh mitoses; the glands above the clavicle may be enlarged as well as th in the mediastinum, which produce recurrent laryngeal paraly Tumor particles are rarely obtained by puncture or in the sputum, are only found in carcinoma, for sarcoma rarely ulcerates. W tumors of the lung are suspected, a trocar rather than a needle sho be used. There is more resistance to puncture in tumor than i pleuritis. Unverricht observed the development of carcinoma at th seat of puncture. In tumors there is less displacement, less invasio o Traube's space and less widening of the chest than in pleurisy, and the breathing is often bronchial, the flatness is intense and the fremitu is increased. (*c*) *Hydrothorax* (*q.v.*), which is usually described as bilateral, may nevertheless be unilateral, especially in disease of the liver, kidneys and heart, where right-sided hydrothorax is observed. Unilateral hydrothorax is sometimes caused by obliteration of the other pleural cavity by adhesions. The fluid readily shifts its level on change of posture, which does not occur in pleurisy. Salicylic acid, iodine and potassium iodide are said to pass readily into transudates, and but slowly and in small amounts into exudates (Rosenbach and Pohl), though this claim has been disproven by Weintraub, Leuch and Feldmann. (The iodine is detected by adding nitric acid and shaking with chloroform, which becomes red colored.) In hydrothorax, the

specific gravity is below 1,014, the albumin is less than 3 per cent. and the sediment shows few white cells and some endothelial cells, which are not pathognomonic of hydrothorax, as has been claimed. Bacteriological tests are negative. (*d*) The presence of *hæmothorax* is determined only by puncture.

3. *Circumscribed pleurisy* in some instances requires differentiation. (*a*) It differs from *pericarditis* in that it shows a more irregular outline, the apex beat is not altered and there is no cardiac insufficiency or pericarditic friction. (*b*) It may be distinguished from *lung cavities*, which show dulness when full of secretion and resonance when the secretion is voided; the fluid obtained by puncture (which is made under a wrong diagnosis) has a higher specific gravity and a larger percentage of fat than that of pleurisy. (*c*) Differentiation from *splenic tumors* is determined by respiratory excursion and palpation of the lower edge of the spleen. (*d*) *Liver enlargements* rarely occur solely upward, save in echinococcus and in abscess of the convexity; these are prone to occur anteriorly, and pleurisy is more frequently detected posteriorly. In tumors of the liver there is usually respiratory excursion which is absent in pleurisy. In rare instances of very large right-sided pleurisy there is a furrow between the costal arch and the liver downward (Stokes). (*e*) It should be differentiated from *subphrenic abscess* (*v.* LOCALIZED PERITONITIS), which may be intraperitoneal (as from liver abscess) or extraperitoneal (*e. g.*, perinephritic). The liver stands higher in the chest because of the paretic diaphragm. There may be a history or physical signs of previous disease of an abdominal organ. The needle, when thrust toward the abscess, reaches the pus only when thrust deep in the lower interspaces. The pus has frequently a fæcal odor. The manometer shows increased pressure during inspiration and decreased pressure during expiration, the converse of the findings in pleural effusion. (*f*) It must be distinguished from *abscess of the liver*. Pleurisy, either serous or suppurative, may complicate abscess of the liver. Differentiation is easy when abscess of the convexity is located forward; when it is situated posteriorly, aspiration alone will distinguish it; the pus shows liver cells, amœbæ, cholestearin, and bile.

Prognosis.—The *immediate mortality* is about 5 per cent., though a prognosis cannot be made in the first week or two. (*a*) The *etiology* is an important factor. (*b*) The *nature of the fluid* is of prognostic import. Fibrinous pleurisy (except when it occurs at the apex of the lung) is the most favorable type. Prince Ludwig Ferdinand considers the prognosis in the effusive forms as follows: (i) most favorable in metapneumonic serous or purulent pleurisy; (ii) next most favorable in serous pleurisy due to the staphylococcus, (iii) less favorable in staphylococcus empyema, (iv) least favorable in tuberculous, fœtid or putrid forms. The prognosis is usually poor in hemorrhagic, chyliform, peracute and bilateral (generally hemorrhagic or purulent) effusions. It is more favorable in effusions which are relatively rich in solid constituents (Méhu and Bernheim); effusions containing little solids are apt to recur. When the freezing point of the exudate is found by cryoscopy to be below that of the blood, spontaneous absorption is unlikely to occur (Rothschild). From a study of 116 cases of effusive pleurisy, Courmont (1906) concludes that the mortality is 25 per cent. in effusions which have an agglutinating power for the tubercle bacillus, and 75 per cent. in effusions which have not. Since the time of Hippocrates, the belief has prevailed that right-sided are less favorable than left-sided effusions. (*c*) The persistence of temperature and sweats, anæmia, recurrences, and external rupture are unfavorable. (*d*) *Sudden death* is more frequent than it is usually believed to be. The author saw three cases in one year.

Opinions vary as to the *ultimate prognosis*. Eighty-seven per cent. of cases become tuberculous in one to two years (Fiedler); 33 per cent die in five years (Bars); 66 per cent. are curable (Osler); 15 per cent. die of tuberculosis, 90 per cent. are in good health after two to five years and 80 per cent. are healthy after five years (Cabot).

Treatment.—*Etiological* treatment can be applied only to the rheumatic and syphilitic forms.

Treatment of *fibrinous pleurisy*, which remains fibrinous, or in the fibrinous or first stage of exudative pleurisy. *Pain* is the main indication; morphine may be given in severe cases, especially when coughing is severe; the ice-bag, poultices, blisters, cups and hot fomentations give little relief when the pain is acute; blisters may eventually cause as much pain and discomfort as the disease itself; the pleura should be splinted by three or four strips of adhesive plaster two inches wide, overlapping the median line before and behind by about three inches, and running around the chest horizontally and obliquely; these should be applied with considerable pressure during expiration that the lung in its lower part and the pleural surfaces may be well immobilized; zinc oxide adhesive plaster is least irritating to the skin. Personal experience with the early use of salicylates, which are advocated by Aufrecht and Fiedler, has not been satisfactory. The patient should be kept in bed. The use of leeches sometimes relieves the pleural congestion.

In *serous pleurisy*, the patient should be kept in bed, and the cough and fever should be treated expectantly. *Laxatives* are given solely to obviate straining at stool, which may induce cardiac weakness; purgatives do not reduce the amount of the effusion. If at the end of the second week the effusion shows no sign of being absorbed, various measures are indicated; Schroth's treatment consists in limiting the amount of fluid ingested to about one quart (1000 c.c.) daily, but it has few adherents, as the method gives no results and exudation is an active, not a passive process; tincture of iodine may be applied, but it is seldom beneficial, and when old is distinctly a local irritant; potassium iodide, digitalis, drastics to produce copious evacuations, pilocarpine (combined with cognac to support the heart), cotton jackets and sweats are often given. Of late a salt-free diet (dechlorination) is advised, as in nephritis (*q. v.*). The author cannot agree that any of these measures are helpful and believes pilocarpine is always dangerous. Transudates may be removed in this way but not exudates.

Aspiration.—Trousseau's indications were (*a*) the vital indication (when life is threatened, because of large exudates); (*b*) moderate effusions with slow resorption, and (*c*) persistent or residual exudates. The author believes that *early puncture* (thoracocentesis) and aspiration are indicated in every case, where two quarts or more (2,000 or 2,500 c.c.) of fluid are effused, whether the temperature is still high or not, and whether pressure symptoms or dyspnœa are present or not. This amount of effusion causes dislocation of the heart, liver or spleen, compression of the lung, or ectasia, as shown by the cyrtometer; intrathoracic pressure may cause sudden death, sometimes without any warning symptoms of cardiac insufficiency. Early puncture does not irritate the pleura nor favor recurrence or suppuration. Early and *repeated puncture* obviates compression of the lung and atelectasis as well as compression and torsion of the large arteries. Aspiration was first advocated by Wyman and Bowditch of Boston more than 50 years ago.

Method of Aspiration.—After the usual surgical antisepsis, a small trocar or large aspirating needle is introduced, which has an elbow connecting with a rubber tube, to siphon the fluid to a vessel below, or connecting with a bottle which can be exhausted by a pump. There is no absolute rule as to the site of puncture. This must be governed solely by the physical signs; for instance, adhesions which hold the lung to the surface vitiate any set law; fluid is usually obtained most advantageously between the scapular and post-axillary lines and low down in the chest. The trocar should be introduced suddenly, to avoid bending of the spine which narrows the interspaces; it should be introduced perpendicularly to the chest wall and not obliquely, in order to avoid injuring the intercostal artery, as a result of which the author once saw fatal hæmatoma and hæmothorax. Whisky and hypodermics should be at hand, because of the possibility of syncope. The use of the pump is necessary in but 10 per cent. of cases, for the fluid can usually be siphoned readily; a tube is attached to the needle or trocar, filled with boric acid solution, clamped with an artery forceps and unclamped, when it is placed in a basin containing the same solution and at a somewhat lower level than the seat of puncture. The fluid, if pumped, should be withdrawn gradually; caution is necessary lest air be pumped the wrong way, *i. e.*, into the pleura. The amount removed need not be great, as moderate relief of intrapleural tension often promotes absorption. The amount to be withdrawn is estimated somewhat arbitrarily at one quart (1,000 c. c., Naunyn and Dieulafoy), or three pints (1,500 or 1,600 c. c., Fraenkel and Bowditch). Complete or nearly complete evacuation may cause the accidents enumerated below and is thought by Litten to induce miliary tuberculosis. Aspiration should be *discontinued* when the exudate becomes bloody (from lung injury), when friction develops, when pain is felt or when coughing (from congestion of the relaxed lung) begins.

Repetition of puncture depends on the results attained; it may be repea every two or three days. *Favorable results* are slowing and strength ing of the pulse, relief of oppression, increased diuresis, and impro appetite; but relief of pressure on the lung and absorption of exudate are of the greatest importance. *Unfavorable results* are syncope due to cerebral anæmia which in turn results from restorati of circulation in the collapsed lung, which contained little blood befo it was relieved by the puncture, a relatively uncommon occurren (*b*) pulmonary œdema, the albuminous expectoration described Terillon, Besnier, Johnson, Duffin, and Scriba, which is due to t sudden withdrawal of too much fluid (two to five liters) at o tapping; it should take a half-hour to evacuate one and a half quar or 1,500 c. c. (Fraentzel); œdema pulmonum is treated most efficacious by hypodermics of morphine, gr. ¼ and some clinicians advise givi it before aspiration; (*c*) sudden death from pulmonary embolism cerebral embolism; the latter results from discharge of clots fro vessels in the relaxing lung, and causes the so-called pleuritic hem plegia; some of these cases are hysterical hemiplegia; (*d*) injury to th heart, liver or spleen; (*e*) pneumothorax or subcutaneous emphysem may occur though the author has never seen either of them. (*f*) Th author once witnessed an extensive hæmothorax and subcutaneou hæmatoma in a pleural puncture, in which the trocar was impatientl thrust in and out during an unsuccessful thoracocentesis. (*g*) Fi brinous bronchitis has apparently followed paracentesis. When repeate punctures fail to produce the desired results, the advisability o operation (as in empyema) should be considered.

Empyema in all cases necessitates operation (practised by Eurypho and after him by Hippocrates). Some cases may recover spontaneously but this cannot be depended upon. A day before operation, aspiratio is indicated when the fluid is under high tension, thereby relieving th heart. Some surgeons advocate treating tuberculous empyema withou operation, as Volkmann treated other cold abscesses; Krönlein ob served, after operation on these cases, recovery in 36 per cent., improve ment in 28 per cent. and death in 36 per cent. Results depend on th etiology and bacteriology. Though the mortality in Velpeau's case was 100 per cent. and that of Dupuytren was 92 per cent., at th present time it is but little over 1 per cent.

Putrid pleurisy should be treated like empyema.

Hemorrhagic effusions may necessitate injections of gelatin su cutaneously; the amount aspirated should not exceed one pint (5 c. c.) and aspiration should be repeated as seldom as possible.

After-treatment is directed toward prevention of thoracic retractio to which end gymnastics and deep breathing are essential. A patie having suffered from a "primary" effusive pleurisy should be treate as one with latent tuberculosis.

(B). PNEUMOTHORAX.

Definition.—Pneumothorax is an accumulation of gas or air in the pleural sac. It was named by Itard of Paris (1803) and was described fully by Laennec (who made the first diagnosis) and Skoda. As it excites pleurisy in more than half the cases, the following varieties are distinguished: *pyo-*, *sero-*, and *hæmopneumothorax.* There is no name for putrid pneumothorax.

Etiology.—*Diseases of the lungs and pleura* cause about 95 per cent. of all cases. In Biach's collection of 918 cases in Vienna, pulmonary *tuberculosis* was causal in 77 per cent., gangrene in 7 per cent., empyema 5 per cent., trauma 3 per cent., bronchiectasis and abscess each 1 per cent., the etiology was unknown in 2 per cent. and the remaining 4 per cent. was due to emphysema, infarct, thoracocentesis, echinococcus and contiguous disease perforating into the pleura, as gastric or œsophageal ulceration, or caries of the sternum. By other writers (*a*) tuberculosis is thought to cause 80 per cent. or even over 90 per cent.; Mosheim found that 84 per cent. were tuberculous, of which 6 per cent. were curable; acute forms are more important than chronic tuberculosis in which there are protecting pleural adhesions; pneumothorax develops in two (Drasche) to six (Eichhorst) per cent. of tuberculous cases. (*b*) Ulcerative lesions of the lung, as gangrene, abscess, bronchiectasis, tumors, parasites and infective infarcts may naturally erode the pleura and admit air. (*c*) Trauma, rupturing of the lung directly or by *contra-coup*, thoracocentesis or stab wounds, occur far less frequently than one would expect. (*d*) Empyema; it may rupture into the lung without allowing ingress of air from the opposite direction. (*e*) Rupture of a healthy lung after coughing or muscular straining is a possible though very exceptional cause. Pneumothorax has followed violent coughing in pertussis.

Perforative lesions in the mediastinum, œsophagus, peritoneum, stomach or colon and subphrenic abscess are uncommon (*v. s.*) causes.

Pneumothorax may occur *spontaneously*. It is always possible, however, that a point of rupture in the lung is overlooked or has healed over, and the fact remains that air enters the pleura chiefly from the lung or from a gas-containing organ. The view held by Oppolzer, Biermer and others that putrefaction may occasionally develop primarily (without rupture) has received confirmation by discovery of the *Bacillus aërogenes capsulatus* and other gasogenic organisms (Lewy, Welch and Fraenkel).

Pneumothorax occurs largely in *adults* and four to seven times as frequently in *males* as in females. Eichhorst and Drasche find it more often on the right side, Powell on the left, and West on either side with equal frequency.

Pathology.—When the chest is opened, air or gas escapes with a force sufficient to blow out a candle. The volume of air may exceed 2,000 c.c., and its tension, which was studied first by Wintrich and then by Weil, compresses the lung and causes it to collapse, dislocat the heart and mediastinum and depresses the diaphragm and su phrenic organs. The gas may be odorless or fœtid. The pathologic and clinical findings vary (*a*) as the gas is encapsulated or occupi the entire pleural cavity; (*b*) as the air is aseptic (producing a pu pneumothorax, or one with slight exudation) or is infected by tuberc bacilli, pyogenic organisms, colon bacillus, or monads according the primal disease; microörganisms excite serous, purulent or putr pleurisy; (*c*) as the air-containing cavity is "closed," "open" (in a bronchus, or sometimes also open externally) or possesses a valv thus allowing passage of air in one but not in the opposite directio These points will be considered below.

Symptoms.—The *onset* is usually *sudden,* for rupture may occ spontaneously or may follow coughing or muscular exertion; it characterized by *sudden pain in the side, cyanosis, dyspnœa* and *co lapse,* which are due to reflex action on the vagus; there may be sudde evacuation of pus from the breaking into the lung of an empyem an urticaria (Gerhardt, Senator) occasionally develops, just as a tox rash may follow echinococcus rupture; decreased diuresis, albumin uria, stasis and subcutaneous emphysema are sometimes observe The onset may be insidious for pneumothorax sometimes constitut an unexpected post mortem finding. In other instances the previou history or the course may justify a presumptive diagnosis, as in case clearly tuberculous; and in still other cases, a latent lung lesio (a small caseous focus) may burst and thus produce its first sympto

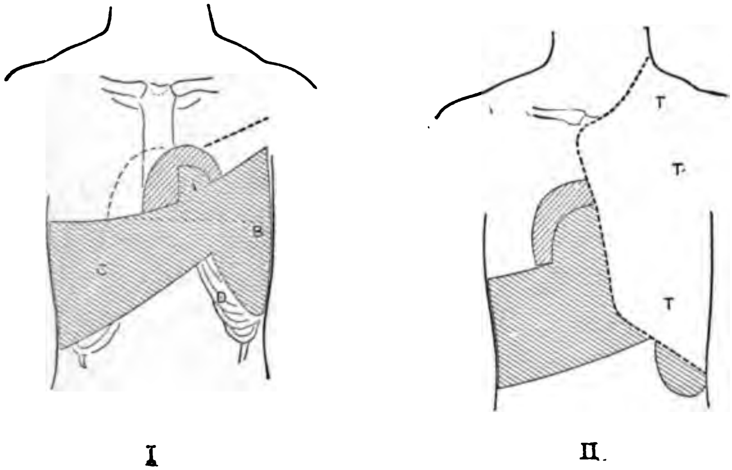
Physical Findings.—1. **Inspection.**—(*a*) The affected *side is di tended* 2 to 4 inches (5 to 10 cm.) and *immobile,* and contrasts sharp with the widely-moving sound lung. (*b*) The *interspaces* are *oblite ated.* (*c*) The *heart* and *liver* or spleen are *dislocated.* (*d*) There a *dyspnœa* and *cyanosis.* (*e*) The *attitude* is characteristic; the patie most often lies *on the affected side* to afford ample play to the soun lung; sometimes there is orthopnœa and if the point of rupture ope into the lung or bronchus, a peculiar decubitus may be assumed, i order to drain the pleura. (*f*) The *x-rays* show that the *lung flattened* near the spine, that the *diaphragm is depressed* and *motion less,* and they also show the shadow of the effusion.

2. **Palpation.**—(*a*) *Vocal fremitus is absent,* unless adhesions hol part of the lung to the chest wall. (*b*) The *chest wall feels resistan* and (*c*) sometimes *splashing,* fluctuation or a "pillow sensation" noted. (*d*) The *dislocated apex beat* and the edge of the liver or splee are felt.

3. **Percussion.**—Somewhat different results are obtained by pe cussion according to the tension of the gas and of the chest wall (*a*) In *open pneumothorax* the note is full and tympanitic, and eve metallic when percussion is made on a solid object, as a coin; thi metallic reverberation was called *bruit d'arain* by Trousseau; cracked pot resonance and Wintrich's change of note may be elicited whe the mouth is opened. (*b*) In *closed pneumothorax* the note is full an

loud; it is tympanitic when there is moderate pressure and the intercostal muscles are relaxed; again the note may be dull (Skoda) if the tension is great, or if the chest muscles are spasmodically contracted, as in sudden pneumothorax (vagus reflex action). (*c*) *Flatness* over the lower thorax usually results from pleural effusion. (This dulness may occur without coincident pleurisy, and, conversely, the author has seen tympany prevail over the entire side, even when there was much coincident pleural effusion.) (*d*) On change of posture, a *changing level of the fluid* is readily elicited, the gas being found above and the fluid below, for the gas acts in this respect as a spirit-level; this change is far more important than in simple pleurisy, as there are no adhesions. (*e*) The *pitch* over the gas-distended part *changes with change of posture* (Biermer's change of note). It becomes deeper as the patient sits up, because the vertical dimension of the chest is thus increased by the pushing down of the diaphragm. (*f*) The *heart dulness* is *replaced by resonance* in left-sided pneumothorax, and is found to the right of the sternum; in right-sided pneumothorax it is pushed to the left axilla. (*g*) The *dulness* of the *liver* or *spleen* is *pushed* well *downward*.

FIG. 36.



I. Left-sided effusive pleurisy, showing relative heart dulness (A), effusion (B), liver flatness (C), and fluid impinging on Traube's half-moon space, of which but a small part remains resonant (D).

In larger effusions reaching, *e. g.*, up to the dotted black line, the relative heart dulness may be pushed over to the dotted line (over C).

II. Left-sided pneumothorax marked by tympany (T. T.), pushing over the mediastinum and also the heart to the right, and pushing down the left lobe of the liver and the spleen.

4. **Auscultation.**—(*a*) The *breath sounds* are usually *absent* in the closed variety of pneumothorax, and this, with ectasia and hyperresonance, is most characteristic. In the open and sometimes in the closed form, the breath sounds are *bronchial*, amphoric or metallic, the voice sounds, especially under the clavicle, are metallic (metallic bronchophony), and coincident rales in the bronchi are also metallic.

(*b*) Air passing through the point of rupture may produce curic *snapping or whistling sounds;* the point of rupture is oftenest at t lower anterior part of an upper lobe, *i. e.*, between the second ar fourth interspaces, and between the mammary and axillary lines. (The *gutta cadens* (the metallic "falling drop") may be due to drippin of fluid from fibrin shreds as the patient's posture is changed, or t rales. (*d*) The Hippocratic splashing (*succussio Hippocratis*) elicited by placing the ear to the chest and sharply shaking the patien it is sometimes heard at some distance from the patient, and in thr cases patients have spontaneously called the author's attention to "splashing in the chest"; it is oftenest heard in the side or in t interscapular area. (*e*) The *heart tones* may be *metallic*, and the hear action may even cause splashing sounds.

Diagnosis.—The ectasia, the resonant note, suppressed or ampho breathing, cardiac and other luxations and succussion are most ch acteristic.

Type.—Contrary to the usual statements, Weil recently proved th dislocation occurs in all types, though it varies in degree. (*a*) In t *open form* Wintrich's change of note on opening the mouth, metal sounds, the fistula murmur, periodic expectoration of large amou of pus, moderate cardiac and other dislocations and no manomet increase of the tension in the pleura are distinctive. Colored flu (methyl blue), if injected into the pleura, is coughed up. (*b*) T *closed type* is distinguished by increased manometric tension, absen of breath sounds, marked ectasia, change of note on sitting but not opening the mouth, and marked luxations. (*c*) In the *valvular for* air may enter the pleural cavity but cannot escape back and therefo pus cannot be voided. Some of the auscultatory findings of the op type are noted. The manometer shows increased pressure. T fistula may become permanently closed by organic change (organ type), so that aspiration of the air is not followed by further accum lation; or the fistula may be only mechanically closed, and air ma accumulate again after its aspiration.

Differentiation.—(*a*) *Cavities* may be simulated by circumscribe pneumothorax, but in cavities the interspaces are sunken, the fremitu is increased (when they are empty), luxation is absent and the Hippo cratic succussion and coin sound are extremely rare. (*b*) *Pleuri* can be distinguished on careful examination. (*c*) *Meteorism* is at onc excluded by filling the stomach or colon with water. (*d*) *Diaphrag matic hernia* is very frequently confused with pneumothorax; out 433 cases collected by Grosser only 12 were recognized. The *x*-ra will determine the condition, as in Dehio's recent case. Respirato excursion is present and the tympany and metallic sounds are usual accompanied by such symptoms of marked incarceration as vomitin colic, obstipation and indicanuria. The intestines push the heart the right. Ninety per cent. of cases of diaphragmatic hernia are lef sided. (*e*) *Pyopneumothorax subphrenicus* (*q. v.* under PERITONITI LOCALIZED FORMS), especially described by Leyden, results fro abdominal lesions, such as cancer, ulcer, appendicitis, disease of the gall-tracts, trauma, and foreign bodies whose history is at least suggestive. Thoracic symptoms, as cough, sputum, and dyspnœa, are not conspicuous, at least early in the process, and the lungs may show perfect excursion, though the diaphragm is sometimes paretic. The manometer shows that the pressure is increased during inspiration and decreased during expiration, the converse of which is true in pneumothorax. The *x*-rays show that the diaphragm is above the fluid, while in pneumothorax it is below the fluid.

Prognosis.—The prognosis varies with the cause, the condition of the lungs, and the character of the fluid, but it is always serious. Cases following exertion in a healthy lung, trauma or infarct (when operated) are relatively favorable. The closed and open are more auspicious than the valvular type. In tuberculosis, pneumothorax seems to arrest the tuberculous focus if it is small (Hérard, Toussant, Fraentzel) and if no mixed infection occurs, but even in these cases complete ultimate recovery is infrequent. Eichhorst observed one case which lasted for five years. Gabb records a case which recovered and relapsed four times, the attacks being two to four years apart. According to Weil 25 per cent. die within a week and 50 per cent. within a month; West observed a mortality of 70 per cent., of which three-quarters died within two weeks, nine-tenths in less than a month and one case in twenty minutes.

Treatment.—The results of treatment are disappointing. (*a*) Narcotics and stimulants are imperative at the time of the rupture, for collapse, dyspnœa, and cyanosis. (*b*) Aspiration is of most value in the serous forms; however, it has been said that we may tap or operate but never aspirate. (*c*) The radical operation is indicated in putrid or purulent pneumothorax. Though some physicians prefer not to interfere in the tuberculous cases on the ground that the compression exerted upon the lung by the pneumothorax is semicurative, others advise operation; Leyden reported 66 per cent. and Richardière 50 per cent. of recoveries.

(C). HYDROTHORAX.

Definition.—Hydrothorax is a transudation of serous fluid into the pleural sac.

Etiology.—(*a*) *Stasis*, from cardiac, pulmonary or vascular factors may be the cause. In failure of the right heart the superior cava is imperfectly drained, which congests the vena azygos and hemiazygos, and this leads in turn to pleural transudation. It may occur alone or precede dropsy but is oftener a part of general dropsy. Local venous obstruction, by pressure or thrombosis, may induce hydrothorax, as from mediastinal tumor or aneurysm. (*b*) *Blood changes*, resulting from multitudinous anæmic, marantic or cachectic states, may be causative; nephritis, cancer and blood diseases may be cited as specific types. Under this caption may be brought diseases—often identical with the above—which alter the *walls of the vessels.*

Symptoms and Diagnosis.—1. The *symptoms* of hydrothorax do not include pain or fever. The disease accentuates, sometimes considerably, the dyspnœa attending the causal stasis or cachexia. The amount of fluid varies from ounces to quarts (or even to two gallons).

2. The *physical signs* are essentially those of moderate effusive pleurisy, except that the rub is absent; the fluid shifts with much greater facility on change of posture and produces a moderate compression of the lungs, and luxation of the heart, liver and spleen. The transudate is usually bilateral (in 83 per cent., according to J. Dutton Steele), but is sometimes unilateral and often right-sided, in cardiac, renal and hepatic disease, or when previous adhesions obliterate the opposite pleura.

3. The *fluid* is clear, slightly green, possibly moderately blood-tinged if it develops during the death agony, and shows a specific gravity of about 1,010 to 1,012; it contains but little fibrin and at the most develops only light delicate flocculi. The potassium iodide test is unreliable (*v.* DIAGNOSIS OF PLEURISY), and centrifuging brings down only a few leukocytes, endothelial cells and red disks, but no bacteria. Right hydrothorax may prove very obstinate, probably because a low grade of inflammation eventually supervenes.

The prognosis and treatment are those of the cause: aspiration often prolongs life.

(D). HÆMOTHORAX.

Blood effusion into the pleura is a rare malady. It may be caused by trauma, scurvy or allied blood diseases, by rupture of aneurysm of the aorta, by ulceration of the pulmonary or pleural veins and by various necrosing lung diseases. It may occur with pleurisy and pneumothorax and has resulted from thoracocentesis. Its symptoms are essentially those of hydrothorax; it is suspected only by a sudden onset with signs of acute anæmia, and is proven only by the exploring needle. Its treatment is symptomatic; for the occasional syncope, the usual stimulants should be given; ice should be applied to the chest, ergot should be given, or a 2 per cent. gelatin solution should be injected for hæmostasis; and operation should be invoked when indicated by the causal lesion.

(E). CHYLOTHORAX.

A very rare affection, in which true chyle or chyliform fluid is found in the pleura. Its etiology is much disputed; (*a*) in the genuine chylous cases true chyle containing sugar is found, though of late years sugar has been found in very many forms of serositis and in most transudates; only 30 cases of this type are recorded. In 13 cases (of which 4 died and of which 9 were right-sided), traumatic rupture of the thoracic duct has been found. In others the duct has been obstructed by thrombosis of the subclavian vein or by glands and tumors. (*b*) In a second group, the pseudochylous (chyliform or adipose), there is no chyle and the milky appearance is due to fatty globules or minute albuminous granules held in suspension. This variety is chiefly tuberculous or carcinomatous. It has been thought that old empyemas may become chyliform. The characters of both forms of fluids will be considered under chylous and adipose ascites, with which they are frequently associated. The signs are those of transudation or exudation. Sixty per cent. of cases occur on the right side.

(F). PLEURAL TUMORS.

Pleural tumors are nearly always secondary to pulmonary, mediastinal or extrathoracic growths. Carcinoma and sarcoma are the most frequent forms; enchondroma and dermoids are very uncommon. The pleura is involved by contiguity or metastasis. Wagner, Fraenkel and Riedel described a primary endothelioma of the pleura, but, as stated under the etiology of pleurisy, it is considered by some as solely inflammatory; clinically it presents the picture of chronic pleurisy plus occasional metastatic deposits in the liver, lungs, kidneys, muscles and lymph glands. The signs are those of lung tumor plus those of pleurisy; the clinical diagnosis of primary tumor is at the best but probable, even if tumor particles are obtained by puncture or if a tumor develops at the seat of puncture.

DISEASES OF THE MEDIASTINUM.

(A). MEDIASTINAL TUMORS.

Mediastinal tumors are very rare (1 in 10,000 medical cases). They are usually primary. Sarcoma is the most common form, and then carcinoma; lipoma, substernal struma, thymic growths, fibroma, osteoma, enchondroma, echinococcus, dermoid (57 cases collected by R. S. Morris previous to 1905), teratoma, gumma, tubercle, leukæmic, pseudoleukæmic and chloromatous adenopathies are rarer forms. Sixty per cent. occurs in males between twenty and thirty years of age.

Symptoms.—Symptoms develop gradually. (1). There may be *local signs.* (*a*) Bulging may be present in the jugulum or under and near the sternum, especially in lymphosarcoma, or it may show in the anterior mediastinum, and cause stretching of the skin. (*b*) There may be pulsation, imparted by the heart, aorta or its branches. (*c*) The tumor or secondarily involved glands may be palpable deep in the jugular notch. The vocal fremitus varies. (*d*) The sternum may be exquisitely tender from erosion. (*e*) Dulness may be elicited under or near the sternum, or, if the posterior mediastinum is involved, in the back; the dulness may blend with the heart, suggesting aneurysm or effusive pericarditis, though it is more irregular than in either of these affections. (*f*) Auscultation gives variable results; sometimes there is bronchial breathing, or if the bronchi are closed the breath sounds may be absent.

2. *Compression symptoms*, as in aneurysm, may predominate in tumors of the posterior and middle mediastinum, though local physical findings may be slight or absent. (*a*) The vagus and its branches may be affected. Paroxysmal asthma and orthopnœa are common. Dyspnœa is the earliest and most frequent symptom of mediastinal tumor, is partly due to compression of the vagus, and partly to tracheal, cardiac and pleuritic involvement. Other vagus symptoms are brazen cough from paralysis of the vocal cords, tachy- or bradycardia, hiccough, vomiting or œsophageal spasm. (*b*) The trachea or bronchi may be compressed from without or may be occluded by ingrowth into their lumen, in which case the symptoms of tracheal or bronchial stenosis may be pronounced. Tracheal tugging and shrinking of one side of the chest (instead of bulging) may be noted. The sputum may contain blood, tumor tissue, or hair. The larynx may show lateral deviation. (*c*) The vessels may be involved. The arteries of the neck may show a systolic stenotic murmur, but the veins are more often compressed, especially the innominates or the superior cava, which cause bilateral or unilateral œdema and cyanosis of the face, neck and arms; enlargement of the veins on the thorax indicate the collateral circulation. (*d*) The heart may be compressed and dislocated, downward and outward if the growth emanates from the anterior mediastinum, forward if from the posterior mediastinum. Dislocation of the liver and spleen are less common. (*e*) The œsophagus may be so compressed and dysphagia may be so extreme that death from inanition results. (*f*) The sympathetic ganglia may show symptoms; inequality in the pupils is uncommon. (*g*) Neuralgias, muscular paralysis or even spinal paraplegia may result.

Diagnosis.—A diagnosis is either positive from the signs, or presumptive from the symptoms. Dyspnœa, ectasia, irregular substernal dulness, venous stasis, bronchial stenosis, heart luxation and the *x*-rays picture make the diagnosis probable; puncture may both eliminate the possibility of aneurysm and withdraw tissue or dermoid material (Türck); the sputum sometimes voids tumor particles, hooklets, and hair. Deep aneurysm (*v.* DIAGNOSIS OF ANEURYSM) is most difficult to eliminate. The irregular dulness and the apex beat at its extreme limit to the left exclude pericarditis. Tumors of the lung and pleura produce less pressure symptoms, though this differentiation is of less moment.

Prognosis and Treatment.—The prognosis is unfavorable, as malignant growths cause rapid emaciation, and death from inanition, compression, exhaustion or pneumonia results within a few months. Benign tumors run a longer course, sometimes five to seven years, or even forty in Lebert's dermoid case. Treatment is palliative. A few successful operations are reported; in twenty operated cases of dermoid, 70 per cent. recovered (R. S. Morris).

(B). MEDIASTINITIS.

Acute inflammation is uncommon, but may follow various local lesions, as pleurisy, pericarditis, pneumonia, acute lymphadenitis and kindred processes (*v.* MEDIASTINAL PLEURISY) by contiguity or by metastasis. (*a*) Acute nonsuppurative cases begin with chill, fever, pain in the mediastinum and tenderness anteriorly over the sternum or high in the back; there may be œdema over the sternum; dysphagia, dyspnœa and vague cardiac symptoms may develop. The diagnosis is difficult, but a presumptive diagnosis is made from acute inflammatory symptoms with mediastinal localization. The prognosis is grave and the treatment is symptomatic; an ice-bag should be applied over the sternum and opiates should be given for pain. (*b*) Of *mediastinal abscess,* Hare (*Prize Essay on Mediastinal Disease,* 1889) collected 115 cases, in most of which the anterior mediastinum was affected. They occurred chiefly in males. Acute cases, which are most often traumatic or sequential to the eruptive diseases, were found more frequently than chronic cases, which were usually tuberculous (*v.* MEDIASTINAL PLEURISY).

Symptoms.—The symptoms are local (mediastinal), as in simple acute mediastinitis, and general (septic). A throbbing retrosternal pain is common and dyspnœa may result from pressure by large pus pockets. Œdema, fluctuation and pointing in the upper interspaces or in the jugular notch may be noted. Fatal hemorrhage may follow simultaneous erosion of the aorta and bronchus. External rupture may occur in the second interspace (Daudé) or internal rupture into the air passages, bloodvessels, œsophagus or pleura; deep burrowing to the abdomen has occurred. The exploring needle is of great diagnostic aid in doubtful cases. Operation is indicated: Auvray (1904) found in the literature, 36 operations with 33 recoveries.

(*c*) *Chronic mediastinitis* (mediastinopericarditis) is discussed under adhesive pericarditis.

(C). MEDIASTINAL HEMORRHAGE.

Small hemorrhages of no clinical importance may result from "blood dissolution," as in icterus, hemorrhagic blood diseases or acute infections. Larger hemorrhages may follow trauma or erosion of the l vessels, both of which usually overshadow the mediastinal inciden

(D). INTERSTITIAL EMPHYSEMA.

(See DIFFERENTIATION OF EMPHYSEMA.)

SECTION IV.

DISEASES OF THE DIGESTIVE TRACT.

DISEASES OF THE MOUTH.

(A). CATARRHAL STOMATITIS.

Etiology.—Simple, acute or erythematous stomatitis is the most frequent form. In children it may develop with dentition, improper feeding, rude cleansing of the mouth, and digestive disorders, especially in those who are poorly nourished; it is part of various infections, notably of the exanthemata and syphilis. In adults it results from thermal, mechanical and chemical irritants; hot food, alcohol, tobacco, various chemicals (iodine, mercury, arsenic, copper, etc.), acidulous vomiting, inflammatory extension from the salivary glands, carious teeth, nose and throat disease, uncleanliness, anæmias and stasis are among its causal factors.

Symptoms.—Symptoms are chiefly redness, swelling and increased secretion, and in the acute forms burning pain which makes eating difficult. The increased saliva is usually acid and irritates the lips or chin if it flows over them. The gums are turgid (*gingivitis*) and the tongue broadened, lax and therefore indented by the teeth, as is also the buccal mucosa. The tongue is generally coated. Vesicles sometimes form and break down into small erosions which heal with cicatrices. The whitish areas sometimes seen are epithelial thickening. Fever, thirst and general symptoms are absent, except in some infantile cases. Acute stomatitis lasts but a week or two, but may become chronic. Transient stomatitis is almost physiological in the new-born.

Treatment.—In children the mouth should be gently washed after each nursing. Cold water or ice mitigates pain. Boracic acid is excellent and safe. In adults the teeth should be cared for. Tincture of myrrh and tincture of rhatany—equal parts—are excellent astringents. Stronger antiseptics may be used in adults but are dangerous in children, who may swallow them; for instance, 2 per cent. sodium salicylate, 2 per cent. chlorate of potash, 1 per cent. carbolic solution; erosions may be touched with 1 per cent. silver nitrate.

(B). STOMATITIS ULCEROSA.

Etiology.—Though probably a mycotic affection, its bacteriology is unknown. In some cases the same microörganisms are found as in Vincent's angina (See DIAGNOSIS OF DIPHTHERIA and ACUTE PHARYNGITIS). Sporadic cases are more common than epidemics, which may occur in asylums or prisons. It may be induced by malnutrition, uncleanliness, oral or dental disease, phthisis, leukæmia, diabetes, toxic factors (principally mercury), and occurs especially at dentition and more in city than in country children.

Symptoms.—The symptoms are those of catarrhal stomatitis plus necrosis and then ulceration. The gums, especially about the incisors and canines, are red, swollen, loose and spongy; they bleed readily, secrete pus and ulcerate; over the ulcers a membrane develops. The teeth are exposed, loosened and perhaps fall out. Necrosis of the jaw sometimes results. The tongue and cheek ulcerate less often and the pharynx is nearly exempt. The breath is fœtid, the saliva is increased, the submaxillary lymph glands and the salivary glands are swollen and mastication is almost impossible. Fever, prostration and signs of sepsis, as erythema, chills, and rapid pulse, may mark the severest cases. Acute cases last one to two weeks and chronic cases weeks or months.

Varieties.—(*a*) *Mercurial stomatitis* may occur in workers in the metals or from clinical administration. The author saw a hospital case of nephritis in which one dose of five grains of calomel caused necrosis of the entire tongue, which rotted off at its root (See INTOXICATIONS). Mercurial and other stomatitis may cause "erosions" of the enamel in the developing teeth of children; they become pitted, discolored and transversely furrowed. (*b*) *Riga's disease*, described by Riga (1881) in South Italy, is an affection in which there is a pearly induration which occurs only between the tip of the tongue and the frænum, appears with the first dentition, sometimes ulcerates, is occasionally epidemic, is often observed with the status lymphaticus and is invariably fatal. (*c*) In *nursing women*, ulcers, 3 to 4 mm. in diameter, may develop from the lymph follicles of the lip and cheek; they usually indicate malnutrition, sometimes are quite painful, and respond to general tonics and hygiene. (*d*) *Herpetic or pemphigoid stomatitis* is seen in neurotics (Jacobi's stomatitis chronica neurotica and is frequently recurrent. (*e*) *Bednar* described (aphthous) *whit plaques* on the posterior part of the hard palate near the alveolar borde of the upper jaw, which may ulcerate; they occur in the first three months of life, and are apparently due to sucking, are not syphilitic, and are ominous only in marantic children. (*f*) *Parrot's ulcers* occur in the new-born on the hard palate near the median line; they occur in marantic children and seldom heal.

Treatment.—Treatment includes that of catarrhal stomatitis. Potassium permanganate 1 to 1,000 relieves the *fœtor ex ore.* Necrotic areas may be touched with silver nitrate solution or equal parts of tincture of the chloride of iron and glycerine. Potassium chlorate gargles may be combined in children with gr. x to xx of the drug every day internally and twice the amount for adults, but it must be remembered that toxic effects may result. Opium and belladonna are used in severe cases for pain and salivation.

(C). APHTHOUS STOMATITIS.

Etiology.—It may occur alone or with fevers and other diseases, sporadically or epidemically. It is probably contagious but its nature is still undetermined; it is classed by some with impetigo, due to the staphylococcus, and by others with foot-and-mouth disease; when due to milk it ceases on withdrawal of the milk. Besides the general causes of stomatitis (*v. s.*), it occurs chiefly in children between ten and thirty months old.

Symptoms.—Symptoms begin with restlessness, slight fever, thirst, salivation and pain in the mouth. Round, yellow-white and slightly elevated areas as large as a pea appear on the surface and edges of the tongue and less often on the lip and buccal mucous membrane. The mucosa is slightly reddened about them. They cannot be scraped off and attempts to do so produce bleeding. Pathologically they are areas of superficial necrosis, into which exudation of fibrin and round cells occurs (stomatitis fibrinosa disseminata). Vesicles are described but seem to be no part of the process, though they may occur on the skin near the mouth. Occasionally there is tumefaction of the lymphatic and salivary glands. Their clinical evolution is rapid; they last one to two weeks, and recurrence is frequent. The *treatment* is that of stomatitis.

(D). PARASITIC STOMATITIS.

Etiology.—*Thrush, muguet, soor,* or *stomatomycosis oïdica* is due to the Oïdium albicans, closely allied to the saccharomyces, which consists of branching mycelia and ovoid granular and refracting spores. It occurs chiefly in very young children whose mouths have been improperly cared for, or who have stomatitis and reduced nutrition. It prevails especially among the poor and in institutions. Dirt are probably the most frequent mode of in develop in adults, as in weakly convalescents, or in tuberculous and diabetic subjects.

Symptoms.—A few thin, punctate, whitish spots develop on the tongue, cheek and soft palate; they become yellowish as the areas grow or fuse. The fungus is at first deposited on the epithelium, between the layers of which it proliferates and penetrates. The mouth is dry, or, with diffuse stomatitis, is moist from increased salivary flow. In the more severe cases the fungus may reach the pharynx, larynx, œsophagus, and even the stomach and intestine. Its deposit is generally limited to areas with flat epithelium, and epithelia of the cylindrical and ciliated order usually escape. It may, in exceptional cases, cause œsophageal occlusion, or bronchopneumonia; or by unusually deep penetration to the submucous vessels it may cause cerebral, renal and other embolism. The *outlook* is good except in marantic subjects.

Treatment.—*Prophylaxis* is most essential in regard to cleansing of the mouth, sterilization of bottles and nipples, and maintenance of the general health by fresh air and by early treatment of dyspeptic disorders. In some cases the stomatitis persists until a wet-nurse is substituted for the feeding with cow's milk. Cleansing the mouth with *borax*, boric acid, sodium bicarbonate and lime water is most effective. If unusual extension occurs, a teaspoonful of a resorcin solution (1 to 100 or 300 parts) may be given every two hours.

Stomatitis may in rare cases result from *other parasites*, as the sarcina or leptothrix, and from such bacteria as the pneumococcus, gonococcus, Friedländer's bacillus, etc.

(E). GANGRENOUS STOMATITIS.

Etiology.—"Gangrene of the cheek," *noma* or *cancrum oris*, is a rare affection. *Age:* of Brun's 415 cases, only 11 occurred in persons over fifteen years of age and only 6 in infants. The author recently saw a case in a man forty years old, which was fatal on the fifth day. *Sex:* more cases occur in girls than in boys. *Malnutrition* and neglect are causes of some cases, and others follow acute infections; 50 per cent. follows measles; typhoid, scarlatina, etc., are less frequent factors. A bacillus resembling that of diphtheria has been isolated (Bac. necroseos). Cladothrix threads similar to those in Vincent's angina may be seen.

Symptoms.—Symptoms usually begin with ulceration of the gums or buccal mucosa near the angle of the mouth. Rapid necrosis and gangrene follow with extensive brawny œdema of the face. The fœtor is intense and much tissue is frequently sloughed off. Perforation of the cheek, necrosis of the jaw and extension to the throat, orbit or ears are not uncommon. Seventy per cent. of cases die within a week; they show septic symptoms, such as high fever, weak heart, prostration, diarrhœa, colitis, and meningitis, or symptoms of lobular pneumonia becoming gangrenous. The other cases exhibit less general reaction and may leave only an extensive scar or fistula on the cheek. The mortality was 92 per cent. in Springer's collation.

Treatment.—Treatment is ineffectual. A full diet and free exhibition of alcohol are indicated as in sepsis, and disinfectants should be applied to the necrotic focus; local measures as excision, the live cautery and caustics are employed, usually without success, and sometimes with aggravation of the necrosis.

DISEASES OF THE TONGUE.

(A). ECZEMA.

In eczema the epithelium thickens and desquamates, leaving reddish, smooth and circular, serpiginous or geographical areas, which heal centrally and extend peripherally. It may cause local irritation or worry to the patient, who may think he has syphilis or cancer. Again, it is an accidental finding on the tongue or cheeks. Its cause is unknown, its course is quite chronic, and its treatment by silver nitrate is quite inadequate.

(B). LEUKOPLAKIA.

Leukoplakia (psoriasis, ichthyosis, keratosis) is an affection closely resembling the "geographical tongue," in connection with which it is often described. Irregularly-shaped, smooth, pale plaques, measuring one to two centimeters, develop on the tongue and less often on the lips, cheek or tonsils; they consist of thickened epithelium. Eighty per cent. of cases occur in men (98 per cent., Fournier); the use of tobacco and alcohol, and gout and gastric disease are probable factors; syphilis is not a cause and antisyphilitic remedies are directly injurious, though Fournier's experience with 324 cases convinced him that 80 per cent. came from syphilis and 20 per cent. from tobacco. Its symptoms are not marked, its course is very chronic and its treatment is nearly always without results. Ten per cent. solutions of sodium salicylate; chromic acid, 5 per cent.; strong silver nitrate applications, specially to fissured places; corrosive sublimate (1 to 500 parts) and the galvanocautery are recommended. Excision is necessary for papillomatous or epitheliomatous outgrowths, which are said to develop one-third to nearly one-half the cases.

(C). ACUTE GLOSSITIS.

Acute glossitis may be localized or diffuse, and usually ends in s
puration. It follows stings, burns, erosions or the entrance of pyoge
cocci through the lymph follicles. The tongue is swollen, sometim
so greatly that it protrudes beyond the lips, and is painful. Eati
and even speaking or oral breathing are impossible. Early scarifi
tion and later incision of purulent foci are indicated.

(D). GLOSSITIS DESICCANS.

Glossitis desiccans is a rare, chronic, intractable affection in wh
deep indentations of the margins of the tongue, and consequent lobu
tion occur. *Melanoglossia* (*nigrities linguæ*) results from proliferat
of the filiform papillæ at the base of the tongue, which appear like h
(*melanotrichia linguæ*, black-haired tongue); they are removed b
10 per cent. salicylic acid solution or by curetting. The *mucor ni*
may cause a black coating on the tongue.

DISEASES OF THE SALIVARY GLANDS.

(A). PTYALISM, SALIVATION, HYPERSECRETION, SIALORRHŒ

Etiology.—The chief causes are (*a*) disease of the mouth, stomatit
dentition; (*b*) toxic substances as jaborandi, tobacco, physostigm
digitalis, mercury, iodides and various metals; (*c*) nervous affectio
as trigeminal neuralgia, diseases of the pons and medulla, pare
dementia, hypochondriasis, and hydrophobia; a close distincti
should be drawn between oversecretion and lesions in which, li
bulbar paralysis, the saliva cannot be swallowed; (*d*) reflex salivatio
which may also occur from diseases of the tympanum through t
chorda tympani, from gastro-intestinal disease (gastric ulcer, panc
atic lesions, etc.) by way of the vagus and sympathetic nerves, and fro
genital conditions, as pregnancy.

Symptoms.—The physiological salivary flow measures seven ounc
to three pints (200 to 1500 c. c.), which pathologically may amount
quarts; a flow of over twelve quarts has been recorded. The saliva
glands are tense, swollen, and even hard. The reaction of the sali
may become neutral or acid; it is chiefly thin and fluid, but may co
tain some ropy mucus or scattered pus-cells; the ptyalin disappea
together with the potassium sulphocyanide. The saliva may trick

into the larynx during sleep, and thus cause violent coughing; it may flow into the stomach, and produce vomiting, of an alkaline or neutral vomitus, especially in the morning; it may stream from the lips, and cause the adjacent skin to become erythematous.

Prognosis and Treatment.—The prognosis depends wholly on the causal affections, whose operation may be transient, intermittent or permanent. Treatment is likewise causal. Any toxic cause should be removed. In nervous and reflex types, potassium bromide is often valuable and the iodides are sometimes efficacious in pregnancy. The more severe cases necessitate opium and occasional doses of belladonna.

(B). XEROSTOMIA.

Hyposecretion or arrested secretion was first described by Jonathan Hutchinson. Besides its occurrence, as a result of atropine and allied remedies, it occurs in diabetes insipidus and mellitus, in some cases of contracted kidney, and finally, as an independent neurosis or a neurotic symptom. Women (80 per cent.) are affected more often than men. The tongue and mouth are dry, red and glistening, which may render speaking, chewing and swallowing most difficult. The tongue may cleave to the roof of the mouth. The digestion of carbohydrates is impaired. *Treatment* consists in painting the mouth with iodine, potassium iodide and glycerine (1, 10, 100 parts respectively), the administration of pilocarpine hydrochlorate $\frac{1}{12}$ to $\frac{1}{8}$ of a grain and galvanic applications to the parotid.

(C). PAROTITIS.

Besides acute specific parotitis (mumps), infection may reach the parotid or other salivary glands by way of the blood stream or by infection entering their ducts. *Acute parotitis* may be simple but is oftener suppurative, the staphylococcus being the most common pyogenic organism. (*a*) Any of the acute infections may be complicated by parotitis, of which typhoid is the most common; parotitis may develop in measles, scarlatina, pneumonia, septicopyæmia, and syphilis. (*b*) Diseases or trauma of the abdominal or pelvic structures are sometimes complicated by acute parotitis, to which Paget drew special attention; they include diseases of or operations on the alimentary and genito-urinary tracts, peritoneal disease, pancreatitis, trauma to the abdominal viscera, which may result from pessaries or from external violence, injury of the abdominal wall, etc.; many of these cases are clearly attenuated sepsis. Wagner, in 1904 was able to collect 61 cases of postoperative parotitis; 31 were operations upon the genitalia; he believes the cause is not hæmatogenous infection, but an infection ascending along the duct from the mouth. (*c*) A rare form of acute parotitis in facial neuritis is described by Gowers.

Chronic parotitis sometimes follows epidemic or symptomatic p titis; it may occur in poisoning by mercury or lead, and in syphil chronic nephritis. The parotid is usually painless, though sometimes both painful and tender. Mikulicz has described a "ch symmetrical hypertrophy of the salivary and lachrymal glands" w is often of long duration.

Treatment.—In acute parotitis, local derivants as leeches, the application of cold and the later use of hot fomentations, and fi of surgical incision, if there is pus formation, are indicated. Ch forms respond slowly to treatment; iodine or mercurial ointn should be employed. Iodide is useful in Milkulicz's disease.

The parotid gland and its duct may become inflated with a persons having such occupations as blowing wind-instruments glass-blowing. In rare cases gas forms in the gland or its duct.

(D). ANGINA LUDOVICI.

Ludwig's angina is generally due to a streptococcic infection b ning in the submaxillary gland, and extending to the floor of the m and to the cervical cellular tissue. In some cases no promoting c is found, or trauma may be its immediate antecedent, but most are secondary to typhoid, diphtheria, scarlatina and kindred a infections. It is the analogue, though differing in location, of a phlegmon of the pharynx and larynx (submucous suppurative inf mation or œdema). The streptococcic inflammation spreads rap causing fever, redness, pain and swelling under the tongue, cell and sometimes gangrene in the neck (*cynanche gangræneuse*). M cation and deglutition may be impossible and glottis œdema is infrequent. The parotids are sometimes invaded. Resolution is exceptional and external pointing of pus is not likely to occur, whe without early surgical intervention, septic and pyæmic complicat or laryngeal and pharyngeal œdema or phlegmon very freque develop.

(E). SIALODOCHITIS FIBRINOSA.

Inflammation of the salivary ducts with formation of memb (Kussmaul) results from infection ascending from the oral cavity is characterized by fever and tenderness, pain and tumefaction of salivary gland, due to retained secretion and infection. The du kept patent by pressure or sounding.

(F). SIALOLITHIASIS.

Stones of calcium phosphate or carbonate occur more frequentl Wharton's duct than in the other salivary ducts. Their usual cau tagnation of the salivary secretion by foreign bodies, bacterial or ungus invasion or mechanical stricture following ulceration. Roberg 1904) found stones in Wharton's duct fifty times, in the submaxillary land twenty-eight times, in Stenson's duct six times and in the parotid land twice.

DISEASES OF THE PHARYNX.

(A). ACUTE PHARYNGITIS.

Etiology.—Acute, superficial, simple angina may be caused by (*a*) rheumatism, cold or exposure; (*b*) infections, either acute (measles, scarlatina, variola) or chronic (malaria or syphilis); (*c*) gout, dyspepsia, or smoking; (*d*) in many cases the cause is obscure; though probably always mycotic, no one germ is especially found. It is most common in youth.

Symptoms.—(*a*) *Local:*—The throat is red, glazed and streaked with mucopus which is easily wiped away. The soft palate and uvula are sometimes swollen; superficial erosions, small vesicles on the palate and swollen mucous follicles are occasionally seen. In some cases the local findings, especially when hidden in the nasopharynx, are very slight in proportion to the local pain and general symptoms. There is a scratching, tickling sensation in the throat which may radiate into the posterior nares or into the ear. The patient hawks, but raises little, unless there is coincident rhinitis or laryngitis. Swallowing is rather painful, slight deafness is quite common, the speech is sometimes slightly nasal, and the angular lymph nodes are painful and tender. (*b*) *Constitutional symptoms* are usually slight. At the onset there are chilliness, moderate fever, aching in the muscles and in some individuals extreme cutaneous hyperæsthesia, which, on superficial examination, may be falsely attributed to changes in the deeper parts of the body. With or without coincident tonsillitis, there may be high fever, and in labile individuals, marked nervous intoxication. Herpes facialis occasionally develops. Complications, as acute endocarditis, or nephritis, are very uncommon. In a few days the process is complete and convalescence is rapid.

Treatment.—(*a*) Local:—painting the entire pharynx with a 20 per cent. silver nitrate solution usually affords not only local but general relief. (*b*) General:—Dover's powder and aspirin, each ten grains, may be exhibited for unusual pain, aconite for high fever, calomel, followed by salines, for elimination, and a 1 per cent. solution of carbolic acid for the irritated throat.

The *pneumococcic sore throat* may be suppurative, pseudodiphtheritic, follicular (like tonsillitis), erythematous or herpetiform; its onset

is severe, like that of pneumonia, it produces a thicker, denser a
more adherent membrane than does the streptococcus and the pr
nosis is generally poor.

(B). CHRONIC PHARYNGITIS.

Etiology.—The causes are: (*a*) repeated acute pharyngitis;
excessive smoking or use of alcohol; (*c*) abuse or over-use of t
voice, as in clergymen, vendors, etc; (*d*) extension from or associati
with chronic nasopharyngitis or laryngitis; (*e*) various conditions
the blood, as in chronic nephritis, syphilis, arthritism, etc. Th
causal factors are most common in adolescence and middle life.

Symptoms.—The symptoms are local. The mucosa of the nas
pharynx is generally red and lined with turgid venules; the pillars a
uvula are relaxed, and the drooping uvula may occasion tickling of t
tongue or even of the epiglottis, thus causing chronic coughing. T
sides of the pharynx may show isolated whitish areas of epitheli
thickening or more often small, round, reddish, hypertrophied lym
follicles (*pharyngitis granulosa*), sometimes in linear distributio
The pharynx is dry and burning, exciting hawking efforts which a
not only unproductive but in themselves are irritating to the phary
and may cause morning vomiting. The mucous glands are at tim
hyperplastic. Slight bleeding occasionally causes unnecessary wor
Middle ear involvement is common, through the Eustachian tub
Pharyngitis sicca is an atrophic type, analogous to rhinitis sic
(atrophica) and marked by a pallid glazing, chiefly of the posteri
pharyngeal wall, and by formation of crusts.

Treatment.—(*a*) The *causal factors*, as improper straining
tension of the voice in speaking, excessive alcoholism or smoking, et
should be corrected respectively, by rest and avoiding irritant foods
stimulants. Alkaline laxatives are frequently beneficial. (*b*) *Galvan*
puncture of the turgid veins or hyperplastic follicles is indicate
marked improvement is probably more frequent than aggravation
the condition by over-zealous treatment. (*c*) *Local medical treatme*
is less efficacious, as 2 to 20 per cent. solution of tannin in glycerin
equal parts of tr. ferri chloridi and chlorate of potash, 1 to 5 p
cent. solution of chloride of zinc, or 2 to 20 per cent. nitrate of silv
solution.

℞

Acidi carbolici	gr. xx.
Acidi tannici	ʒss.
Aluminis	ʒj.
Glycerini	℥ij.
Aquæ rosæ	q.s. ad ℥v.

M. et ft. gargarismus.
S.—As gargle, properly diluted.

(*d*) For dry *atrophic* pharyngitis, iodine, potassium iodide and gl
cerine (1:10:100 parts, respectively), massaged thoroughly into th
pharynx, is the best known measure:

℞

Iodi	gr. iij.
Potassii iodidi	ʒss.
Glycerini	℥j.

M. et S.—Locally.

(C). PHLEGMONOUS PHARYNGITIS.

Angina phlegmonosa, especially described by Senator, is not superficial as is simple pharyngitis, but invades the submucosa. It is usually primary but may be metastatic from distant suppuration. The throat is painful, swollen and œdematous, the neck enlarges and suppuration occurs with high fever, septic symptoms and difficulty in swallowing and breathing, especially when the subject lies down. The inflammation may be hemorrhagic or necrotic. Phlegmonous angina is the analogue of suppurative submucous laryngitis and of angina Ludovici. Without surgical intervention or early spontaneous rupture into the throat, death occurs from septicopyæmia, mediastinitis or from œdema of the glottis.

(D). RETROPHARYNGEAL ABSCESS.

Various forms are described. (*a*) The *primary* form occurs in children under two years of age; the early symptoms are dysphagia, a nasal voice and retropharyngeal suppuration, either as a visible protrusion on the posterior pharyngeal wall or as a palpable fluctuation there; in a week or two dyspnœa, cyanosis and inspiratory retraction of the chest develop. It is a primary lymphadenitis. (*b*) Secondary types are less common in (*i*) caries of the cervical spine, (*ii*) diphtheria, erysipelas, scarlatina and (*iii*) suppurative inflammation in the retropharyngeal lymph glands. The secondary type is caused by infection travelling along the lymph vessels, or rarely by metastasis. Unless surgical measures are instituted or spontaneous rupture occurs early, death occurs from asphyxia, mediastinitis, glottis stenosis, inhalation pneumonia or sepsis. After incision the head should be promptly thrown well forward to obviate occlusion of the air passages.

(E). PHARYNGEAL ULCERATION.

Syphilitic and tuberculous ulceration have been considered. Follicular ulcers are superficial and small and, like all ulcers in the mucous membrane only, heal without a cicatrix. Ulceration also occurs in diphtheria and the diphtheroid group, due chiefly to the streptococcus. Malignancy too, causes ulceration. Cancer and tuberculosis are usually diagnosticated with ease, and syphilis is separated *ex juvantibus* by the use of mercury and iodides in doubtful cases.

Vincent's angina is discussed under DIAGNOSIS OF DIPHTHERIA; according to Vincent, it constitutes two per cent. of all anginas including diphtheria. It occurs especially in children between six and ten years of age and in medical students and servants in anatomical laboratories, between the years of eighteen and thirty. Tobacco, scurvy, anæmia, tuberculosis, syphilis, scarlatina, measles and the eruption o the wisdom teeth are predisposing factors. The two causative organisms were first described by Rauchfus (1893); (*a*) the *fusiform bacillu* has pointed, sometimes rounded ends, is sometimes bent, measures six t twelve μ in length and may be flagellated; (*b*) the *spirillum* or spir ochæte is long, delicate, twisted and always occurs in severe form They stain with carbol-fuchsin but not by Gram's method.

Symptoms.—(1) In the first period, there is *congestion* and œdem (2) In the second stage, a grayish yellow *membrane* forms which thick, friable and cheesy and is usually removable. (3) Under t membrane, an *ulcer* develops; it is usually single, round or oval a has an irregular edge, an uneven granulating floor and seldom a extension laterally.

The general symptoms are lassitude, indigestion, vomiting or e staxis; fever is uncommon and complications rare (albuminuria, e teritis or noma). Syphilis and diphtheria require differentiation. T affection heals in one-half to seven weeks, under applications of io locally, pencilling with silver nitrate, gargles of hydrogen peroxide chlorate of potash internally.

DISEASES OF THE TONSILS.

(A). ACUTE FOLLICULAR OR LACUNAR TONSILLITIS.

Definition.—An acute, largely parenchymatous, inflammation of th tonsils, due to local mycotic invasion and marked usually by decide constitutional reaction.

Etiology.—(*a*) *Bacteriologically*, the streptococcus is the most fr quent cause, though the staphylococcus and Fraenkel's diplococcus a also encountered; it is claimed that somewhat attenuated diphthe bacilli are found, particularly at the time of diphtheria epidemics, in such cases it is more correct to consider it as a sub-type of diphthe (*q. v.*). (*b*) The still unknown virus of *rheumatism* is thought to g access to the blood by way of the tonsils or pharynx; affections co monly regarded as associated with rheumatism, as erythema nodosu chorea, and some forms of endocarditis, may follow acute tonsill (Packard). Tonsillitis may begin the rheumatic cycle, as descri under rheumatism in children, or it may appear later in the rh matic process, with endocarditis, pericarditis, arthritis, and chor Endemics and the spread of the disease through a household or inst tion strengthen the theory that tonsillitis is an acute contagion. (*c*) *Cold* and *dampness* are directly predisposing causes; these conditions obtain in the United States largely in the spring, in some other countries largely in the fall months. (*d*) Tonsillitis occurs chiefly in youth and adolescence; infants are seldom affected. (*e*) *Individual predisposition* depends on some unknown factor and tonsillitis itself is of etiological importance, for one attack may induce recurrence. (*f*) *Poor hygiene* and possibly sexual activity, especially in young married people (Shepard), may be conducive to tonsillitis.

Symptoms.—(*a*) The *onset* occurs with chilliness or rigor, severe *pains in the back* and limbs, *fever*, rising within a day to 103 or even to 106 degrees in children or susceptible adults, *pain in the throat*, particularly on swallowing, and tenderness of the glands at the angle of the jaw. (*b*) *Local findings:*—the *tonsils are swollen* and soon show on the deeply injected surface three to ten yellowish-white or creamy points, corresponding to the crypts or lacunæ; the crypts contain bacteria, epithelium, leukocytes and sometimes cholestearin or fatty needles and are essentially small abscesses. Pathologically the tonsil is the seat of serocellular infiltration. Occasionally there are small superficial areas of necrosis, slight erosions or even formation of membrane. The voice may acquire a somewhat nasal twang, the pain increases for a few days and the angular glands remain both painful and tender, whence the head is often held rigidly forward and the jaws are opened only with difficulty. Pain in the ear is not uncommon. (*c*) The chief *constitutional symptoms* have been considered under (*a*). Anorexia, heavily coated tongue, feverish breath, hyperæsthetic skin, marked depression, accelerated pulse and respiration, are common. In some cases the systemic symptoms are disproportionately severe and children, as well as some adults, may exhibit delirium; in two cases seen by the writer, in adults, retraction of the neck with headache, delirium and vomiting was thought indicative of meningitis. Herpes facialis is quite frequent.

Complications.—Complications are not common. Acute pericarditis, endocarditis or nephritis may develop. Functional heart murmurs, febrile albuminuria and erythema (from the toxins or from medication) are not very infrequent. As a rule, however, convalescence is complete within a week, though marked prostration and considerable residual swelling remain for some time. Repeated attacks may cause nephritis or endocarditis.

Diagnosis.—The typical case cannot be confused. (*a*) *Diphtheria* produces a membrane, more whitish in color, which has an almost pathognomonic tendency to spread to the uvula, soft palate or pharynx; it leaves bleeding raw points when it is stripped off; it recurs after removal; it sometimes extends to the larynx; it is followed not infrequently by paralysis, which almost never results from tonsillitis; it responds with more or less promptness to serotherapy, and shows in practically all cases the Klebs-Loeffler bacillus. (*b*) *Tonsillitis necrotica* leaves an ulcer; its bacteriology is disputed, some cases showing the fusiform bacillus and spirilla of Vincent (see ULCERATIVE PHARYNGITIS and DIPHTHERIA), and others are alleged to be due to the Bacillus diphtheriæ; in some cases there is much fever; in others none; the general reaction is severe. (c) The possibility of incipient *scarlatina* should always be kept in mind.

Treatment.—1. Prevention.—The subject should be hardened by cool baths or sponges, and tonsillotomy should be performed. Douching of the nose and gargling with Dobell's solution should be part of the daily toilet.

2. Local Measures.—Though many local remedies are recommended, the best in the author's experience is *silver nitrate*; after cocainizing the throat, a small probe is bent at right angles near its end and introduced into each swollen crypt in order to puncture it and secure free drainage; then Dobell's solution should be applied, undiluted, on small bits of cotton in each crypt; this is followed by 20 per cent. silver nitrate solution; the results are often immediate, sometimes aborting the attack. Squeezing out the crypts with a spatula is extremely painful but relieves the patient when the physician has no probe at hand. Hot water or hot fomentations may be applied to the neck.

3. Constitutional Treatment.—In a relatively small number of cases sodium salicylate, given as in rheumatism, affords relief. Hydrargyri biniodide gr. $\frac{1}{500}$ given every fifteen minutes for ten doses often aborts the process. Guaiac, though generally used, has produced unsatisfactory results in personal experience; the dose is tr. guaiaci ammoniat. ♏ x to xl every two hours. Aspirin, and Dover's powder āā gr. x, relieve pain in the throat, body, and limbs. Aconite and belladonna are often beneficial in children with high fever, and rather less so in adults; they are given as in coryza or influenza.

(B). SUPPURATIVE TONSILLITIS.

Etiology.—The etiology is almost identical with that of the follicular type. Suppurative tonsillitis is most common in adolescence, and individual disposition for unknown reasons is an important factor.

Symptoms.—The early symptoms, local and general, resemble those of the follicular type but are often more severe from the onset; one, or less often both, tonsils are greatly swollen, tense, œdematous, deep red and may show distended lacunæ. The pain often radiates to the ears. The swollen tonsils may come in contact, or if but one tonsil is swollen it may reach over to the sound side. There is often coincident stomatitis and a free salivary flow. The palate and uvula are frequently œdematous and covered with muco-pus. Swallowing causes great pain in the throat, the jaw cannot be depressed because of the swollen, painful angular glands, and the speech is nasal. The toxæmic prostration is often profound, the fever ranges between 103 and 105 degrees and the pulse between 100 and 140.

Incision in the first two or three days evacuates no pus and only blood and serum escape. In a few days fluctuation is felt in the tonsil, or anterior to and above the tonsil (peritonsillar abscess). Introduction of a probe into the early and often unsuccessful incision is followed by escape of pus and by immediate relief. The abscess may break spontaneously, usually forward and rarely toward the throat, when, in rare cases, especially when rupture occurs during sleep, suffocation or aspiration pneumonia may follow. In equally rare instances ulceration may reach the carotid with sudden and fatal hemorrhage.

Treatment.—(*a*) For prevention *tonsillectomy* by the knife or galvanic snare is indicated after an individual tendency is manifested. (*b*) In *local treatment* an ice-bag placed over the neck is beneficial. Local applications to the tonsils are at the best uncertain remedies. It is good practice, early in the course, to introduce a knife, covered with adhesive plaster to within half an inch of its point, above and somewhat inside the tonsil; blood and serum escape, tension, œdema and pain are relieved and the later abscess more readily points toward the cut; the wound is carefully opened with a blunt probe every day; early incision is preferable to scarification. (*c*) *Salicylates* apparently help and possibly hasten maturation. *Opium* and *coal tar products* relieve the pain to some extent. (*d*) In exceptional cases, intubation, tracheotomy or immediate excision of the tonsil are indicated, because of great swelling or collateral œdema. (*e*) The diet is necessarily liquid. (*f*) In *convalescence*, iron and strychnine overcome the resulting prostration and anæmia.

(C). CHRONIC TONSILLITIS.

Synonyms.—Chronic inflammation; tonsillar hypertrophy; hyperplasia of the pharyngeal or lingual tonsils; adenoids; aprosexia.

Etiology.—(*a*) Repeated *acute attacks* of tonsillitis cause chronic hypertrophy in some cases, and in others the tonsils gradually indurate and shrink. (*b*) Waldeyer's "Schlundring" of *lymphadenoid* tissue consists of the *two tonsils*, the adenoid tissue in the nasopharyngeal vault (Köllicker's pharyngeal tonsil, or "*adenoids*" in the common acceptation), and the so-called *lingual tonsil*. Variously combined hypertrophies of these structures may be noted, occasionally as congenital growths, but most frequently in young children, and sometimes in adults. They generally begin during the third or fourth year and may develop until puberty. Adenoids are found in 1 per cent. of children (Meyer), though other statistics give 3 per cent. or even higher figures. Rickets, scrofula, the lymphatic constitution and various acute infections involving the throat, as diphtheria, are cited as predisposing conditions.

Symptoms.—**1. Local Changes.**—(*a*) The *enlarged tonsil* may contain in its crypts cheesy plugs, which occasion an extreme *fœtor ex ore*. The plugs may be expressed; calculi may result from a deposit of lime salts. The pharyngeal mucus is increased, is sometimes tinged with blood, and is expectorated with difficulty. (*b*) *Adenoids* in the nasopharyngeal vault occur alone or with tonsillar hyperplasia. They are most often papillomatous, less often sessile; they are reddish, very vascular and range up to the size of a bean. (*c*) Hypertrophy of the "*lingual tonsil*" often disturbs deglutition. Enlarged tonsils and adenoids produce most of the following changes by stenosing the posterior nares.

2. Sequences.—(*a*) *Oral breathing* is the earliest symptom, and is first noticed during the night, when sleep is disturbed by night terrors (*pavor nocturnus*) and by disordered and sometimes irregular breathing, for a time stertorous, and then interrupted. Lying down may occasion asthmatic seizures or Balne's paroxysmal cough. (*b*) After some time *changes in the mouth* are noted. The *palatal vault* is *high* from atmospheric pressure in the mouth, and the transverse measurement between the upper teeth is proportionately diminished; there is less room for the teeth to erupt. (*c*) In the nose, the *anterior nares are small* and retracted, and the voice is nasal (*rhinolalia claustra*); the consonants *n* and *m* are pronounced with difficulty. Bloch associated oral breathing with stuttering. The senses of smell and taste are obtunded. Hearing is impaired by pressure of adenoids on the Eustachian tubes by inflammation in the tubes or by retraction of the tympana from low atmospheric pressure in the nasopharynx. (*d*) The *facies* is characteristic; it is *vacuous* and *apathetic*, the nose is pinched, the mouth open and the lips swollen. The so-called "adenoid habitus" may be observed without adenoids, as a stigma of degeneracy. (*e*) The *mental* [illegible] is altered in chronic cases; the cerebral reaction is slow, the memory tardy, the disposition sullen and the power of concentration diminished; this latter condition was named aprosexia by Guye. Woodward refers the physical changes to some toxin (cytotoxin) elaborated in the hyperplastic lymphoid tissue. (*f*) There are other *nervous symptoms*. Headache is common. Adenoids cause some cases of habit-spasm. Night terrors have been mentioned. Enuresis nocturna results from carbon dioxide operating on the brain during sleep (Schech). (*g*) *Development* is sometimes *retarded*. (*h*) Changes in the *contour of the thorax* were noted by Dupuytren (1828), J. M. Warren (1839) and Lambron (1861). The pigeon- or *chicken-breast* is the most common deformity. The sternum juts forward, from which the ribs slant sharply backward; at the level of the diaphragm there is a circular depression, and inspiratory retraction is caused by the contraction of the diaphragm. The *funnel breast*, marked by a deep depression of the lower part of the sternum, is less commonly due to adenoids, and like the chicken-breast also results from other causes. The "emphysema chest" may result from adenoids. (*i*) Scheir finds that the cervical lymph glands are also enlarged and that the blood evidences a decrease in hæmoglobin, two- to three-fold increase in the white cells, and a distinct lymphocytosis. (PLATE XI).

Diagnosis.—The oral breathing, the adenoid facies and mental disturbance, palpation of the adenoids in young children and their

PLATE XI.

FIG. 1.

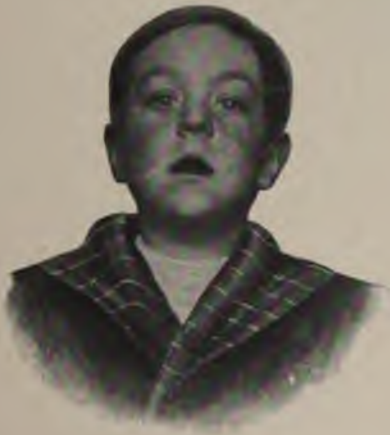
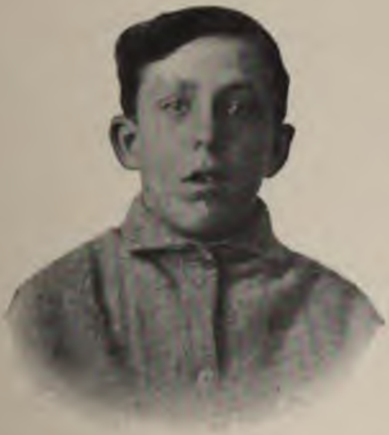


FIG. 2.



s of the "Adenoid" Facies. (Posey and Wright.)

rhinoscopic detection in older ones, and the mere inspection of hyperplastic tonsils determine the diagnosis. In the *prognosis* of untreated cases, the possibility of deafness, mental deficiency, retarded growth, asthma, and emphysema should be borne in mind. Colds, acute recurrent tonsillitis, diphtheria and probably other infections are more likely both to develop and to produce disproportionate symptoms. In some few cases the hyperplastic tissues may indurate and atrophy, but this spontaneous issue should have no essential influence in their management.

Treatment.—Palliative treatment by astringents is useless. The general health should be maintained by out-door life, an ample diet, hydrotherapy, and by cod-liver oil, iron and other measures which are employed in scrofula and tuberculosis. Hypertrophied tonsils should be removed at once by the *knife* or in several sittings by the *galvanic cautery*, for details of which the reader is referred to special treatises. Adenoids large enough to cause symptoms should always be removed early under anæsthesia, by the finger or curette; ether is far less dangerous than chloroform, which frequently causes death in any disease of the lymphadenoid tissues (the *status thymicus* or *lymphoid constitution*). The sequences above described usually subside soon after operation, though oral breathing is a habit somewhat hard to break. At night a bandage should be used to keep the mouth closed.

DISEASES OF THE ŒSOPHAGUS.

(A). INFLAMMATION, NECROSIS, ULCERATION.

1. **Œsophagitis.**—This is an uncommon finding at autopsy. Its *causes* are (*a*) mechanical, as from foreign bodies, strictures or dilatation of the œsophagus; (*b*) chemical, as from alcohol, tobacco, corrosive poisons or acid vomitus; (*c*) thermal; (*d*) hypostatic, in cardiac or pulmonary affections; (*e*) rarely extension from pharyngeal or laryngeal inflammation; (*f*) acute infections, as measles, smallpox, scarlatina, and diphtheria. Œsophagitis is almost physiological in the new-born after the ingestion of the first meals of life. Its pathology in general is that of pharyngeal or nasal inflammation; it is marked by local or diffuse, acute or chronic redness, swelling and hypersecretion. Special *pathological forms* are follicular catarrh, sometimes resulting in follicular ulcers or retention cysts in the submucosa; exfoliative œsophagitis (o. dessicans superficialis); the fibrinous form, observed in various acute infections, in uræmia and rarely in hysteria; genuine diphtheria; the pustular eruption of smallpox; the toxic form resulting from caustics or acids; the very rare phlegmonous or purulent

form (only 12 cases are on record) which occurs as a single bulging submucous abscess or as diffuse submucous suppuration and often perforates in many places into the œsophagus which then presents a sieve-like aspect; forms due to the oïdium albicans, or ray fungus, of which primary cases are reported. In chronic cases the epithelium may thicken (leukoplakia) or hypertrophy (in papillomatous forms).

Symptoms.—Symptoms are absent in slight grades of inflammation. In marked cases there is *pain*, which is sometimes continuous and substernal, and at other times is elicted by speaking, movement or pressure on, or movement of, the spine. *Dysphagia* is constant in marked inflammations. Mucus, blood, pus or eschars, depending on the nature and cause of the inflammation, are brought up by gagging efforts. Fever is common in the more severe types, in which the local symptoms may be wholly subordinate to the causal disease (diphtheri smallpox, etc.). In inflammation due to acids or alkalies, the history local symptoms and eschars in the mouth and pharynx are distinctiv

Treatment.—Treatment is usually palliative at the best, depending on the cause. Narcotics are given for pain, demulcents as aca and ice, ice-cream and a liquid diet for the local irritation, and if the cause œsophageal irritation nutrient enemata are given (*v.* GASTRIC ULCER).

2. Necrosis.—This may be due to intense corrosive poisoning; to pressure from without, as from struma, aneurysm, foreign bodies or sounds which are left in the œsophagus to dilate strictures; or to decubitus or "bed sores of the gullet," first described by Dittrich (1850) in connection with laryngeal perichondritis, due to pressure of the larynx upon the gullet and promoted by fever, inanition and infections. *Œsophagomalacia* is softening of the part by regurgitation into it of the gastric acid; it occurs chiefly after death, but some few cases appear to develop during the death agony.

3. Ulceration.—Aside from cancer (*v. i.*), follicular ulceration, caustic ulcers, syphilis and tuberculosis, there is the peptic or round ulcer. *Peptic ulcer* was first described by Cruveilhier, who correctly identified it with round ulcer of the stomach. Twenty-three cases are recorded in the literature. It appears in four main conditions (*a*) by extension from the round ulcer of the cardiac orifice, (*b*) in ulcers at the pylorus or duodenum, which produce gastric dilatation, (*c*) in multiple hemorrhagic infiltrations of the œsophagus, stomach and duodenum and (*d*) in alcoholism, arteriosclerosis and other as yet imperfectly investigated conditions. Its chief symptom is *dysphagia*; its pathology and complications, as hemorrhage, perforation and stenosis, as well as its treatment, are practically identical with those of gastric ulcer, with which it is often associated.

(B). STENOSIS OR STRICTURE OF THE ŒSOPHAGUS.

Stenosis is the most important and frequent of œsophageal diseases. In this connection von Mikulicz's conclusion is important, *viz.* th the œsophagus is a closed tube only in its cervical segment and that its cardiac orifice is not physiologically closed.

Etiology.—(*a*) *Interstitial* factors, *i. e.*, in the wall of the œsophagus, are the most important; *cancer* (*v. i.*) causes 90 per cent. of all stenoses (v. Leyden, Kraus, Rosenbach); stenosis from *ulceration* by caustics and acids comes next; then comes stenosis by diverticula or pouches (*v. i.*) which, bulging out of and beside the œsophagus, exert upon it a lateral compression; other rarer ulcerative stenoses result from healing gummata, tuberculous lesions, smallpox and other suppurative affections, and from peptic and diphtheritic ulcers; muscular spasm causes a few cases; congenital stenosis or atresia is very rare (15 cases recorded) and occurs chiefly as a *cul de sac*, incomplete development or fistulous communications with the trachea or bronchi. (*b*) *Extra-œsophageal* factors, compressing the tube from without, are next in importance and include *aortic aneurysm* or abnormal branches from the aorta; vertebral lesions, (tumor, cold abscess, exostosis, great deformation); anthracotic lymphadenitis and malignant lymphoma; thyroid or thymic inflammation or hypertrophy; mediastinitis callosa, pericarditis effusiva, pulmonary neoplasms, and luxation of the hyoid bone or clavicle. (*c*) *Intra-œsophageal* factors comprise the fewest causal agents, as foreign bodies, pedunculated polyps which usually form from the lower pharynx, and Oïdium albicans.

Apart from the special *pathology* of these causes, stenosis occur chiefly (*a*) at the lower third of the œsophagus, (*b*) near the level of the bifurcation of the trachea, or (*c*) high up in the œsophagus. The stenosis is more often single than multiple and more often annular than longitudinal; it varies from slight stenosis to complete atresia, in which the tube may be seen as an impervious cord. Above the stenosis the tube is dilated and its musculature is usually vicariously hypertrophied in order to force food through the narrowed point.

Symptoms.—Symptoms vary with the cause and its extent; thus mechanical *dysphagia* may be quite secondary to aortic aneurysm and may vary from time to time with the pressure exerted upon it; in the cicatrizing ulcers dysphagia is the only symptom. When stenosis is advanced, the food and later the fluids taken, *regurgitate* after a short time, if the stenosis is low, or at once if the stenosis lies just below the pharynx; as a rule, the subjective localization described by the patient is unreliable. The *regurgitated food is neutral*, macerated, fermented, and often contains round cells, mucus, or blood, but never HCl nor pepsin. Œsophageal *pain* is common, but its occurrence depends on the causal lesion. *Singultus* is rather frequent when the lesion lies near the diaphragm. Dyspnœa may result from pressure upon the air passages by the dilated portion of the œsophagus above the stricture.

Physical Examination and Diagnosis.—The chest should be examined for aneurysm, retrosternal struma, and lung tumors, and the finger should be introduced as far as possible down the throat, to exclude the possibility of lesions situated high up in the œsophagus or larynx. Illumination of the œsophagus (*œsophagoscopy*) may give excellent results if done by an expert, but its general application is very limited. Local signs are elicited chiefly in three ways—by sounding, by auscultation and by the x-rays.

1. **Sounding.**—A soft sound is far less dangerous than a hard one, at least for the first examination and does the least damage in cases of acute œsophagitis, aneurysmal compression, and ulcerating cancer. It is not sufficient to pass one stricture, as there may be others lower down, whence the sound should be passed into the stomach. The length of the œsophagus is 10 inches or 25 cm. (or 15 per cent. of the patient's height); from the upper incisor teeth to the œsophagus 6 inches or 15 cm., from the teeth to the cardia 16 inches or 40 cm. Recently it is maintained that the last measurement should be 19.7 inches, or 50 cm. The length of the cervical portion is 2 inches or 5 cm., of the thoracic 6¾ inches or 17 cm., and of the abdominal part 1¼ inches or 3 cm. From the teeth to the level of the tracheal bifurcation is 9 inches or 23 cm.

2. **Auscultation.**—Deglutition is marked, physiologically, by two murmurs, which can be heard with the stethoscope six to eight seconds after the beginning of the act, first a short murmur and then a longer one. In cases of marked stenosis the swallowing sound is heard at a point just above the lesion; this is a valuable help when aneurysm is suspected and sounding is therefore feared. In the cervical part of the œsophagus the noise results chiefly from the movements of the pharynx; in the thoracic segment the stenotic murmur is heard to the left of the spine from the first to the ninth dorsal vertebra. Stenosis near the tracheal bifurcation often gives a delayed first murmur, and a gurgling, spurting character in both murmurs; in the lowest segment the murmurs are heard just above the cardia, at the ninth or tenth dorsal vertebra.

3. **X-rays.**—The x-rays may show the outline of the dilatation above the stricture (*v. i.* DILATATION OF ŒSOPHAGUS).

Prognosis.—The prognosis depends on the cause. Cancerous stricture may ulcerate, thus relieving the local symptoms, though the cachexia increases. Strictures resulting from caustics often improve. In cases where the cause cannot be relieved, the same symptoms result as in œsophageal cancer (*q. v.*). Perforation into the lungs, pleura, or air passages, or into the mediastinum or vessels, may follow. Aspiration pneumonia, which is often gangrenous, is not infrequent.

Treatment.—The remediable etiological factors are few, except that iodides may be given in syphilis or aneurysm. Foreign bodies may sometimes be removed by a long forceps, though of course with the direction of the œsophagoscope. In cancerous and aneurysmal strictures, probing and dilatation are dangerous. Dilatation by sounds is attended by the risk of perforating the œsophagus. Sounds should be introduced beside the index finger, and the patient's head should be thrown slightly backward; severe gagging may be overcome by painting the pharynx with cocaine, though it is often as much psychical as œsophageal, and assurance is a better preventive than drugs. The subject should first swallow some olive oil to facilitate passage of the sound. In passing the sound it should be held like a pen and pushed with exceeding gentleness. Trousseau's tapering ivory olive points are excellent, but care is necessary, and successively larger sizes should be used. Sounds may be left in the œsophagus for some minutes or an hour, or even permanently, but pressure necrosis may then result. It is at times necessary to introduce filiform bougies, such as are used in passing very narrow urethral strictures. Silver balls (2 to 7 mm. in diameter) with threads attached, may be swallowed; they should be carefully pulled out after remaining in over night. When the obstruction is high, external rather than internal œsophagotomy is advisable, though œsophageal surgery is still in its infancy. As first practiced by von Bergmann, after opening the stomach, dilatation may be employed from below. Impermeable strictures call for gastrostomy and rectal alimentation. See TREATMENT OF INTESTINAL OBSTRUCTION for thiosinamin.

(C). DILATATION. DIVERTICULUM.

Etiology.—*Dilatation involves the entire circumference; diverticulum involves but part of it.* Secondary dilatation may occur above stenosis of the œsophagus or above the cardiac end of the stomach. It seldom extends more than an inch and a half (4 cm.) above the stricture; it rarely follows pyloric stenosis. Primary (idiopathic) dilatation may be local or total. Marked dilatation above the cardia, Luschka's so-called "fore-stomach" is an example of the local form, which is usually congenital. The diffuse idiopathic dilatation results without any causal organic stenosis and is seen chiefly between the years of twenty and forty. Its actual cause is disputed. Most clinicians incline to the view advanced in 1888 by Meltzer, of New York, that its cause is a *cardiospasm* due to abolition of cerebral inhibition on the cardia; in fact Kraus reports a case with great atrophy of both vagi; Rosenheim maintains that atony and ectasia are primary and cardiospasm secondary. The role of trauma, acute œsophagitis and congenital weakness of the œsophageal musculature is uncertain. *Cardiospasm* produces a great hypertrophy of the cardiac muscle, even six times its normal thickness, and diffuse total dilatation so that the œsophagus may measure 18 inches (46 cm.) instead of 10 inches (25 cm.) in length or 12 inches (30 cm.) in circumference (Luschka). Though Zenker collected but 18 cases in 1878, the number is increasing; nearly a dozen were reported in 1904 (Sippy, Tyson, Kinnicutt, and Sossen), and up to 1905 over 90 are recorded. The mucosa is catarrhal, thickened, or eroded, and the muscularis is hypertrophied or fatty.

Symptoms.—The symptoms are gradual or sudden in onset. There is *dysphagia*, which may often be overcome by effort; the patient feels that the food does not reach the stomach. *Regurgitation* of the food, especially the portion last swallowed, usually occurs soon after eating or when the patient lies down; it may measure a quart, is neutral, decomposed if it has been retained for some time, and contains lac and butyric acids but no hydrochloric acid, pepsin or rennet. T regurgitated food may again be swallowed (rumination). Vomiting course is impossible. The pressure of the stagnant food or fluid in t gullet may produce vagus symptoms, such as dyspnœa or palpitatic The *sound*, when introduced, moves freely from side to side, but me at least temporary resistance at the cardia, though pressure usua overcomes the obstructing cardiospasm. One sound, introduced in the stomach, may recover HCl, pepsin, etc., but another, introduc *to* but not *beyond* the cardia, recovers fluid or food with a neutral action; methylene blue solution poured through the first tube retu blue; that recovered by the second tube is colorless. Distention of œsophagus with carbon dioxide gives *tympany*, or with water *duln* near the left of the sixth to the ninth dorsal vertebræ, sometimes ab the left costal arch, or even in the epigastrium. The œsophagosc may reveal the dilatation; a distinct shadow may often, though always, be seen by using the *x*-rays after the patient has eaten potat or bread which have been well mixed with bismuth. The *sec* *swallowing bruit* may be absent or altered, or sometimes sounds l fluid running into fluid. Marasmus and constipation and often de result, but the clinical course may cover years or decades.

Treatment.—Treatment is rather unsatisfactory. Internal galv ization or faradization has been recommended. Olive oil ℥ss. bef meals may help the fluid (or the sound) to pass the cardia. Continu use of the stomach tube may be effective, but in some instances caus œsophagitis; its life-long use is often required. Lockwood's pne matic bag may be introduced empty on a sound into the stomach, th inflated and withdrawn slowly and gently through the cardia; Strau praises this method. Gastrostomy and rectal feeding may be ind cated. Operation should not be delayed too long. The cardia ha been operated upon a number of times. Mikulicz operated six times—with five cures—by entering the stomach, forcibly distending the cardia and rupturing its fibers submucously.

Diverticulum.—This involves only an arc of the œsophageal circumference. Two types are distinguished: 1. *Zenker's pressure diverticulum* (pulsion-diverticulum) of which Rosenthal (1902) could collect but 180 cases. It is found chiefly in men (77 per cent.), and is a pouch which forms very gradually on the posterior œsophageal wall, usually near the junction of the pharynx and œsophagus, and nearly always consists of only the mucosa and submucosa,—a kind of pyriform or bottle-shaped œsophageal hernia. Its onset is very gradual, for the muscle, which is probably congenitally weak, yields before intra-œsophageal pressure, trauma, foreign bodies, and the stagnating food. The early *symptoms* are signs of pharyngo-œsophagitis, hawking and dysphagia, and food entering the sac rather than the œsophagus; the axis of the sac, which dips between the gullet and the spine, is roughly parallel with that of the œsophagus; many of its symptoms are those of a high œsophageal stenosis. The food stagnates and regurgitates with a fœtid odor. In 33 per cent. of cases a tumor appears in the supraclavicular region, which is dull when filled with food or tympanitic when distended with carbon dioxide; it sometimes can be smoothed out, but this makes the food regurgitate. The murmurs on swallowing are protracted (in 50 per cent.). A sound, if introduced, usually enters the sac, and another, if slightly bent, enters the gullet; this also differentiates the diverticulum lower in the gullet, which is even less common. Examination by the œsophagoscope is generally negative, but the x-rays may show a characteristic shadow. There may be pressure on the heart, trachea, vagus, recurrent laryngeal nerve, and brachial plexus. The clinical course is usually protracted, even to twenty or forty-nine years, and the spontaneous evolution is inanition in over 50 per cent. of the cases, gangrene (15 per cent.) or perforation with cervical phlegmon. *Treatment* includes feeding by means of the sound, rectal nutrition, gastrostomy, and surgical resection; this was first performed by von Bergmann (1890), 24 cases being recorded with 20 per cent. mortality. Diverticula low in the œsophagus can only be treated by long-continued sounding.

2. *Rokitansky's traction-diverticulum* is frequent; according to Riebold it is found in 3.5 per cent. of all autopsies. It is due to extra-œsophageal traction, which draws a small part of the gullet out into a funnel-like form. Over 80 per cent. is due to indurative periadenitis around caseous or anthracotic lymph glands. Cicatricial mediastinitis from pleuritis, pericarditis or vertebral disease may produce the same effects. It usually, therefore, occurs at the level of the tracheal bifurcation. Diverticula may result from both traction and pressure. Traction-diverticulum begins early in life, is usually single, is less marked on the anterior or lateral wall, barely exceeds five to ten mm. in depth and is rarely discovered during life, though some have been seen with the œsophagoscope. Its chief danger is ulceration at its apex and perforation into the trachea, bronchi, vessels, pericardium, pleuræ, or mediastinum, with resulting gangrene of the lungs, fatal hemorrhage, pneumothorax or phlegmonous mediastinitis. There is no treatment.

(D). CANCER OF THE ŒSOPHAGUS.

Etiology.—Its frequency is rated at ¼ to 1 per cent. of autopsies, and it is said by Petersen-Borstell to rank third among cancers (stomach 33 per cent., uterus 15 per cent. and œsophagus 6 per cent.). Its etiology is unknown. (*a*) Scars, trauma, ulceration and traction diverticulum are promoting causes; (*b*) 75 per cent. occurs in males, and 80 per cent. between the fortieth and sixtieth years; 8 per cent. occurs before the fortieth year; Stewart records a case aged twenty-three, and Heimann one aged nineteen.

Pathology.—Cancer of the œsophagus is almost always single and primary, though cancer of the stomach, pharynx or mediastinum may rarely involve the œsophagus secondarily. Most cases are squamous *epithelioma*, arising from its pavement epithelium; scirrhus, colloid and medullary forms are rare. *Localization*; most cases (57 per cent.) develop in the lowest third of the œsophagus; many (33 per cent.) in the middle, and the least (10 per cent.) in the upper third; its favorite sites are near the cardia, at the tracheal bifurcation level and just back of the cricoid cartilage. The muscularis sometimes hypertrophies above the cancer, where some dilatation may occur, but not very frequently.

Symptoms.—The onset is usually insidious. Signs of *œsophageal stenosis* (*v. s.*) develop in varying grades; stenosis generally advances slowly but is occasionally sudden. It may lessen as the cancer ulcerates. Its symptoms are those of *stenosis*, as *dysphagia*, rather indefinitely located pain, regurgitation of food and perhaps also of bloody mucus or of necrotic cancer tissue. Its signs are also identical, as obstruction on sounding, alteration of the murmurs, sometimes the detection by the œsophagoscope of a papillomatous or ulcerated neoplasm, and quite rarely tumor particles found in the fenestræ of the tube. *Cachexia* develops; constipation, indicanuria and peptonuria are usual; acetonuria is fairly frequent. Extension, by contiguity, occurs to the spine, mediastinum, and cardia, or in 60 per cent. of cases by *metastasis*; the mediastinal and peribronchial lymph nodes are usually invaded, the supraclavicular often and the upper cervical seldom.

Complications.—There may be pressure on the recurrent laryngeal (13 per cent.), vagus or mediastinal veins and arteries, on the sympathetic ganglia (13 per cent.) or brachial plexus. *Perforation* occurs in over 50 per cent., most often into the larger air passages, when aspiration pneumonia or gangrene may follow; into the lung, pleura (ichorous pleurisy or pneumothorax resulting); into the aorta (fatal hemorrhage), heart, pericardium, and mediastinum. Pyæmia, pulmonary tuberculosis, and marantic thrombosis, occur in some cases. *Death* is the inevitable outcome, usually within thirteen months; it occurs with emaciation, subnormal temperature, small slow pulse, slow shallow breathing, flattened abdomen, and perhaps intoxication symptoms, as coma and fever.

Diagnosis.—The diagnosis is based (*a*) on the fact that there is a stricture of the œsophagus, which in 90 per cent. of all cases is carcinomatous; (*b*) on evidences of neoplasm, as age, cachexia, cancerous nodes, metastases, and perhaps tumor particles on the sound. (*c*) Other tumors, as lipoma, myxoma, myoma (11 cases, Bryant, 1905), fibroma, or sarcoma, are of greater pathological than clinical importance, except accessible fibropapillomata of the upper gullet or lower pharynx.

Treatment.—The treatment is that of stricture or stenosis; nourishing enemata, feeding by the stomach tube, gastrostomy and for pain, narcotics are indicated. Œsophagectomy is seldom necessary, as less than 10 per cent. of these tumors occur in the cervical portion, accessible to surgical interference. Most of the operated cases die. As to gastrostomy, Schmidt in 1897 reviewed 236 cases, in which 45 per cent. did not live longer than a week, and nearly 90 per cent. not more than one hundred days.

(E). PERFORATION; RUPTURE; HEMORRHAGE OF THE ŒSOPHAGUS.

1. *Perforation* may result from causes within or without the œsophagus. Internal causes include ulcers, foreign bodies, diverticula, cancer, and use of the sound; external causes are aneurysm, gravitating abscess, lung cavities or gangrene, suppurating lymph glands or trauma. It is marked clinically by mediastinitis and cervical cellulitis, which is usually phlegmonous. Œsophageal fistulæ may communicate with the air passages, from which fluid or food are seen by the laryngeal mirror to be expectorated; a communicating tuberculous cavity may become flat when the patient drinks or eats, and the flatness is changed to tympany when the food or drink is expectorated; this is well tested by giving the patient milk, which will be brought up from the larynx. Aspiration gangrene, pneumopericardium, pneumothorax, and fatal hemorrhage are quite common causes of death.

2. *Spontaneous rupture* (so-called) is a very rare affection, there being only twenty-five certain cases on record. It is often confused with œsophagomalacia, in which the rupture is not longitudinal nor clean-cut as in genuine rupture. The first case, and one of the clearest, was reported by Boerhave (1714). It follows over-eating or severe vomiting, and most victims have been alcoholics. There is great and sudden pain over the lower œsophagus (where most ruptures occur) and over the xiphoid, severe vomiting, hæmatemesis, collapse, and subcutaneous emphysema. Death is the rule from collapse, phlegmonous or ichorous mediastinitis, pleuritis, or pneumothorax. Treatment is wholly expectant.

3. *Hemorrhage* may follow all varieties of œsophageal ulceration, external or internal trauma, toxic œsophagitis, softening, and aneurysmal or other perforations. Varicose veins in the lower œsophagus, first described by Le Derberden (1837), are most often complications of liver cirrhosis, less often of syphilis, senile atrophy, and pylethrombosis. The diagnosis and localization of the bleeding point is at best only suggested by the history and previous physical findings. *Treatment* consists chiefly of keeping the patient absolutely quiet, of giving morphine, and of rectal feeding, as in gastric hemorrhage.

(F). MOTOR AND SENSORY DISTURBANCES OF THE ŒSOPHAGUS.

These will be considered under AFFECTIONS OF THE VAGUS (*q. v.*, under NERVOUS DISEASES).

DISEASES OF THE STOMACH.

(A). ACUTE GASTRITIS (ACUTE CATARRH, ACUTE DYSPEPSIA)

Acute gastritis is a better term than catarrh, which implies a supe ficial inflammation of the mucosa. Simple gastritis will be first co sidered.

I. Simple Gastritis.

Etiology.—It is a very common primary and secondary affectio (*a*) Its most important *mechanical* cause is improper food or imprope eating; rich food, an excessive amount, *partial mastication*, washin down the food with water, hurried eating, eating when very hungr idiosyncrasies to certain kinds of food, decomposed articles containin bacteria (B. coli) or ptomaines (tainting of food is most common in h weather), and excessive ingestion of cold water and fruit are accesso factors. (*b*) *Thermal* factors include too hot or too cold food especially when taken on an empty stomach. (*c*) *Chemical* cause aside from acids and caustics (see TOXIC GASTRITIS), alcohol ranks for most; autotoxins may excite acute gastritis, as those of acute chronic nephritis, cholæmia, or gout. (*d*) Some cases are clearly i *fectious;* the condition may occur in variola, measles, typhoid, gripp and pneumonia, as a secondary manifestation, or occasionally as independent febrile affection; this happened in the Christiana epide reported by Hausemann (1880), in which 6,000 persons were affect within three weeks. (*e*) Certain conditions are somewhat *predisposi* to acute gastritis; for example, stasis (from diseases of the heart, lu or liver), tuberculosis, cancer, syphilis, chlorosis, diabetes, leukæmia, affections of the higher alimentary mucosa. In some cases a familial even hereditary tendency is noted, known as "the delicate stomac by the laity. Nervous factors, as anger or excitement, are someti apparently causative, though they may be confused with the digest neuroses.

Symptoms.—**1. Digestive Symptoms.**—These are sometimes abse as was demonstrated by Beaumont in the celebrated St. Martin ca of gastric fistula. The *appetite* is usually *lost* or perverted. The *ton* *is thickly coated* with white fur and rather dry mucus. The *mouth* *dry* and there is a disagreeable taste, great *thirst*, even though t salivary flow is often increased. Hiccough, *eructations*, a sense gastric distention, pressure, or *pain* and tenderness over the stoma are common. Nausea and *vomiting* characterize the more mark cases; the vomitus is pale yellow, bitter, and contains little or no hydrochloric acid, though lactic, butyric and fatty acids are common; ther is much mucus, which is frequently flecked with blood. Correspondin anatomical changes are found, though few uncomplicated cases come to autopsy; the mucosa, particularly near the pylorus, is red, swollen, smeared with adherent mucus, sometimes slightly eroded or dotted with punctate ecchymoses; the dilated racemous vessels, as seen by the gastroscope, or through a gastric fistula, are less conspicuous at autopsy for the blood in great part leaves the organ after death; microscopically the salient findings are leukocyte emigration and granular degeneration and swelling of the mucous and other glandular cells.

2. General and Other Symptoms.—Herpes is an occasional finding. *Constipation* is the rule, though diarrhœa or icterus sometimes occurs in gastro-enteritis. The *urine* is *scanty* because of the vomiting; it deposits a lateritious sediment and sometimes contains indican or acetone. Nervous symptoms—as frontal *headache*, vertigo, intercostal neuralgia, and depression—are common, but convulsions (acetonæmia) and other marked nervous manifestations occur infrequently in young and sensitive individuals. Fever is quite uncommon; when present, it is usually irregular and ephemeral.

Diagnosis.—The *diagnosis* is not difficult when the course is short and afebrile, as is usually the case. With fever or some constitutional symptoms it is an excellent practice to regard the acute gastritis as a symptom, until, at least, typhoid, exanthematous disease, or nephritis can be excluded by the later clinical evolution. Confusion is possible with a tabetic crisis or with the vomiting of pregnancy and hysteria.

Treatment.—(*a*) *Prophylaxis* embraces the etiological factors. (*b*) *Complete rest of the stomach* for one or more days is the first active indication; no rectal feeding is required for twenty-four to forty-eight hours, by which time the gastric equilibrium is in great part restored; warm water may be given by rectum to unload the bowels and to allay thirst. (*c*) If *gastric irritation* persists, the stomach should be thoroughly washed out by the *stomach tube*, using warm water with a spoonful of sodium bicarbonate to the quart. Gastric lavage is a better measure than the use of calomel or salines, which may irritate the inflamed, perhaps eroded mucosa. For the same reason emetics (apomorphine hypodermics, or ipecac by mouth) are to be avoided. *Epigastric fomentations* alleviate pain. Obstinate irritation and vomiting usually respond to Bartholow's prescription, modified as follows:

℞

Ac. carbolici	gr. iv.
Ac. hydrocyanici dil.	ʒss.
Bismuthi subnitratis	
Sodii bromidi	
Cerii oxalatis	āā ʒiss.
Mucilaginis acaciæ	q.s.
Aquæ menthæ piperitæ	q.s. ad ℥ij.

M. et S.—One teaspoonful without dilution every half to one hour for six to ten doses.

Opiates give immediate relief, but secondary nausea is an insuperable obstacle to their use. (*d*) *In convalescence*, dilute hydrochloric acid ♏ v to xv and bitters are given after meals, to stimulate and supplement the gastric secretion. The diet should be simple, moderate in bulk and the food should be thoroughly chewed.

II. Gastritis Toxica (Venemata).

Etiology and Pathology.—Acute toxic gastritis is caused by va alkalies, acids, and metallic poisons. Sulphuric acid produces a eschar, nitric acid a yellow one, alkalies a brown, copper a gre blue, silver a deep black eschar, and phosphorus produces an op milky stain. The pathological changes are usually severe, particu in places with which the corrosives remain longest in contact, a pylorus and posterior wall; these changes range between si epithelial desquamation and catarrh, to hemorrhagic infiltra diphtheroid patches, suppuration, and ulceration.

Symptoms.—The symptoms are those of gastritis simplex, but severe; pain is intense in the mouth, throat, œsophagus, and stom and is not relieved by the repeated vomiting. The vomitus is bloody, the epigastrium tender, and the abdomen tympanitic.

Prognosis.—The *outcome* varies; (*a*) collapse after a few hours day or so is frequent, with small pulse, shallow breathing, subno temperature, bloody albuminous urine, and other symptoms w vary with the poison ingested. (*b*) The stomach symptoms subside, and death may occur later from blood or visceral changes. Strictures may develop, or (*d*) there may be atrophy of the secr glands (achylia gastrica).

III. Phlegmonous Gastritis.

Etiology.—This is a rare form, of which 85 cases are recorded (1 It is an acute suppurative inflammation of the stomach, usually ca by the streptococcus; it begins in the submucosa and infiltrates the coats diffusely or forms a circumscribed mural abscess. It may primary affection (only 20 are recorded, 1905), but most often oc secondarily (75 per cent.) in sepsis, the acute infections, anthrax, local diseases as gastric cancer. The writer has seen only one which followed erysipelas. Eighty-five per cent. occur in adult m Alcoholism and traumatism are predisposing factors.

Symptoms.—The symptoms are gastric and septic. Besides symptoms of acute gastritis, the vomitus sometimes contains pus, epigastric pain is intense, meteorism is well marked, and there are n symptoms of peritonitis, which is a most common complication. few cases an epigastric tumor marks the circumscribed variety. Se usually in a severe form, causes the high fever (103–5 degrees) pronounced status typhosus, the small pulse, occasional icterus, petechiæ.

Diagnosis and Treatment.—A *diagnosis* is almost impossible, *outcome* is generally fatal during the second week, and the *treatm* symptomatic, *viz.*, rectal nutrition and stimulation, narcotics for and vomiting, and hydrotherapy for fever. Some cases apparently by rupture into the stomach with cicatrization, and, if the affection is suspected, the advisability of operation can be considered.

IV. Diphtheritic Gastritis.

This form is due to the Bacillus diphtheriæ, and is very rare. Diphtheroid gastritis, caused by streptococcic and other infections, is more common; it may develop after gastritis toxica, smallpox, pneumonia and kindred maladies.

V. Parasitic Gastritis.

This form is, in rare instances, due to the Oïdium albicans, favus parasite (Kundrat), anthrax bacilli, larvæ of insects and tineæ.

(B). CHRONIC GASTRITIS.

Chronic gastritis (dyspepsia, catarrh) is characterized pathologically by degeneration of the parenchymatous and infiltration of the interstitial tissue, and clinically by mucus formation and decreased secretion.

Etiology.—(*a*) The factors are the same as in acute gastritis, from repeated attacks of which chronic inflammation may result. Abuse of drugs, especially cathartics, is an important factor. Alcoholism and improper eating are most important. Eating hurriedly or at irregular hours, inadequate mastication, washing down the food with much fluid, excessive use of coffee, tea, ice water, carbohydrates, pastries, or fried foods, are salient factors. The chewing of tobacco and to a lesser degree smoking may cause chronic gastritis; stomatitis, carious teeth, and pyorrhœa alveolaris, are important causes. Most cases occur in adult males. (*b*) It often accompanies other gastric diseases, as cancer or ectasia. (*c*) It is frequent in tuberculosis, in the anæmias as chlorosis or pernicious anæmia, and in nephritis, gout, diabetes, syphilis, amyloidosis, and Addison's disease.

The acid form (see SYMPTOMS) of chronic gastritis is usually caused by excesses in eating, drinking, and smoking; the subacid form, by the abuse of spirits.

Pathology.—The pathological changes are observed chiefly in the *pyloric* region. The vessels are dilated, imparting a reddish-brown color, which in the later stages changes to gray. The mucosa is swollen, lax, and covered with tenacious mucus. Sometimes punctate hemorrhages or erosions are observed. In a few cases the mucosa is rough, even papillomatous (*gastritis polyposa, état mamelloné*). The muscularis is not essentially changed, as a rule, but is sometimes hypertrophied near the pylorus, or it may be atrophied. Histologically, the glandular cells are granular and fatty, or show a mucoid degeneration which extends from the surface toward the deeper layers; the parietal cells cannot be distinguished from the principal cells, the glands are often large, sacculated, or cystic, and the tubules show irregular ramifications; eventually the glandular structures may completely atrophy (see next sub-heading, ACHYLIA, ATROPHY). The interstitial tissue shows leukocytic infiltration.

Symptoms.—The symptoms resemble those of acute gastritis, but begin more *insidiously* and endure longer.

1. Oral Symptoms.—The *tongue* is generally, but not always, *coated* with moist white fur, reddish at its tips and indented on its edges; these conditions may also result from alcoholic or nicotine stomatitis. There is frequently a bitter, sour, *salty taste* in the mouth, and sometimes a marked *fœtor ex ore*. Increase in the saliva results chiefly from the coincident stomatitis.

2. Gastric Symptoms.—(*a*) *Gastric pain* sometimes results from spasm of the gastric musculature but is less frequent than is a sense of distention or pressure after eating. Soup and fluids (unless they are cold) generally cause no distress; coarse or hard foods, as beefsteak, hard bread, smoked meat, cabbage, hard-boiled eggs, or cheese, do cause distress. (In functional dyspepsia, any kind of food, hard or soft, may cause distress). In some cases of gastritis there is distress when the stomach is empty. There may be some tenderness, but it is much less than in cancer or ulcer and responds readily to treatment. (*b*) *Belching* of gas is common; eructations may be odorless, or may contain fatty acids and even inflammable gas. Eructations of hydrochloric acid are rare. (*c*) *Nausea* is more frequent than is vomiting, which may result from other coincident conditions, notably from chronic pharyngitis. The vomitus shows the same findings as the test meal (see GASTRIC CHEMISM). In alcoholics morning vomiting is common (*vomitus matutinus potatorum*), chiefly of saliva swallowed during the night, and of mucus.

3. Gastric Chemism.—Our intimate knowledge of the chemical changes in the stomach dates from the introduction of the stomach tube by Leube, and the vast amount of work done by many workers, especially Riegel, Ewald, and Boas. After washing out the stomach, the Ewald test breakfast is given, consisting of a piece of dry bread and a cup of weak tea without sugar or cream. In an hour the stomach tube is again introduced and the gastric contents withdrawn by expression (the patient compressing the abdomen and straining), or by Ewald's aspiration bag, similar to Politzer's ear bag; it is seldom necessary to pour in any water unless some condition obtains in which the hydrochloric acid is much decreased. Innumerable errors result from confidence in a single examination and assigning undue importance to the gastric chemism alone. It is best to examine the contents chemically without filtration.

(*a*) The *gross examination* of the stomach contents shows coarse particles of food when the hydrochloric acid is absent or greatly reduced, or fine subdivision when it is present in fair amounts. A rancid odor denotes fatty acids, and a foaming appearance is caused by fermentation. *Mucus* is seen in practically all cases, and most clinicians do not recognize a simple gastritis without mucus formation, though Ewald distinguishes (i) simple gastritis in which the acid and ferments are decreased; (ii) a mucous gastritis, in which the acid is greatly decreased, the ferments are present, and there is much mucus; and (iii) an atrophic gastritis which will be considered separately. Mucus usually indicates a reduction in the hydrochloric acid. Normally there is little or no gastric mucus, though there is rather more with an amylaceous than with a proteid diet. Mucus of gastric origin is intimately mixed with the food and is not yellow, nummular, pigmented, or mixed with cells, as occurs when the mucus comes from the mouth or respiratory tract.

(*b*) The *HCl is reduced.* It is recognized by the Günzburg test (2 gm. phloroglucin and 1 gm. vanillin dissolved in 30 c.c. of absolute alcohol); a few drops of the solution are added to an equal amount of the filtered gastric contents and slightly heated, without boiling, in a porcelain dish, when a carmine-red color develops, indicating free hydrochloric acid even in dilution of 1 to 20,000 parts, and not reacting to lactic or butyric acids; the solution should be kept in a dark bottle, for it degenerates in the sunlight. The solution should not be kept over two months. Boas recommends the following test, which gives the same reaction as Günzburg's test and is less expensive: resorcin 0.5, sugar 0.3, dilute alcohol q. s. ad 10.0 gram. HCl exists in a combined form or as free acid, which latter probably serves as an antiseptic.

To find the *total free HCl,* one of three simple methods may be employed. (*a*) Filter paper is saturated with a 0.5 per cent. alcoholic solution of dimethylamidoazobenzol, and dried; HCl colors this solution a cherry-red color; when the reaction is obtained on the paper with the gastric contents diluted three to six times, about the normal percentage of HCl is present; if obtained with less than three dilutions, subacidity (hypochlorhydria) exists; if with more than six dilutions there is hyperacidity (hyperchlorhydria). (*b*) To 10 c.c. of the gastric contents, 25 to 30 c.c. of Günzburg's reagent is added, and then, little by little, the decinormal soda solution is added until the characteristic red no longer appears on the porcelain dish; the percentage of HCl is obtained by multiplying each c.c. of decinormal solution used by 0.00365 (for each c.c. of the decinormal solution is neutralized by 0.00365 of HCl), and again multiplying by 10. (*c*) To 10 c.c. of gastric contents is added a few drops of the indicator (0.5 per cent. solution of dimethylamidoazobenzol in strong alcohol); it is placed in a beaker and, if free HCl is present, the yellow color imparted by the indicator will change to bright red; a decinormal solution of caustic soda (4 gm. to 1,000 c.c. of distilled water) is then added drop by drop to the gastric contents, until the color of the indicator has been changed from red to bright yellow; the number of c.c. of decinormal solution used is multiplied by 0.00365 and again by 10 to give the amount of free HCl. Very often the result is expressed terms of the number of c.c. of decinormal solution used; *i.e.*, if 4 c were employed, 4 x 10 = 40, the free HCl. The usual amount is 30 40.

The *total acidity* (HCl, lactic and other organic acids, acid ph phates and albumins, albumoses and peptones) is estimated by addi two drops of a 1 per cent. alcoholic solution of phenolphthalein an indicator) to 10 c.c. of the gastric contents; to this is added, dr by drop, a decinormal solution of caustic soda (4 gm. to 1,000 c.c. distilled water) until the red color, due to the alkali acting upon phenolphthalein, no longer disappears on gentle shaking; this, ho ever, is not the true end reaction, for the decinormal solution must added till the rose-red color is no longer even deepened in hue by alkali. As 4 to 8 c.c. of the decinormal solution are usually requir 4 or 8 x 10 = normal total acidity; *i. e.*, 40 to 80 expressed in terms of decinormal solution (corresponding to 0.15 to 0.25 per cent. HC If the acid reaction of the gastric contents is due to HCl alone, and organic acids are present, each c. c. of the decinormal solution c responds to 0.00365 gm. HCl and therefore the number of c.c. quired to neutralize the gastric juice x 0.00365 x 10 = the per cent HCl. In most cases determination of the total acidity is sufficie A total acidity under 20 gives no reaction with Congo paper; *i. e.*, th is no free HCl. The bread of the test breakfast (phosphates chie accounts for the acidity of 5 to 6; if the total acidity ranges from 5 t we may be almost certain that the gastric glands are atrophied.

Combined Hydrochloric Acid.—"The method elaborated by Co heim and Krieger, modified by Fisher, is accurate and convenie The method is as follows; Determine the total acidity by titratin c.c. of the gastric filtrate with 0.1 normal NaOH solution, using a 1 cent. alcoholic solution of phenolphthalein as an indicator. Take anot 5 c.c. of the filtrate and sufficient calcium phosphotungstate solutio make 20 c.c. and call this Ta_1. Let it stand three or four minutes, a animal charcoal, and filter. Determine the total acidity of this filtra using a 1 per cent. solution of rosolic acid as an indicator; call this T Then Ta_1 minus Ta_2 equals the combined HCl in the 5 c.c. of filtrate of the gastric contents used. If the gastric contents show absence of free HCl, proceed as follows:

1. Determine the hydrochloric acid deficiency by adding 0.1 nor hydrochloric acid, using a 1 per cent. alcoholic solution of dimet amidoazobenzol as an indicator. Determine the total acidity of mixture, using phenolphthalein as an indicator. This total aci minus the quantity of hydrochloric acid added will give the origi total acidity of the 5 c.c. of the filtrate of gastric contents employ
2. Take 5 c.c. of the gastric filtrate, add 0.1 normal hydrochl acid sufficient to make the new total acidity at least 60 per cent.; a sufficient calcium phosphotungstate solution to make 30 c.c., a titrate with rosolic acid as an indicator, calling the resulting acidity T The difference between Ta_3 and Ta_2 will represent the quantity

hydrochloric acid combined with all the albumin in the 5 c.c. of gastric filtrate employed. Call this total CHCl. Then this total CHCl minus the hydrochloric deficiency equals the combined HCl in 5 c.c. of the gastric filtrate.

Employed subacid gastric contents	5	c.c.	
HCl deficiency (dimethylamidoazobenzol)	1	"	= 20
Total acidity (phenolphthalein)	3	"	= 60
Original total acidity	(60-20)	"	= 40
Ta_1 Added HCl in excess	3	"	= 60
Added calcium phosphotungstate	18	"	=
Ta_2 Total acidity (rosolic acid) of	30	"	= 30
Total CHCl Total possible combined HCl	(60-30)	"	= 30
Actual combined HCl in gastric contents	(30-20)	"	= 10

Ta_2 — Ta_3 = Total HCl.

Total CHCl — HCl deficiency = combined HCl." (Musser, *Medical Diagnosis*.)

In chronic gastritis the *hydrochloric acid* is *found decreased*, or even absent by these tests. In a few cases, as described by Riegel and Boas, the acid is normal, showing a total acidity of 43 to 56; this form is called gastritis acida, though its separation as a type is scarcely justifiable; these are usually light or incipient cases and probably some of them are neurotic.

(*c*) *Rennet and pepsin* are rarely totally absent, though usually *decreased*. Pepsin is tested by mixing the gastric juice with egg albumin; 25 c.c. of normal gastric juice will dissolve 0.05 gm. of serum albumin in one hour, the same amount of fibrin in one and one-half hours, and of egg albumin in three hours; Petit claims that pepsin will dissolve 500,000 times its weight of fibrin in seven hours. Pepsinogen is tested in the same way with the addition of a few drops of hydrochloric acid, which liberates the enzyme (pepsin) from the pro-enzyme (pepsinogen). Boas especially has insisted on the importance of the rennet ferment, which coagulates milk; if no rennet is present, the gastric filtrate will not coagulate milk.

Lactic acid is seldom found (see CANCER OF STOMACH). The motility of the stomach is unimpaired. Leukocytes and epithelial cells are found at times, and in rare cases shreds of gastric mucosa (Gastritis exfoliativa, Parisier).

4. **Other Symptoms.**—(*a*) *Vertigo e stomacho læso* (Trousseau),—gastric vertigo, is probably neurasthenic rather than gastric. The so-called dyspeptic asthma is not really asthma, but a dyspeptic dyspnœa, and probably results from pressure of the distended stomach on the heart. Headache, physical and mental lassitude, a degree of melancholia, intercostal neuralgia, tender points between the shoulders or over the lower cervical spines, and palpitation, are fairly common. (*b*) The bowels are generally *constipated;* diarrhœa and tympany at times result from marked gastric fermentation or extension of the catarrh to the gut, when catarrhal icterus may result. Boas and Schmidt find regularly undigested connective tissue in the movements. (*c*) The *urine* is frequently reduced and shows a lateritious deposit, phosphates and oxalates. (*d*) The general nutrition is usually fairly well maintained

if the intestines functionate well. (*e*) The motive and absorptiv power of the stomach is variously regarded; it may be impaired, normal or even exaggerated, the food being sometimes hurried into the intestine.

Diagnosis.—Leube insists that chronic gastritis should never be diagnosticated until cancer, ulcer and functional disease have been finally excluded. Its cardinal points are (*a*) its steady, slow, chronic course; (*b*) the presence of mucus, and (*c*) decreased secretion. (For its differentiation *v. i.* ULCER.)

Treatment.—1. Prophylaxis.—Recurrence of the trouble is prevented by treatment of the causal conditions; digitalis is indicated in gastric stasis of cardiac origin, and iron in anæmia.

2. Hygiene.—Hygiene concerns regularity in living, moderation, recreation, massage, rational exercise, and avoidance of haste in eating.

3. Diet.—F. A. Hoffman justly remarks that every one "has th stomach he deserves." Slowness of eating, drinking only after the sol part of the meal is eaten, and thorough mastication, are frequent curative with no medicinal treatment. The importance of thoroug insalivation of the food is manifest when we remember that the ptyali operates for nearly an hour in the unchurned contents of the cardia end of the stomach. As Ewald states, "die Diät der Magen-Kranke fängt im Munde an." The *teeth* should receive such care as the treatment of pyorrhœa, extraction of carious teeth, and fitting of prope plates. Food should be taken in moderate amounts, and the stomac should be given sufficient rest, though most dyspeptics do not ea enough (von Noorden). Pawlow has shown that the relish of foo excites the flow of the gastric juice; he names it the "appetite juice.'

KIND OF FOOD—The term "easily digestible food" is extremel relative, but the kind of food is less important than careful masticatio and insalivation. In severe or alcoholic cases an absolute milk die may be tried, giving equal parts of milk and Apollinaris water, to whic grains x of sodium bicarbonate, grains x of sodium chloride, and grai v of magnesia levis are added. Predigested milk and Mosquera's be jelly (see TYPHOID) soon become distasteful to the patient, and are i dicated chiefly in severe catarrh with much mucus formation or wi atrophy. Generally speaking, the diet should be mixed, but protei are superior to carbohydrates, which are prone to ferment. The *pr teids* which may be given are chopped lean meats, scraped beef, boil mutton, veal, roasted steak, broiled tenderloin, pigeon, chicken, eg boiled one minute, somatose, and nutrose. Chittenden has shown t we eat twice as much meat as is necessary. Beef tea, heavy fresh be hard-boiled eggs, pork, game, fish, warmed-over (therefore hardene meat, turkey, goose, duck, smoked or cured meat (possibly exce ing bacon) and cheese must be interdicted. *Carbohydrates* are be withheld when there is obstinate fermentation. Those which m be allowed are arrowroot, well cooked sago, tapioca, or rice, white bre zwieback, and toast; those which may be occasionally allowed or tri later are mealy potatoes, spinach, pea or bean soups; those to be fo bidden are substances containing much cellulose, hard bread, brown bread, oatmeal, coarse vegetables, uncooked green vegetables, most fruits, pies, and pastries. It is interesting to note that most of the carbohydrates ingested usually reach a maximum amount in the intestine in two hours (some reach the bowel in ten minutes), which time is twice the maximum time for proteids and two and a half that for fats. Carbohydrates, therefore, should be eaten before the proteids, that they may the earlier leave the stomach. *Fats* are a necessary part of the diet, especially moderate amounts of butter and cream. Von Noorden insisted on their use. Other forms of fat are injurious. Greasy soups are to be avoided.

Accessories.—No seasoning is necessary, but liberal quantities of salt may be used. Liebreich insists that condiments in moderate amounts are helpful. Tea, coffee and alcoholic beverages are usually prohibited. Smoking should be interdicted or restricted.

4. **Gastric Lavage.**—Lavage is indicated when there is formation of mucus, fermentation, deficient secretion, and delayed digestion. It is generally considered the most helpful measure but in the writer's experience is seldom indicated as there is no stagnation of food. Morning lavage is best for removal of mucus, and lavage before the evening meal for delayed digestion or fermentation. Warm water is used, to which sodium chloride or bicarbonate, two spoons to the quart, may be added to dissolve the mucus, or salicylic acid (1 to 1,000) to modify fermentation. The stomach should be washed until the water returns clear. When the use of the stomach tube is resisted or contra-indicated, as by exceptional nervousness, modified lavage may be employed, the patient drinking before breakfast two glasses of warm water to which a teaspoonful each of sodium chloride and sodium bicarbonate has been added. This process is essentially the same as drinking the Kissingen, Wiesbaden, and other waters. The sodium bicarbonate acts locally, and the sodium chloride acts both locally and systemically, for small doses increase the hydrochloric acid and large doses reduce it by increasing the alkalinity of the blood. Carbonated waters may be used, which stimulate secretion and absorption and neutralize abnormal acids.

5. **Medical Treatment.**—(*a*) The value of hydrochloric acid is disputed. Its first indication is to convert pepsinogen into pepsin, whence it is useless in atrophic gastritis save as it stimulates the flow of bile. Its second indication is as a digestant; many specialists deny that it has this action, maintaining that sufficiently large amounts cannot be given; but Stewart in his excellent articles says that he employs doses of 20 drops and gives them every twenty or thirty minutes until ʒ j to ij have been given. Its third indication is as an antizymotic, which Boas holds is its sole indication; it should not be given until about half an hour after meals, during which period the saliva is acting upon the carbohydrates; some writers incline to the view that at the best hydrochloric acid is only a stomachic, but Pawlow has shown that it increases the flow of pancreatic juice and perhaps of bile. (*b*) Boas takes the extreme view that

pepsin is useless. It should be given as a powder, for solutions rapidly deteriorate and are usually strongly alcoholic. Like malt preparations, many forms offered on the market are inert. Pancreatin is very frequently ineffective. Taka-diastase is beneficial in some cases. (*c*) *Bitters* are best exhibited before meals; though their mode of action is disputed and their stimulation of secretion has been denied, they surely act upon the appetite or on the nervous structures, and increase the flow of saliva and gastric juice and probably also the pancreatic juice. Strychnine or nux vomica is the best of this group of drugs and is especially valuable in alcoholic cases. All bitters should be given in solution, as their bitter taste is their chief mode of action.

℞
Strychninæ sulphatis . . . . gr. j.
Acidi nitrohydrochlorici . . . . ʒj.
Tr. gentianæ comp. . . . . ℥iv.
M. et S.—One teaspoonful half an hour after meals in half a glass of water.

Condurango bark (fluid extract ʒss) is prepared by macerating the bark in cold water. Some bitters, as gentian, may irritate the stomach and induce diarrhœa. (*d*) *Antizymotics* (antifermentatives), as creosote or beta-naphthol, often cause irritation. Carbolic acid gr. j, thymol gr. ss to j, resorcin gr. ij to v, after meals, act on the stomach, and salol gr. x and beta-naphthol gr. x (in capsule because of its bitterness) are intestinal antiseptics. (*e*) *Gastric sedatives* are seldom indicated except in the hyperacid type. Bartholow's combination (see ACUTE GASTRITIS) meets all indications. Silver nitrate is sedative, astringent, and alterative, but may cause nephritis or argyria; it is given in pill form, half an hour before meals, in doses of one-fourth of a grain (perhaps with quite small doses of opium or belladonna). Zinc oxide, gr. iij to v, given before meals, is valuable in alcoholic gastritis. Extract of belladonna in one quarter of a grain doses is helpful in pain, especially in hyperacid gastritis. Pyrosis and spasm may be considered under this heading. In pyrosis of fatty and other organic acids, alkalies may be employed, though sparingly; Trousseau gave sodium bicarbonate grains v, creta preparata grains x, and magnesium carbonate grains v after meals. Spasm from gas or overloading of the stomach is modified by spirits of chloroform ♏ xx and tincture of asafœtida ʒj; spasm suggests erosions near the pylorus, when extract of belladonna gr. ¼ and subnitrate of bismuth gr. x are distinctly indicated, after meals. (*f*) *Other measures*, as faradization, massage, and hydrotherapy, are less valuable than in gastric atony and sensory neuroses of the stomach. In constipation cathartics should be avoided, as they cause irritation and constipation recurs. Hot water, gastric lavage, colonic flushings, and perhaps the mildest saline aperients, are indicated.

Achylia Gastrica.

Etiology.—The term was introduced by Einhorn in 1892 to designate al suppression of gastric digestion. Atrophy of the stomach was t described by Austin Flint (1860) and by Fenwick (1877), Ewald, incke, and Einhorn. It most frequently occurs secondarily in can- of the stomach and in toxic gastritis, but also in other gastritides, ncer of distant organs (mammæ, uterus, intestine, œsophagus), betes, pernicious anæmia, tabes, and in neuroses. In rare cases it velops as a seemingly primary affection, possibly as a congenital ndition (Martius). It occurs oftenest in middle or advanced life, ugh isolated cases are reported at eighteen to twenty-five years of e.

Pathology.—Pathologically two main forms exist, (*a*) one *cirrhosis ntriculi*, in which the stomach is very small and thickened so that its lls may measure several centimeters in thickness, and (*b*) another, *thisis ventriculi*, in which the walls are thin or "wasted" and the mach is lax or dilated. In some cases atrophy has been found in issner's and Auerbach's plexuses. The glandular structures are sted (anadenia).

Symptoms.—Symptoms may be absent if gastric motility is good l the intestinal glands are normal (von Noorden). If the same pro- s exists in the gut, diarrhœa, marasmus and death result.

1. **Gastric Symptoms.**—There are no pathognomonic gastric symp- ns. Pain is variable; it may be absent or moderate, though in ne cases lancinating pain and other symptoms like those of hyper- orhydria may exist (Einhorn). The appetite is also variable. Vomit- does not always occur but when present the vomitus always ntains coarse, poorly digested particles.

2. **Test Meal**—The recovered food is coarse; the total acidity is o 4, corresponding to the amount in the food ingested. No HCl ound (achlorhydria), no pepsin (therefore no peptones), no rennet erefore no coagulation of milk), no blood, and according to most iters no mucus; according to Riegel and Schmidt, some mucus may present, due to metaplasia of the gastric mucosa to one resembling it of the intestine, the cells of which secrete mucus; as they in turn ophy the mucus disappears. Lactic acid is found but rarely. le gastric *motor power* is usually unimpaired, and the stomach is ten emptied in less than an hour. In the contracted cirrhotic form, e stomach may hold but a few ounces; in the phthisic form ectasia ay occur. In some scleroses of the stomach a sound is introduced to the stomach with difficulty, hydrochloric acid is present, the mach cannot be distended with gas, and its capacity is enormously creased.

3. **Other Symptoms.**—Headache and vertigo are common. Con- pation is the rule; in some cases diarrhœa may be the sole salient mptom, so that it apparently indicates an intestinal lesion (Oppler).

Symptoms like those of pernicious anæmia (Henry and Osler) or like gastric cancer (author) mark some cases. The acidity of the urine which is normally present at the height of digestion, is naturally absent. The test meal establishes a clear diagnosis.

Prolonged observation may be necessary in order to exclude the secondary atrophic gastritis of cancer. Amyloid gastric disease and nervous achylia are eliminated by consideration of other somatic findings or symptoms.

Prognosis.—Life may be prolonged for years by compensating intestinal digestion, but recovery is impossible after actual atrophy develops.

Treatment.—Treatment is necessarily palliative. Thorough mastication of food is imperative. Cellulose is to be avoided, and thick, plain, leguminous purées should be given. Butter is well tolerated. The meals should be small and frequent (every three or four hours). HCl should be given in full doses (♏xx to xxx), Pawlow having demonstrated its stimulating action on the pancreatic flow. Water relieves the pain, which resembles that of hyperchlorhydria (Einhorn).

(C). DILATATION OF THE STOMACH; MOTOR INSUFFICIENCY.

The nomenclature of this and allied conditions is a matter of dispute; dilatation of the stomach (gastrectasia) is only a symptom, and is understood as a more or less lasting dilatation with motor insufficiency.

1. Acute Dilatation.

Acute dilatation and atony (loss of muscular tone) are rare, but have been observed by Fagge, Fraenkel, Hunter, Osler, and Boas.

Somewhat over 50 cases of acute dilatation are recorded.

Etiology.—(1) Most cases follow abdominal trauma or laparotomy (36 per cent. according to C. Thompson); gall-bladder operations are the most frequent type. The two cases seen by the writer were in women over fifty years of age who had been operated for fibroids of the uterus. (2) It may follow acute infections as pneumonia or typhoid; or (3) overloading of the stomach; in one of Kolisko's autopsies in Vienna, the author saw an enormously dilated stomach in a man who had, on a wager, taken a large number of charged siphons, and died soon afterward from gastric distention and reflex paralysis of the heart. (4) Most cases occur between twenty and thirty years of age. (5) Traction or obstruction of the superior mesenteric vein is the most favored etiological factor.

Symptoms.—The stomach is enormously distended; there is vomiting of brown-gray to black material which often runs out of the mouth, may be very copious and sometimes is offensive. There is usually no free HCl; sometimes there is lactic acid; succussion sounds are fairly frequent. The abdomen is greatly distended. There is much collapse and often abdominal pain.

Prognosis and Treatment.—Eighty-five per cent. die, and usually within a week. The indications are: early gastric lavage; rectal feeding; strychnine in large doses and a few doses of atropine (under which the writer observed one recovery); salt solution under the skin, and other measures for shock; and in appropriate cases gastrostomy or gastro-enterostomy.

2. Chronic (Permanent) Dilatation and Motor Insufficiency.

In the following discussion it must be borne in mind that various grades of distention and atony may occur, that dilatation may be associated with increased power of the gastric musculature or with atony, and that motor insufficiency (inability of the stomach to propel food into the intestine) is not necessarily, though very often, associated with dilatation of the stomach. Dilatation and motor insufficiency are most prevalent in middle or advanced life.

Etiology.—**1. Pyloric Stenosis.**—Its causes and symptomatology are varied. It is most often cancerous, but may also follow round or corrosive ulcers, chronic gastritis leading to hypertrophy of the pyloric musculature (stenosing gastritis), benign tumors of the stomach, as pedunculated polyps occluding the pylorus, and congenital stenosis, of which Shaw and Elting (1905) collected 145 cases; under one year of age 45 cases of congenital hypertrophic stenosis are reported, most of which were less than four months old. It may also result from congenital duplicature of the pyloric mucosa, from rotation of the stomach on its axis, from angular bending of the fixed pylorus by sinking of the stomach (gastroptosis), or from adhesions due to cholecystitis. Ricard and Chevrier collected nineteen cases of tuberculous pyloric obstruction. Stenosis of the duodenum produces essentially the same results as pyloric stenosis and may follow duodenal ulcer, diverticulum, or twisting. The pylorus may suffer compression stenosis from tumors of the liver, colon, kidney, or pancreas. Temporary or permanent pyloric spasm (*v.* Gastric Neuroses) from fissure, erosion, ulcer, or hyperchlorhydria may cause the obstruction. Payr in 1904, collected 58 cases in which pyloric stenosis was associated with intestinal stenosis (chiefly from round ulcer). The pathological sequence of pyloric stenosis is retention of food within the stomach and hypertrophy of its musculature in attempts, sometimes partially successful and at other times vain, to force the food through the narrow or closed pylorus. The analogy is close between this change and that observed in hypertrophied and dilated bladder from prostatic enlargement, or hypertrophy with dilatation of the heart in obstructive valvular disease. In all three instances the hypertrophied muscle eventually degenerates. Dilatation of the stomach may be considered, then, as a disturbed compensation.

2. **Muscular weakness** (motor insufficiency, insufficiency of t stomach, hypotony, atony, myasthenia gastrica), constitutes, in co trast with the first group of causes, a functional and, generally spea ing, a more reparable type. (*a*) Atony dependent on general caus may occur in acute infections, hysteria, neurasthenia, anæmia, a possibly in cerebrospinal disease. The atonic tendency may be a quired or congenital. Rickets is thought to cause certain juven cases. (*b*) Atony may result from gastric conditions, as trauma, ca cer, gastritis, hypersecretion, peritoneal adhesions, cholecystitis wi pyloric adhesions, diaphragmatic pleurisy, and diastasis of the musc recti abdominales. Overloading the stomach with heavy meals, e ing indigestible foods or excess of vegetables and carbohydrates, be drinking, milk in large quantities, much water ingested with hea meals, as in diabetics, swallowing of foreign bodies by insane subjec etc., may develop muscular insufficiency. The stomach is usual enlarged, contains residual food, and shows a lax, often wasted a thinned musculature.

Symptoms.—The symptoms of dilatation (again emphasized to a symptom only) differ greatly in degree, ranging from absolute pylo stenosis to the least motor weakness. The symptoms are partly m chanical, partly chemical.

1. **Gastric Symptoms.**—(*a*) The condition of the tongue is variabl it is sometimes coated, sometimes clear. (*b*) The appetite is al variable. (*c*) There is usually thirst and dryness of the throat, mo frequently in hypersecretion than in cancer. (*d*) Singultus, *pyros* of fatty acids, oppression over the stomach, a cramped feeling or on of unrest, and eructation of various gases, are frequent. Gases ar never formed without motor insufficiency; the kind of gas is of mor scientific than clinical interest, but includes hydrogen sulphide, hydro gen, oxygen, nitrogen, carbon dioxide, and even inflammable gase (*e*) *Vomiting* is always present in pyloric obstruction and in marke motor insufficiency. It occurs easily, and is often rather belched u than vomited. It occurs when the stomach is full, frequently durin the night or at intervals of days; five quarts may be evacuated. It i acid, sour, and often greatly decomposed. Three layers are noted, brownish foam, a gray-brown fluid, and a sediment of food, stones, an seeds, which may have lain for days in the stomach. Gas often bubble up as the vomitus stands. If gastric juice only is found, it indicate hypersecretion with motor insufficiency. Chemically, butyric acid peptones, and, in cancer, lactic acid, are found; when the obstructio lies in the duodenum below the papilla of the common duct, bile i also present. Microscopically undigested meat or starch, in benig stenosis sarcinæ, and in cancer the Oppler-Boas (lactic acid) bacilli yeast, and other bacteria, are found. HCl is absent in cancer. (*f* *Pain* is frequent; it is cramp-like, boring or burning.

2. **Test Meal, Motility, and Resorption.**—(*a*) The *chemism* depends on the causal affection, particularly if it is gastric (see GASTRITIS, ULCER, CANCER, HYPERCHLORHYDRIA, HYPERSECRETION). Hyperchlorhydria

and hypersecretion (except in malignancy) are usually present and are sequential rather than causative. (*b*) *Motility* is best tested by *Leube's test dinner*, consisting of 13 ounces (400 c.c.) beef soup, 7 ounces (200 gm.) beefsteak, one to two ounces (60 gm.) bread, and 7 ounces (200 c.c.) water, which should leave the stomach physiologically in not more than seven hours. If food is found somewhat later, stagnation is present; if found in the morning when Leube's meal has been given at night, there is great stagnation. The long stay of food in the stomach shows practically what the vomitus shows, *i. e.*, fermentation. Fluid is not absorbed by the stomach, whence the measure of its residual amount is important. There is more fluid in the stomach than was ingested, which is accounted for by hypersecretion of the gastric juice, actual transudation, mucus and perhaps regurgitation from the intestine, or hemorrhage. Other tests are less reliable;—the *oil test* consists of administering three ounces (100 c.c.) of olive oil, 70 to 80 per cent. of which should leave the stomach in two hours; if more than 20 to 30 c.c. remain after this time, it is indicative of motor insufficiency. *Salol*, grains xv, given just after eating, should normally reach the intestine within an hour, where it is split up into salicylic and carbolic acids; in testing the urine passed in forty-five to sixty minutes after eating, salicyluric acid should be found (reacting violet to tincture of the chloride of iron); salol is normally eliminated in twenty-four hours, and its presence in the urine for a longer time also indicates delayed absorption and dilatation; two errors are possible in this test, *viz.*, its possible decomposition by alkaline mucus in the stomach, and its failure to decompose in the gut when there is acid intestinal fermentation (Brunner and Huber). Resorption may be tested by giving five grains of potassium iodide after eating. It should be absorbed and secreted by the saliva in seven to fifteen minutes; this is tested by adding nitric acid to the saliva and touching with starch paper, which will show the blue iodine reaction; a possible error is the prompt absorption of the KI when food is not absorbed. In general a well-worked-up homogeneous chyme means that the motor function is normal; coarse particles occur when there is motor insufficiency.

3. **Physical Signs.**—(*a*) *Inspection* in a good light is very important. A visible prominence of the dilated organ may be seen below the navel, sometimes showing clearly the abnormally low upper and lower curvatures, the upper curvature never being seen normally. In obstruction a peristaltic wave passing from left to right, or an antiperistaltic wave in the opposite direction, often tells the whole story. "Stiffening" of the stomach is an infallible sign of pyloric stenosis. Peristaltic waves may be elicited by applying ether or ice to the epigastrium. The *x*-rays show the outline of the viscus after ingestion of considerable bismuth. (*b*) *Palpation* confirms the above findings and also elicits splashing (*clapotage*) of gas and water in the stomach, a sign on which no great reliance is placed, for splashing occurs in healthy stomachs. The hand may palpate the final gush of gastric contents into the gut. (*c*) *Percussion*—The normal vertical dimension of the

stomach is 4 to 5.5 inches (10 to 14 cm.) and its maximum capaci three pints (1,600 c.c.). Filling the stomach with water and percussin with the patient standing, outlines both the lesser and *greater curvatur* which may even reach to the pubes. Siphoning the water and th distending the stomach with air pumped in, or with carbon dioxi gas, replaces the flatness by tympany. (The carbon dioxide is pr duced by giving sodium bicarbonate ʒj to ij dissolved in water, f lowed by an equal amount of tartaric acid in water.) When doubtf the colon may be distended with gas or fluid. The greatest distenti is at the fundus. With both gas and water in the stomach, the no changes with change of posture, the fluid being in the lower and t gas in the upper parts, as in ascites; the most experienced men a knowledge that they have confused enormously dilated stomachs wi ascites and ovarian cysts and have introduced a trochar; Jadon pu lished a case in which the stomach contained 45 quarts. Leube w the first to palpate the lower end of the stomach by means of a rig sound; this has justly been practiced less frequently of late year the normal distance from the upper teeth to the greater curvature 23.5 inches (60 cm.) which is increased in dilatation to 27.5 inch (70 cm.) or more. (*d*) *Auscultation* elicits the splashing which ma be heard by the patient, and sometimes a fine crepitation or hissin noise as gas is generated, a splashing or dripping sound as the patie drinks, or metallic heart tones. The *x*-rays and gastric illuminatio are less certain and easy methods of diagnosis.

4. **Complications.**—(*a*) *Constipation* is the rule, and the stools ar hard, because less fluid passes the pylorus. (*b*) The *urine* is ofte *alkaline*, its chlorides are lessened, and its amount is reduced to pint or less as a result of the acid vomiting; triple phosphates a common and acetone and diacetic acid are occasional. (*c*) The skin often dry, and (*d*) emaciation is common, even in benign stenosis muscular insufficiency, because food is imperfectly absorbed; some it is lost to the organism by fermentation and tissue-destroying toxi possibly develop. (*e*) Auto-intoxication from fermentation may d velop, causing headache, vertigo, drowsiness, psychoses, polyneurit tetany, slow pulse, and nervous dyspnœa. Tetany (Neumann ar Kussmaul) occurs oftenest in hypersecretion or stenosis from canc or ulcer; its symptoms are considered under TETANY, and it may merely noted here that 73 per cent. of cases die (101 cases combin from Riegel, Albu, Fraenkel-Hochwart, etc.).

Diagnosis.—The etiology, vomiting, impaired motility as shown Leube's test dinner, the visible outlines confirmed by percussion, a the low lesser curvature which is not seen normally, distinguish gastre tasia easily; its cause may be determined with difficulty only. Th stomach may be physiologically large (*megalogastria*), but then the is no motor insufficiency, no vomiting, and no stagnation. *Gastropto* will be considered later. The *degree* of *stasis* is determined by wash ing out the stomach at varying times after the test dinner. In *pylor* *stenosis* there is greater dilatation than in simple motor insufficienc (or atony), and marked peristalsis is always indicative of obstruction. When a diagnosis of pyloric obstruction is established, the benign must be differentiated from the malignant type; *benign pyloric obstruction* runs a more remittent longer course, of over two years', even of fifteen years' duration; HCl and the ferments are usually increased and are always present, sarcinæ are often and lactic acid seldom present; in the writer's experience stenosis due to round ulcer is, in fully half the cases, attended by subacidity or anacidity; while in *cancer* (*v.i.*) the march of symptoms is unfortunately progressive, and cachexia, severe anæmia with leukocytosis, hydrops, pyloric tumor, the Oppler-Boas bacillus, and metastases, develop with relative rapidity. The *spasmodic form* runs an intermittent course. The *prognosis* depends on the etiology and amenability to treatment; spastic (spasmodic) types are curable; relative stenosis may somewhat improve.

Treatment.—1. *Etiological* factors, especially those of atony, should be treated.

2. *General measures*, tending to tone the musculature, are (*a*) *abdominal massage*, which should be practised only when ulceration and inflammation are certainly excluded; (*b*) *drugs*, especially strychnine, which is often combined with atropine and the application of an ice-bag to the epigastrium; ergot and creosote are less valuable; (*c*) *hydrotherapy;* (*d*) *faradization* of the stomach; external application is seemingly as efficacious and certainly less difficult than intragastric application, though opinions vary greatly; and (*e*) *abdominal binders or supports*. These five points in treatment avail only in atony.

3. *Gastric therapy* is indicated. (*a*) *Gastric lavage* removes the fermenting contents, relieves the organ of pressure exerted by accumulated food and fluid, cleans off the mucus, lessens vomiting and auto-intoxication, and may permanently help atony and temporarily mitigate the effects of pyloric stenosis. The stomach should be washed with luke-warm water until it returns clear of food and mucus; salicylic acid and sodium bicarbonate may be added, as in chronic gastritis; the patient soon becomes accustomed to the tube and welcomes the relief afforded; it is generally said that morning lavage is best, but washing before the evening meal is preferable, as the organ then suffers less stagnation through the night; the stomach should rest for a few hours before food is taken. (*b*) *The food* should be well divided, nourishing, and not too voluminous; it should be given with very moderate quantities of water, in small amounts and at intervals of three to four hours. The average diet consists of meat, eggs, leguminous purées, very thoroughly cooked rice, and small amounts of butter; generally speaking, fats and carbohydrates are more prone to fermentation. P. Cohnheim advises an ounce or two of olive oil in pylorospasm and in moderate benign stenosis. A dry diet was early advised by von Swieten, but moderate amounts of fluid aid in propulsion of food into the gut. Of course much depends on the causal disease; a fluid diet is indicated in great pyloric stenosis, and when the hydro-

chloric acid is decreased. Meat should be given if the hydro
acid is normal or increased and the pylorus is not greatly st
Recently there has been a reversion in favor of one or two fai
meals a day, thus giving the stomach ample time to empty its
to rest. Water is not absorbed by the stomach, as proven
Mering, who also found that the absorption from the stomach of
peptone, dextrin, and alcohol, is attended by actual excretion o
into the stomach. Riegel believes a small amount of water i
ficial. In pronounced cases peptonized milk and water are gi
rectum to supplement gastric feeding. In all cases careful indiv
zation is indicated; the exact degree of insufficiency should be
by Leube's meal, *i. e.*, the time required for absorption, and the
should be fed accordingly. (*c*) *Posture*—The seriously affected
should lie on his right side one to two hours after eating, t
relieving pressure upon the greater curvature of the stomac
directing the food toward the pylorus. (*d*) *Gastric medication*
CHRONIC GASTRITIS, ULCER, CANCER, HYPERCHLORHYDRIA). (*e*
sinamin—(See INTESTINAL OBSTRUCTION).

4. *Surgical intervention* is imperative in severe or malignant s
and when medical treatment fails. It embraces (*a*) pylorecto
which Dryendorff collected 188 cases with a 57 per cent. mo
(*b*) pyloroplasty (29 cases with a 21 per cent. mortality), (*c*)
enterostomy (215 cases with a 43 per cent. mortality). In 153
tions for benign stenosis Moynihan had only two deaths. As
in ulcer and cancer (*v. i.*) the earlier recorded mortality has bee
reduced. According to Shaw and Elting, in the pyloric sten
infancy the mortality under medical treatment is 72 per cent.,
surgical 56 per cent. In eleven operations for tetany with
stenosis (Jonnesco), eight cases recovered.

(D). CHANGES IN FORM, SIZE AND LOCATION OF THE STO

Form.—The antrum cardiacum and fore-stomach are œsop
rather than gastric anomalies. The *antrum cardiacum* is a con
bulging of the œsophagus just below the diaphragm, and th
stomach is a congenital dilatation above the diaphragm. The
congenital form of narrowness of the pylorus as well as of hou
stomach.

The acquired *hour-glass deformity* is more common and ma
from spasm (hypertrophic muscle), cancer, ulcer, or adhesions
the middle of the organ, as well as from abdominal tumors, per
tuberculosis, twisting of the stomach or hernia of it through the
colon. Mayo Robson (1904) in 23 cases found ulcer as the caus
tumor in 4 and perigastritis in 4. It may co-exist with pyloric st
Randolph and Thomas observed 2 cases in one family.

Symptoms may be lacking in the lesser degrees of deformity;
higher grades they are those of stenosis. (1) Only a part of

amount of water introduced into the stomach may be recovered (Wölfler's first symptom). (2) After the stomach has been washed till the water returns clear, the fluid may suddenly become clouded (Wölfler's second symptom). (3) The stomach is emptied by the stomach tube and yet splashing is obtained (from the cavity nearest the pylorus); Jaboulay called this paradoxical dilatation. (4) Inflation by carbon dioxide produces distention first of the left and later of the right half of the epigastrium (von Eiselsberg's first symptom). (5) Normally when the stomach is distended and the gas passes through the pylorus into the bowel, there is a sharp gurgle over the pylorus; in the hour-glass stomach the gurgle is heard two to four inches to the left of the median line (von Eiselsberg's second symptom). (6) On gastric distention by carbon dioxide Moynihan found tympany only over the upper part of the stomach and not over its lower half. (7) When the stomach is distended, a furrow may appear (Eichhorst and Schmid-Monard); Moynihan saw the furrow in 10 cases. (8) Filling the stomach with water and illuminating it by a light introduced into the stomach, the cardiac portion appears translucent and the pyloric part dark (Ewald). (9) A rubber balloon (introduced into the stomach and then inflated through the stomach tube) fills or outlines a part only of the stomach (Hemmeter).

Treatment is surgical. Moynihan successfully operated on 23 cases; he enumerates the surgical measures as follows: single or double gastroenterostomy, gastroplasty, gastrogastrostomy, partial gastrectomy, and dilatation of the stenosis.

In *situs viscerum inversus* (see DISEASES OF THE HEART) the pylorus lies on the left side.

Size.—Megalogastria and microgastria are usually accidental findings if not associated with other gastric disease.

Location.—The stomach at birth is vertical, and in adult life is generally held to lie so that three-quarters of it is to the left of the median line. Rosenfeld has recently insisted that the whole stomach is disposed to the left of the mid-line, and that the organ's physiological posture is vertical.

Gastroptosis.—(See ENTEROPTOSIS.)

(E). ROUND ULCER OF THE STOMACH.

Definition.—Ulceration of the stomach is characterized in the majority of cases (*a*) etiologically by its occurrence chiefly in young women or in chloranæmic subjects; (*b*) pathologically by a "punched out" funnel-like ulcer, oftenest near the pylorus, and (*c*) clinically by localized pain and tenderness, hæmatemesis, and hyperacidity.

Frequency.—It is found in 2.5 to 5 per cent. of all autopsies (W. H. Welch, Orth, and von Jaksch). It occurs four times less frequently in America than in Europe.

Etiology.—The causes of ulcer are not exactly known. (*a*) *Age and sex*—Most cases (70 per cent., Lebert) occur between *twenty and* *thirty* years of age, especially in women. Isolated cases occur in very young and in the fœtus; in children E. C. Cutler collected cases; 60 per cent. occurs in women, in whom the disease occurs to four times as frequently as in men. Ulcer develops most freque in men between thirty and fifty years of age; the author has seen cases in men over sixty. (*b*) *Anæmia* and chlorosis are strongly disposing factors. Anæmia plus (*c*) *hyperacidity* are usually hel be important factors; the writer believes Rosenheim and Cohnheim correct in stating that hyperacidity is only a result; 73 per cent Leube's cases of chlorosis showed hyperacidity. (*d*) Changes in *vessels* are of etiological importance. As a normal access to the stom of arterial blood prevents its autodigestion, it is thought by some interruption in the local circulation is the cause of ulceration; Virc believed that embolism was causal, and Klebs held that arterial sp was an etiological factor. Embolism is proven to follow burns. will be noted, however, that ulcer often occurs at an age when arte changes are rare, though syphilis, alcoholism, arteriosclerosis, car and hepatic disease apparently cause some of the cases in later (*e*) *Occupation* is a factor. Many cases occur in servant girls an cooks; (*f*) *trauma* in cooks, shoemakers, and in tailors, is an access factor, for the tasting of hot foods and pressure against the stom promote the development of ulcer. (Some writers hold that there two leading causes for ulcer: anæmia and pressure.) The pressur corsets, the swallowing in certain trades of particles of iron, porcel and glass, and imperfectly masticated or improper food, may be brou under this caption. External trauma is a distinct factor. (*g*) O alleged causes are heredity; vomiting, and even hysteria, have b considered as causal. Letulle and Martin hold that (*h*) *bacte* *necrosis* causes ulcer, which is clearly shown by Dieulafoy's cases pneumococcic hemorrhagic ulceration of the stomach.

Pathology.—Scars indicating healed ulcers are three times as f quent at autopsy as open ulcers. The ulcer is known as *ulcus simp* (to distinguish it from malignant, or syphilitic ulceration), *ul* *rotundum*, as the smaller ones especially are round, *ulcus peptic* and *perforans*. The peptic ulcer occurs almost exclusively in t stomach, lower œsophagus, and upper duodenum. (See DUODEN ULCER.) Its general morphology was first described by Cruveilhi (1829) and Rokitansky (1839); it has a "punched out," clean-c appearance; in contour it is round or oval in small, and irregular large, ulceration; it is usually small, but sometimes invades most the surface of the stomach, and in one case it measured four by seve and a half inches; it is funnel-shaped in old cases; its apex is eccentr and is directed toward the serosa, its sides are terrace-shaped, and floor is usually clean. In 80 per cent. the ulcer is *single*, though a ca with as many as 34 ulcers has been reported. Extension is mo common in acute than in chronic cases and is more toward the pe toneum than laterally. Microscopically there is usually no infiltrati with round cells. Its *localization* is chiefly near the pylorus on t posterior wall or lesser curvature; its *complications*, as hemorrhage and perforation, and its *sequelæ*, as pyloric stenosis and deformations, will be considered under clinical symptoms.

Symptoms.—The *general clinical picture* varies, cases being typical, doubtful, or entirely latent.

In the *typical case* the symptoms develop gradually with a sense of epigastric pressure after eating, which later becomes a more violent, cutting, *localized pain;* this is observed less commonly directly after eating than one-half to one and a half hours thereafter; it lasts throughout the period of digestion and ceases only with passage of the food into the bowel or after vomiting. The appetite is usually good; *hyperacidity* is noted after a test meal or in the vomitus; focal *tenderness* occurs over the epigastrium, and in fully half the cases *bleeding* from the stomach or blood in the stools is observed.

The *typical triad* of cardinal symptoms, taken together or possibly singly, makes the diagnosis most easy; (1) pain with localized tenderness, (2) hæmatemesis, and (3) hyperacidity.

1. Pain.—The most important, characteristic and constant symptom is pain, which is usually paroxysmal, is localized, and occurs during digestion. It is boring or burning, seldom lancinating, and is due to irritation of the gastric nerves aroused by peristalsis, nerve erosion, or hyperacidity. The *diet* affects the pain greatly; milk, for instance, is well tolerated in certain cases, but solid food usually produces excessive pain, and very *hot* or very *cold foods* or beverages almost invariably cause discomfort. This disappears quickly with rectal feeding. The seat of the pain varies in different patients, but is always the same in a given case. In the great majority of cases it is *epigastric* and is very often quite near the ensiform. Ewald and others describe it as characteristically cramp-like in the pyloric region (in the right mammary line under the liver). Sometimes there is wide irradiation toward the sternum, ribs, intercostal spaces, shoulder, scapula, and more infrequently even in the brachial plexus and pulmonary branches of the vagus, resembling that of angina pectoris (Traube) or even producing sympathetic pain (Mueller) in the arms and legs. The pain varies with change of position, depending on the localization of the ulcer, but there is always pain when the patient lies on the left side.

In relation to eating, there is a difference of opinion. Sometimes the pain occurs two to ten minutes after eating, the old "wound pain," supposedly due to contact of food with the eroded nerves in the ulcer. Again, the pain occurs at the height of digestion, *i.e., one-half to one and one-half hours after ingestion of food;* this is caused by the hyperacidity of the gastric juice. Patients fear to eat because of the inevitable pain. Pain may occur after meals for months, and disappear to recur after months. (In some cases of ulcer there is constant flow of gastric juice [*v.* HYPERSECRETION] which may result in two kinds of pain, one of which is present when the stomach is empty and the other at the height of digestion.) Pain may be worst at the time of the menses. Pain from the scars or adhesions of an ulcer is infre-

quent, is rarely cramp-like but usually drawing or vague, and son e
times continuous. In old ulcers pain is atypical or wholly abse t.
The time of development of the pain does not locate the ulcer, thou gh
if it comes on at once it may suggest œsophageal or cardiac ulcer; r,
if it occurs two and a half hours or more after eating, it is suggesti ve
of ulcer of the duodenum.

Tenderness is most often found under the ensiform or near the na el
(if there be gastroptosis). It is usually *circumscribed* and is more oft en
and more advantageously found on the lightest touch, rather than on
deep palpation. A diagnosis from tenderness alone is somewhat unsa fe.
Tenderness in the back (Cruveilhier) suggests ulcer on the poster or
wall of the stomach and is found in 25 to 33 per cent. of cases over an
area of one or two inches to the left of the tenth to twelfth dorsal or
upper lumbar vertebræ. The abdomen, back, and chest, are of en
exquisitely hyperæsthetic. In a few cases pressure is grateful (Osl r).
Palpation must be guarded, for perforation has followed undue pr es-
sure.

2. Hæmatemesis.—The second cardinal symptom is hæmateme is.
Some authors consider it the most important and certain findi g.
Its frequency is variously given at from 25 to even 80 per cent.; he
average figure is probably 50 per cent. Vomiting of blood in ulce is
most frequently from ulcer in the pyloric region (posterior wall
lesser curvature), or, less frequently, from ulceration reaching the li
pancreas, or spleen. Hemorrhage when small may be overloo
(occult hemorrhage), or blood may be found only in the stools; mel
alone is found in 11 per cent. of cases. It is said that the hemorrh
must amount to a pint to turn the stools black. It may be large
sometimes prove fatal; indeed it may be so acute that no bloo
vomited and the stomach and bowels are found to be filled with
massive clot. The usual signs of internal hemorrhage exist, as colla
pallor, moderate rise of temperature, brain anæmia, hæmic m
murs, amaurosis, and rarely, optic atrophy or hemiplegia. If pro
it is voided *bright red*; if slow and moderate the acid turns it d
"*coffee-grounds vomitus.*" If red blood cells cannot be found in
vomit because of complete disintegration of the cells, blood ca
detected by treating it with acetic acid, extracting with ether an
testing with the guaiac and turpentine reaction, when blood
shown (Bial) even if the spectroscope shows nothing. "Occult
rhage" occurs in 25 to 50 per cent. of cases (see HÆMATEME
CANCER); it is not as constantly present as in gastric cancer. J
Steele concludes that the tests for *occult hemorrhage* are
first, to determine the length of the various periods of th
treatment of ulcer. Second, to detect the tendency to blee
the course of gastric ulcer and by appropriate medical a
measures to anticipate and prevent serious hemorrhage.
determine when the medical treatment may be consid
failed and surgical treatment is indicated. Fourth, pe
may prove helpful under certain circumstances in det

velopment of a cancer upon the floor of an ulcer. Blood is found macroscopically in the stomach washings less frequently than is to be inferred from text-book descriptions, and apparently normal vomitus may show blood microscopically or chemically. Profuse hemorrhage usually indicates deep ulceration. Hemorrhage is said to complicate chronic more than acute cases, though this does not correspond to personal experience. The hemorrhage is usually fairly profuse in ulcer; it may be spontaneous during sleep or rest or may result from excitement, physical exertion, or improper diet. In a case of "chronic dyspepsia" with vague symptoms, the writer inflated the stomach with the usual amount of carbon dioxide; the patient collapsed in the office and vomited nearly a quart of blood.

3. Hyperacidity.—This is the third cardinal finding. Though it has often been stated to be a constant finding by Riegel and others, Hemmeter finds it in 90 per cent. and Leube in 50 per cent.:

HCl according to	Normal acidity.	Hypochlorhydria.	Achlorhydria.	Hyperchlorhydria.
Ewald...........	57%	9%	....	34%
Eichhorst.........	36%	34%	23%	7%
Author...........	46%	20%	4%	30%

The variance noted in these figures is due, in the author's opinion, to combining old callous ulcers with the fresh chlorotic ulcers; in old ulcers hyperchlorhydria is present in only 10 per cent., and they explain most of the cases of decreased acidity; in fresh ulcers, hyperacidity occurs in 70 per cent. Hyperchlorhydria is probably due to irritation of the vagus (Pawlow). Complicating cancer, hypersecretion or catarrh may cause subacidity.

Should a test meal be given in cases of ulcer, to determine whether an excess of acid exists? Certainly not, when the diagnosis is determined by the pain, tenderness, and vomiting of blood, or when the ulcer lies at the cardiac end of the stomach. The stomach tube, however, is used by Riegel, Boas, Rosenheim, Fleiner, and others, not only for diagnosis in atypical cases, but also to relieve the more dangerous vomiting and to introduce silver and bismuth into the stomach. Organic acids, as lactic, are absent, the carbohydrates are poorly digested, and the albumins are rapidly converted into albumoses or peptones. The motility of the stomach is normal in one-quarter and slow in three-quarters of the cases. If pyloric obstruction exists the mineral acid is still more increased.

4. Other Symptoms.—*Vomiting*, present in 75 per cent., is a less valuable diagnostic than pain, although they usually occur together. Some cases never vomit, or do so only several hours after meals. It occurs at the height of the pain, one, two or three hours after eating, whereas in dilatation or hypersecretion it occurs much later. Vomiting as well as pain depends directly on the diet, heavy meals increasing and appropriate diet lessening each. It may be uncontrollable. Some degree of parallelism exists between the pain and vomiting, the latter usually relieving the former. The vomitus usually consists of fine particles, is sour, grits the teeth, and shows much acid and no fermentation. Dyspepsia occurs from catarrhal gastritis, dilatation, or other complications. The appetite is very frequently good or even exaggerated. Acid eructations (pyrosis) may cause a burning sensation back of the sternum or between the shoulder blades. If there is much œsophageal burning it indicates incontinence of the cardiac orifice. Diarrhœa is rare. Constipation is due to the anæmia, diet, rest, vomiting, possibly reflex inhibition from the stomach upon the motor nerves of the bowel or to impairment of peristalsis by scars and adhesions. Dysmenorrhœa and amenorrhœa are not infrequent. Blood may be found in the stools. Fever is a complication. The nutrition varies and emaciation may be observed (cachectic type). Anæmia is more common in men and may become profound. The urinary findings are not characteristic. Reduction of the chlorides cannot be used to differentiate from carcinoma. The urine is slightly acid or even alkaline, particularly when the stomach is dilated. The phosphates are increased; with dilatation, indican and ethereal sulphates are increased. Acetonuria and diaceturia sometimes result from inanition and from rectal feeding.

Clinical Forms of Lebert.—(*a*) The *hemorrhagic* form may be either acute or chronic; hemorrhage may be the first symptom, or the last and fatal one. (*b*) In the *perforative* form there may be entire latency of previous symptoms. (*c*) In the *dyspeptic* type, dyspeptic symptoms are most prominent; the evidences of ulcer are absent, or there are atypical symptoms, such as pain, which is not sharp, vomiting, which is infrequent, and excess of hydrochloric acid, but no increase in mucus. (*d*) The *cachectic* form is characterized by old ulcers, by cicatrix producing dilatation, and by chronic hypersecretion; this form may be confused with carcinoma, especially when there is *no* excess of acid. The other types are as follows: (*e*) *gastralgic* or neuralgic form; (*f*) *vomitive* form; (*g*) latent ulcer (scars at autopsy); (*h*) a form with *tumor;* (*i*) *recurrent* type; (*j*) *stenotic* type; (*k*) *carcinomatous* type.

Clinical Course.—The course is usually chronic, for ulceration is frequently long latent before diagnosis is possible, and probably lasts some time after all symptoms have disappeared. Pain usually indicates incomplete cicatrization or certain other sequences, but cessation of pain is no certain criterion of healing. The course may be acute and rapid, especially under appropriate therapy. The course lasts from three to five years (Lebert) or even twenty to thirty-five years; this is usually due to such complications as pyloric obstruction, dilatation, or adhesions. Very large ulcers probably never heal. They frequently *remit* with appropriate therapy, but may recur with the resumption of the old diet. It is questionable when they can be considered cured: certainly only when all symptoms have been absent for six months.

Localization.—This can rarely be determined accurately (Riegel and Leube). According to Gerhardt, tenderness and tumor are indicative of ulcer on the anterior wall, but hemorrhage and dorsal pain suggest posterior involvement. Pain in a lateral decubitus suggests localization in the fundus or pylorus; dilatation, in the pylorus or duodenum; pain under the ensiform during the act of deglutition suggests cardiac ulcer and contra-indicates the use of the sound. Ulcers are palpable in only 0.1 per cent. of cases (Leube). The percentage of involvement of the cardia is 6.3; of the fundus, 3.7; of the lesser curvature, 36.3; of the posterior wall, 29.6; of the pylorus, 12; of the anterior wall, 8.7. "In over 80 per cent. of the cases it occurs in a relatively small area about the pylorus, lesser curvature, and posterior wall."

Complications and Sequelæ.—(*a*) *Perforation* (6 per cent.) may occur into the peritoneum, usually with symptoms of collapse, peritonitis, and cessation of vomiting (Traube), though Brunner finds vomiting in 66 per cent. and Gross in 33 per cent. of such cases. Perforation may be prevented by the left lobe of the liver, pancreas, or spleen, or by omental, pericardial, and pleural adhesions. The danger of perforation in ulcer of the anterior wall is seven times as great as in that of other locations. Perforation of a latent ulcer may be confused with rupturing appendicitis or salpingitis. Perforation may occur into the heart, colon, gall-bladder, portal vein, pericardium, chest wall, or abdominal wall; or it may reach the space between the liver or spleen and the diaphragm, producing pneumothorax subphrenicus, which is suggested by pleurisy, epigastric and hypochondriac pain, stiffness in the back, painful eructations and swallowing, singultus, dorsal decubitus, or local œdema. (*b*) *Simple peritonitis* (perigastritis) may cause a friction murmur over the stomach. (*c*) According to Rosenheim 6 per cent., Liebert 9 per cent., or Zenger, the majority of cases of *cancer* develop from ulcer. Two years ago the author saw with Dr. Harpole a case of carcinoma developing in an ulcer scar in a girl twenty-six years old. The typical symptoms of ulcer are followed by loss of weight, and persistence of symptoms, usually by gradual lessening of hydrochloric acid and other evidences of carcinoma. (*d*) *Adhesions* about the stomach may interfere with its motility, produce deformation, such as the hour-glass stomach or diverticulum, neuralgia which outlasts the ulcer, and obstruction of either orifice of the stomach. Dilatation of the stomach usually, but not always, indicates pyloric stenosis; it may also indicate cicatrizing ulcer or result from spasm of the pylorus. Scars may, in rare cases, produce intestinal obstruction. Troublesome adhesions may be finally so stretched as to relieve all symptoms.

Diagnosis.—The diagnosis is determined by (*a*) the etiological factors, (*b*) the cardinal symptoms, and (*c*) the various clinical types enumerated. In general the diagnosis is easy in the young, but may be difficult in anæmic or chlorotic individuals.

Any two of the cardinal features confirm the diagnosis. *Pain* is often the only symptom; if it is atypical, hyperacidity may indicate ulcer, though the latter is often observed without ulcer; Leube proposes for such cases the therapeutic test, although both cases ma respond to the treatment. According to Riegel the chief difference i that pain in simple acidity is less regular than in ulcer and that localized tenderness is absent, though he admits that these criteria ma fail. Some cases of seemingly primary hyperchlorhydria are found t be ulcers. Where pain alone exists one must always remember th gastric crises of tabes. Ewald has called attention to the pain of smal hernias in the *linea alba.* Pain may cause confusion when it occur in the transverse colon, which lies in close apposition to the stomach but it usually ceases on evacuation of the bowels. Without carefu examination movable kidney may be confused with ulcer. The pai in *gall-stones* is paroxysmal, but more irregular, and is independent o eating. Icterus in gall-stones is not common. The gall-bladder i tender, and may be distended and palpable. The paroxysm in gall-stones is often attended by fever, which is not present in ulcer, and b a chill. The paroxysm is longer. The liver may be large, especiall the right lobe. The pain more often radiates to the right side an to the shoulder, and the vomiting is not that of ulcer, though hyperacidity and gastric dilatation may follow cholelithiasis. Chroni poisoning, as by arsenic, may be suspected, as in the case of th Duchess of Orleans until Littré made the diagnosis of ulcer.

Hemorrhagic erosions and fissures of the stomach present no constant clinical picture. After eating there is a sense of burning over th entire stomach, which is not altered by change of position. Th chemism and tender points of ulcer are lacking, though Cohnheim usually finds hyperchlorhydria; he also believes that solid food relieve the pain. Shreds of tissue are always found, according to Pariser, which cease with cessation of pain. Fatal hemorrhage is recorded.

In *duodenal ulcer* (as in inflammation, new growths of the liver, perihepatitis, cysts, and chronic pancreatitis), hyperacidity is absent, although in some instances of duodenal ulceration hyperacidity has been observed. Duodenal ulcer occurs mostly in men, in alcoholics, and after burns. Pain is often absent in duodenal ulcer, but whe present occurs later than in gastric ulcer and in the parasternal line Vomiting gives less relief, and the vomitus is usually alkaline, contain bile, and may digest fibrin (Boas). Icterus is not uncommon, thoug not constant enough for diagnostic reliance. Hemorrhage into th bowels is more common than in ulcer. Its sudden appearance wit recurrence and profound anæmia is most suggestive.

Pyloric spasm, considered so characteristic of ulcer by most writer may also occur in myocarditis, aneurysm, coronary atheroma, tæni ascaris, the spastic form of intestinal constipation, or chronic nic tinism (Ageron).

Differentiation from *carcinoma* is not very often necessary, thou in middle-aged patients confusion with ulcer may occur. In Krau clinic, the author saw two cases; one was an old man with cachex pyloric tumor, and decreased hydrochloric acid, which was diagnos cated cancer; the other was a young girl with vomiting of blood, in whom the diagnosis of ulcer was made. The post-mortem examination showed that the old man had a round ulcer and the girl had a cancer. The author has seen five cases of carcinoma under the age of thirty. The table on page 626 considers differentiation from cancer, gastritis, gastralgia, and hyperchlorhydria.

Prognosis.—Though ulcer occurs most frequently between the ages of twenty and forty, it is most fatal between forty and sixty. It is generally stated that the mortality ranges between 6 or 8 and 10 or 13 per cent. of the cases, of which about 4 per cent. is due to hemorrhage and 6 per cent. to perforation. Débove and Redmond give the high death-rate of 50 per cent.

	Recovery.	Death.	Improvement.	No Improvement.
Eichhorst..........	85%	8%	4%	3%
Leube..............	74%	2.4%	22%	1.6%
Köhler............	61%	6%	30%	3%

Very deep and old ulcers rarely heal. The deeper the ulcer the greater the danger of hemorrhage and perforation. Ulcers with coincident hypersecretion give a relatively poor prognosis. Carcinoma must be considered in cases which do not respond to treatment. One of the author's cases died from exhaustion, although the autopsy showed that there had been perfect healing of the ulcer. In over one thousand cases the deaths from hemorrhage and perforation (each 1 per cent.) are about equally divided (Leube).

Treatment.—1. **Prophylaxis.**—Prophylaxis concerns care of the stomach in anæmia, and chlorosis, and with a history of ulcer, care in the diet. The mouth should be kept clean to avoid parotitis.

2. **Absolute Rest.**—Absolute rest in bed for a month or more was first recommended by Cruveilhier, in addition to which Leube recommends the frequent application of hot fomentations to the epigastrium, which relieve pain within a few days. Women should remain in bed during menstruation, even after recovery is seemingly complete.

3. **Absolute Rest of the Stomach.**—Though opinions differ somewhat, it is true that distention of the stomach by even small quantities of food or water excites peristalsis and secretion, distends the ulcer, and prevents granulation. Rest permits the stomach to contract and allows the edges of the ulcer to come more rapidly and completely in apposition. The more absolute the rest of the stomach the more rapidly the ulcer granulates and cicatrizes. The time for complete granulation varies with each case, and chronic ulcers show little or no tendency to cicatrize. An ice-bag promotes contraction of the stomach. *Rectal feeding* is imperative, and the usual rules should be followed; peptonized milk, grape sugar, baby foods, meat juice, and eggs, should be given, in amounts not to exceed a total of eight ounces, and at eight-hour intervals; they should be injected slowly, with the patient on his left side, and should be given warm, which precautions obviate

	CANCER.	ULCER.	CHRONIC GASTRITIS.	GASTRALGIA.	HYPERCHLORHYDRIA.
Etiology.	75% over 40 years.	75% under 40 years—2–4 times oftener in women.	Males.	Women.	Males.
Tongue. *Appetite.*	Heavily coated. Usually anorexia.	Usually clean and red. Good: only fears to eat.	Gray-white fur. Poor.	Normal. Variable. Sometimes perverted.	Clean. Good, often increased.
Pyrosis.	Often pyrosis.	Water brash.	Frequent.	None.	Present.
Belching.	Usually fœtid belching.	Usually absent.	Copious.	Variable.	Usual.
Pain.	The pain is less intense, but more constant; seldom free intermissions, during which no distress is felt in gastric region. Pain not increased at the height of digestion. Tenderness less frequent and intense.	Intense; appears shortly after meals; grows severer on pressure; disappears at the end of digestive period; perfectly free periods more frequent; paroxysmal.	May be present but not pronounced and rarely actual pain but tenderness of diffuse character. Lancinating pains in atrophic gastritis only.	The pain appears without regularity, and not dependent upon meals; may be relieved by pressure; intervals of several days free from pain. Tenderness rare; conforms to outlines of the stomach. Electrical test (Leube) during digestion; if pain decreases, it is gastralgia; if it does not, may be ulcer or gastralgia.	The pain appears about one or three hours after meals, disappears after eating, especially meat, milk or eggs, or after the administration of bicarbonate of sodium. Pain less regular.
Vomiting.	Usual. Hours after eating or once a day or alternate days, etc. Copious, coarse mucus, fermented.	Frequent—at height of digestion—smaller amount, finely, homogeneous, no mucus, acid.	Frequent; mucus.	Rare; no mucus.	Much less frequent, then acid and no mucus.
Hæmatemesis.	Frequent — small, dark, like coffee grounds, decomposed, usually late in disease.	Frequent—copious, bright; may be early symptom.	None.	None.	None.
Test Meal.	Early reduction HCl and later, total absence; ferments decreased or absent; lactic acid present; impaired motility and absorption; abundant mucus, Oppler-Boas bacillus.—yeast—possibly tumor shreds; coarse undigested particles of food.	Hyperchlorhydria often; ferments normal or increased; no lactic nor Oppler-Boas bacilli; food well digested. No mucus.	Decreased HCl (rarely increased, gastritis acida); later may be achylia.	Normal or variable. If HCl is decreased, the ferments are normal.	As in ulcer.
Tumor.	In 80 per cent.	In but one pro mille (from pyloric spasm, hypertrophy, cicatrix, etc.)	None.	None.	None (very rarely from pyloric spasm)
Collateral Symptoms.	Cachexia—œdema.	Absent.	Absent.	Absent. Nervous stigmata often.	Absent.
Course.	Part of a year.	Longer; helped by treatment—aggravated by arsenic.	Chronic. —ditto.	Arsenic helps.	Like ulcer.

excessive peristalsis. A piston syringe should never be used; a fountain syringe with a soft tube is always used. Nutrient rectal enemata can be retained almost invariably if the technique is carefully carried out. Wine or spirits almost always irritate the bowel. Rectal tenesmus is obviated by administering a short time before the enema, an opium suppository of one-half grain of the extract. Though rectal feeding gives the patient but one-quarter of the food necessary to maintain metabolism, it is sufficient, because the patient is absolutely quiet. Thirst should be relieved by allowing the patient to rinse his mouth out, but he should swallow nothing, either water or melted ice, and by giving two administrations daily of warm saline enemata.

4. **Pain.**—Rest of the stomach relieves the pain in almost every case; the use of narcotics should be limited as much as possible; small doses of opium or morphine by rectum, or atropine, may be given. There is, of course, danger that the patient may develop the opium habit.

5. **Superacidity.**—Though many physicians would begin the treatment of superacidity at once, the author is convinced it should be initiated ten days to two weeks after commencement of treatment, during which time the stomach has had absolute rest. Then Carlsbad water should be given; it should be lukewarm, because hot water opens the ulcer; it should be given in small quantities and taken in sips. It was formerly held that alkalies were incompatible with digestion, but Ziemssen, Leube, and others, have disproven this point. Carlsbad water consists of sodium sulphate 50 parts, sodium bicarbonate 6 parts, and sodium chloride 3 parts; it possesses no intrinsic value other than its alkalinity; it neutralizes the acid, thereby tending to check the ulceration, to aid intestinal digestion, and to cleanse the stomach. Ziemssen administered half an ounce in a pint of water; this was taken in quarters every ten minutes, the last dose being given one-half hour before meals. Vichy water may be used. Roberts prescribed a lozenge composed of magnesium carbonate, grains 3½; chalk, grains 2½; sodium chloride grain 1, which, dissolved slowly in the mouth, increased the salivary flow and neutralized the excess of acid.

6. **Diet.**—After ten days of rectal feeding, five or six meals daily of predigested milk with lime water should be given in small quantities, that the edge of the ulcer may not be distended. The saccharate of lime is five times as rich in lime as is lime water. Cooked milk is more easily digested and curds less. The casein both engages and neutralizes the acid. If peptonized milk is distasteful it may be administered as a thin gruel; this is especially indicated when the stomach is very irritable and painful. Potato purée, arrow-root, wheat-meal, baby foods, meat juice, and buttermilk, may be given; soups with meal and alkalies which precipitate the casein in finer form, Mosquera's beef meal, beef peptone jelly, and ice cream, may also be given; but it should be remembered that the milk diet recommended by Cruveilhier is most efficacious. After the patient has been on the above mentioned diet for ten days, soups without fat, rice, sago, soft-boiled eggs, calves'

brains, and chicken, may be administered. Raw eggs are diges with difficulty, but slightly boiled or poached eggs are less indigesti and may be given with bread-crumbs from the inside of the lo Custards may be given. Tea and coffee should be forbidden. Su was considered deleterious by Cruveilhier, but Strauss has found t it lessens the amount of acid and he recommends that the pati should have a dram of a 20 per cent. solution. After fifteen d Leube permits grated beef, and after about another week, ten roast beef, chicken, and pigeon, to which others would add lamb, m aroni, thymus gland, and bread. After the fifth week, roast veal a soufflés may be given. Cohnheim gives olive-oil freely at all tim he finds that it relieves spasm and inhibits hyperchlorhydria.

7. **The Bowels.**—Plain enemata may be administered after the fi or second week.

8. **Drugs.**—Much has been written in regard to the administrati of bismuth and silver. *Bismuth* may relieve pain and is taken m advantageously in solution; the author does not believe that bism has any curative value; its use is less efficacious than absol and prolonged rest of the stomach. Fleiner, Kussmaul, and othe administer bismuth by means of the stomach tube. The objectio to large doses are that they may accumulate in the ulcer, the dr may cause pain, and is expensive. Chalk and talcum are no mo efficacious than bismuth. *Silver* has been used, and some write even wash the stomach of the patient before meals with a solution 1 to 500 or to 1,000 (Reichmann). The silver is readily precipitate by the acid or the albuminoids, and unless given in capsule or pill by the stomach tube may produce nausea, burning in the mout diarrhœa, and even argyria. Iodide is indicated in syphilitics, whom it is well tolerated.

9. **Symptomatic Treatment.**—*Pain* is best relieved by rectal alime tation, as is vomiting, which sometimes may necessitate the admin tration of bismuth, minute doses of carbolic acid in mint water, the use of the ice-bag or sinapisms. *Hemorrhage* is treated by absol rest of the stomach, intestine, mind, and body; morphine quiets pe stalsis and mental excitement. No styptics or astringents should given and ergot is contra-indicated. Syncope promotes spontane coagulation, and stimulation should be avoided as long as possib In severe hemorrhages the use of saline transfusions should be co sidered, but more than one pint should seldom be given, for death h occurred from reaction and renewal of hemorrhage. Gelatine inj tions may be indicated for severe hemorrhages.

10. **Surgical Treatment.**—Seventy-five to 96 per cent. of cases, a cording to Leube, either heal absolutely or improve greatly und proper medical treatment; this is especially true of fresh ulcers. Leu in his widely quoted article gives the following indications for surgic interference: (*a*) When the *hemorrhage* is small and repeated, a persists in spite of rectal nutrition; in single large hemorrhages oper tion is contra-indicated. Moynihan holds a similar view, and lost b

1 out of 70 cases of chronic hemorrhages, which result he attributes to his treating the ulcer instead of performing simple gastro-enterostomy. Savariand estimates the operative mortality at 50 and Rodman at 33 per cent. Tuffier (1905) finds a mortality of 36 per cent. (56 cases). (*b*) *Gastro-enterostomy* is indicated for mechanical difficulties, pyloric obstruction, persistent pyloric spasm, and pain; obstruction at the cardia, or hour-glass stomach, also requires surgical treatment. Thirty-one cases of jejunal ulcer have followed operation, since Braun's first case; it develops usually after a year or more (ten days to seven years). Mickulicz in operations for benign gastric disease finds a 7.5 per cent. mortality in his series of 1897–1899. Moynihan in 15 operations for hour-glass stomach had a mortality of 20 per cent., and in 100 cases of gastro-enterostomy a mortality of 2 per cent. (*c*) *Perigastric adhesions* or abscess, and collections of pus beneath the diaphragm, necessitate operation. (*d*) In cases of *perforation* the outlook is fairly good if early operation is undertaken and is performed at once when the initial shock has passed. The best figures are those of English and Graffe with 50 per cent., of recoveries; of Ferrier and Hartmann, with 47.5 per cent.; and, between 1900 and 1904, of H. A. Hare, with 37 per cent. Donati, of Florence, collected in 1905, 1,041 cases of operation on ulcer cases; the mortality in perforation was 48 per cent.; in gastro-enterostomy the mortality with the use of Murphy's button was 7.5 per cent.; with suture anastomosis, 14.5 per cent.

Surgical interference is necessary in a limited number of cases. Sahli is opposed to excessive surgical interference, and argues that (i) reports only show that many of the patients stand an operation; (ii) gastro-enterostomy is indicated only for anatomical stenosis, too many cases of spasmodic stenosis are called anatomical, and at operation too many contracted pylori are called cicatricial. (iii) Gastro-enterostomy is not proven beneficial for hemorrhage, diet being the important indication. Cases operated on often die from hemorrhage after the operation. (iv) No proof exists that ulcers heal better with gastro-enterostomy than with medical treatment. (v) Excision of the ulcer is too radical. The writer's experience justifies the conclusion that (*i*) fresh (chlorotic) ulcers always, and (*ii*) chronic ulcers in half the instances, belong to internal medicine; (*iii*) half the inveterate cases respond to medical treatment; (*iv*) most of the cases "chronic dyspepsia," and almost none of the cases with hemorrhage, call for surgical interference.

(F). CANCER OF THE STOMACH.

Cancer of the stomach is more frequent than cancer of any other organ; it constitutes 33 to 50 per cent. of all cancers, and is found in 2 per cent. of autopsies.

Etiology.—This is as obscure as in other carcinomata. (*a*) *Age*—75 to 80 per cent. of the cases occur in subjects after the fortieth year. According to Lebert 1 per cent. occurs in persons under thirty years; 18 per cent. in those between thirty and forty; 61 per cent in those between forty and sixty, and 20 per cent. in those over sixt years of age. Welch's figures give 2 per cent. under thirty, 13 per cent between thirty and forty, 54 per cent. between forty and sixty, and 2 per cent. over sixty years of age. Every clinician has observed number of cases in persons under thirty years. The author has see five, which leads him to believe that many more cases occur than i indicated by Débove's collection of 32 cases under the thirtieth year Rare congenital cases are recorded (Wilkinson, Débove, Collingsworth). Of late years cancer is apparently becoming more frequen and more common in relatively young persons. (*b*) *Sex*—It is slightl more frequent in males (6 to 5). (*c*) *Heredity* is noted as an etiologica factor in 6 to 17 per cent., and in some families the number of case of carcinoma is astonishing. (*d*) Ulcer of the stomach is an anteceden in at least 6 per cent.; C. Graham found ulcer in 60 per cent. of hi cases. (*e*) Other causes are less definite. Trauma, external pressure as in shoemakers, or internal ulceration by caustics, is noted in som cases. Pulmonary tuberculosis and alcoholism are slightly predisposing factors. Cancer is almost unknown in certain tropical an subtropical localities, and in temperate zones a topical disposition i occasionally apparent.

Pathology.—(*a*) In *origin* it is an abnormal growth of the glandula epithelium, as was first proven by Waldeyer, which unlike mere adenomatous or papillomatous proliferations, extends beyond the mucosa into the other coats of the stomach and very often into other organ and tissues. Cancer may develop in the seemingly normal mucosa, i an atrophied mucosa, or in open or cicatrized ulcers. (*b*) It is almost *always primary*, is very rarely metastatic, or extends to the stomach by contiguity; such secondary growths are usually from mammar or pancreatic cancer; Welch has collected 37 cases. It is also almos invariably single.

Histology and Morphology.—A tumor, in the clinical sense, usuall develops, although in some cases the cancer is infiltrative and fl (*carcinoma planum*). There are three main types: (*a*) *Carcinom fibrosum* (scirrhus), the most frequent type, contains much poor vascularized stroma and relatively little carcinomatous tissue; it hard, and, leading to much induration, is prone to stenose the pyloru where it is especially frequent, or to stenose the cardia; if it is diffu it causes the stomach to shrink (carcinoma atrophicans); its onset insidious; it grows slowly and ulcerates less than other types. (*Carcinoma medullare* is next in frequency; the cell elements prepo derate over the stroma; there is much soft, vascular tissue, and t knife scrapes out much "cancer juice"; it may form cauliflower-li exuberances; early extensive degeneration, ulceration, and metastase are common; this type includes the cylindrical celled adenocarci oma, epithelioma, or the very vascular fungus forms. (*c*) *Carcino gelatinosum* (colloides) is least frequent; it produces nodul tumors or diffuse infiltrations, both of which contain colloid m terial; it inclines rather to extension by contiguity than to metastatic deposits.

The *gastric mucosa* shows focal wasting of the peptic- and acid-secreting epithelium and substitution by cylindrical epithelium, round cells, and connective tissue. This *secondary atrophic gastritis* explains the chemism of gastric cancer—*viz.*, diminution in the secretion of acid, rennet, and pepsin; in some cases it has regressed after surgical extirpation of the tumor, the secretion of acid and pepsin returning.

Localization.—Cancer occurs (*a*) in the pylorus in about 60 per cent., (*b*) in the lesser curvature in nearly 20 per cent., and (*c*) in the cardia in 10 per cent. of the cases; in round numbers, therefore, about 90 per cent. of cancer occurs in these sites; this is explained by their greater exposure to mechanical insult, their relative fixation, and the greater muscular activity of the pylorus and cardia; many tumors, apparently pyloric in origin, really begin in the lesser curvature, whence, in other words, a palpable pyloric tumor often means one of considerable extension; this is an important surgical point.

Complications.—(*a*) *Ulceration*, which is really more a part than a complication of cancer, results from inadequate vascularization; it is usually central and does not necessarily depend on the size of the tumor. In some cases the tumor may ulcerate almost completely, and Ziegler has observed total disappearance, though metastases declared the nature of the scar. (*b*) *Hemorrhage* or slow seeping of blood is caused by ulceration, as is also (*c*) *perforation* (3 per cent.) into the peritoneum, or, by adhesions, into adjacent abdominal organs or vessels, into the lung, pleura, or pericardium, or into the abdominal wall, of which latter 22 cases are recorded; perforation is not more common because adhesions often prevent perforation. (*d*) *Stenosis* of the cardia or pylorus is common, sometimes causing hypertrophy of the muscle fibers, which appear on section of the stomach as red lines. (*e*) *Dilatation* of the stomach follows pyloric obstruction, atrophy is observed in cardiac localization or in carcinoma atrophicans (diffuse cancer), and hour-glass deformation may result when an annular scirrhus develops in the mid-stomach. (*f*) *Local extension* by contiguity occurs by the submucous lymphatics, and there is early involvement of the lymph nodes along the lesser curvature; other lymphatics are soon invaded; the peritoneum is quite often involved, and less often there is extension into the portal vein. (*g*) *Metastases* occur in 50 per cent.; they are distributed as follows: the liver is involved in 28 per cent., the peritoneum in 37 per cent., the lymphatics in 23 per cent., the pancreas in 20 per cent., and the lungs in 8 per cent.

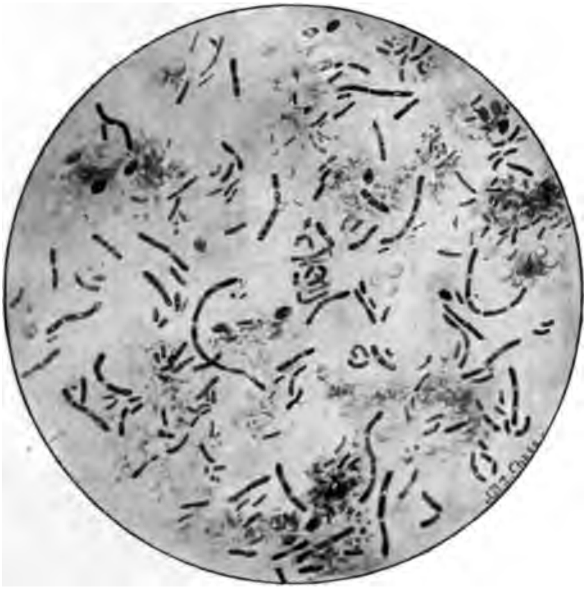
Symptoms.—**General Clinical Picture.**—Anorexia, distaste for meats, mild dyspepsia, gastric oppression, and eructations after eating, usually mark the insidious onset. Much less frequently the disease begins acutely or follows closely on symptoms of gastric ulcer. Vomiting occurs and then symptoms of gastric distress after eating solid food, and of mechanical or secretory insufficiency develop. There is gradual loss of weight together with loss of strength and endurance. Examination reveals anæmia, cachexia, emaciation, lax skin, flabby muscles, and often a tumor near the pylorus. A test meal shows absence of free hydrochloric acid and imperfectly digested food. The symptoms progress steadily, or remissions occur, especially early in the disease. The ankles become puffy and the prostration is profound. Ulceration may relieve pyloric or cardiac stenosis, but gradually within a year after the first marked symptoms death results, from the exhaustion due to pain or vomiting, from local complications, or intercurrent disease.

1. General Digestive Symptoms.—(*a*) The *tongue* is almost always heavily coated, not being cleaned by mastication, and there is generally a bad taste in the mouth. The appetite is decreased or absent in 85 per cent. and there is a special distaste for meat and fats; the appetite is very rarely excessive; a normal appetite may be retained when the motor power is good and cancer develops on an ulcer. (*b*) *Pain* is present in 90 per cent. It is usually diffuse, dull, oppressive, and less severe than in ulcer. It is increased several hours after eating, or in the night, but is not wholly relieved by vomiting. Localized pain and tenderness are suggestive of circumscribed peritonitis. Pain may be reflected up the sternum, between the shoulders, and to the acromion. (*c*) *Vomiting* (in 85 per cent.) is an obstinate and rather advanced symptom, resulting chiefly from pyloric stenosis, with which it runs more or less parallel, and also from coincident gastritis. It appears late in the day or in the night, and may occur only on alternate days. There may be no vomiting in cancer of the curvatures, in diffuse cancerous infiltration involving the muscularis, or in pyloric cancer when ulceration is marked. The vomitus contains much mucus, poorly digested food, little or no free hydrochloric acid, much lactic acid, and often blood, which may be obvious or occult. Fæcal vomiting usually but not always indicates gastrocolic fistula (of which 70 cases are recorded). (*d*) *Hæmatemesis*, present in over 50 per cent. of cases, is due to parenchymatous oozing, and is therefore moderate in amount; it is dark in color, as the blood is retained long in the stomach and is converted from oxyhæmoglobin to hæmatin. It seldom reaches the stools because of pyloric stenosis. The author has seen four cases with fatal, profuse, bright hæmatemesis. (*e*) *Constipation* is present in 75 per cent., diarrhœa in 20 per cent., and normal movements in 5 per cent. of the cases (Hemmeter); in two cases the writer has seen ichorous stools resulting from necrosis of the lower jejunum and of the ileum.

2. Special Gastric Symptoms.—(*a*) *Tumor* is felt in 80 per cent. and seen in 40 per cent. of the cases, but is not an early symptom. It is most often missed in persons with a wide thorax and rigid abdominal muscles. In most instances it occupies the *right epigastric area*, as a round, ovoid, irregular mass; this is best determined when the knees are drawn up and the mouth is open, after the bowels have been flushed out and the stomach distended; in some cases the tumor may be felt lower or even on the left side. Examination should be made with the patient in several postures, as on the side, on the face, genupectoral position, etc. Anæsthesia may facilitate detection. The tumor may be palpable one day and not to be found on the next. *Distention* of the stomach *by gas* (von Frerichs) brings forward tumors of the anterior wall and greater curvature, obliterates tumors of the posterior wall and lesser curvature, and pushes downward and to the right tumors of the pylorus; motility on distention excludes adhesions. If the pylorus is incontinent from ulceration or infiltration, the gas escapes rapidly into the gut (Ebstein). The *x*-rays outline the stomach after administration of bismuth. With or without distention, a *visible peristaltic wave* suggests pyloric obstruction. Percussion is wholly subordinate to palpation and inspection, but auscultation occasionally elicits friction or a stenotic murmur over the abdominal aorta. *Respiratory excursion,* though generally stated to be absent if the stomach is not adherent to the liver, is often present, though less in degree, than the excursion of the liver or spleen; if adherent to the omentum, there is no excursion. The possibility of *expiratory fixation* is more important; if nonadherent tumors of the stomach, intestine, and omentum, are grasped at the height of inspiration, their ascent upward during expiration can be prevented (Minkowski's expiratory fixation); on the other hand, tumors of the liver and spleen, or tumors of the stomach, if adherent to the liver, must ascend during expiration; *i.e.,* they cannot be retained where they were grasped and felt during inspiration. (*b*) *Chemism*—(i) Bird (1842) found the hydrochloric acid reduced, but Van der Velden (1879) showed the especial frequency of achlorhydria in gastric carcinoma. Absence or decrease of hydrochloric acid is not pathognomonic, as it is also absent, permanently or temporarily, in numerous other conditions, as nervous dyspepsia, fevers, cardiac, renal or hepatic disease, pernicious anæmia, cachectic states, gastric atrophy, amyloid degeneration, and toxic gastritis. Benjamin Moore states that the hydrochloric acid is absent in 66 per cent. and decreased in 33 per cent. of carcinoma involving other organs than the stomach; the writer has repeatedly seen cases of rectal cancer in which digestive symptoms and achlorhydria have led to an erroneous diagnosis of gastric cancer. In cancer it is, as stated, due to the secondary *atrophic gastritis.* After administration of an Ewald test breakfast of a slice of stale bread and a cup of weak tea without cream or sugar, the food is withdrawn in an hour and tested as described under chronic gastritis. If there is no stagnation the total acidity ranges from 6 to 8; in stagnation it is higher from lactic and other acids. The absence of free hydrochloric acid is often observed early, but is especially marked in the advanced stages. In very exceptional cases free acid may persist, both in those cases which develop from peptic ulcer and in those without this antecedent. (ii) The *pepsin* and *rennet* are decreased in 66 per cent. of cases (*v.* Chronic Gastritis). (iii) *Lactic acid,* which is always pathological in appreciable quantities, is present in 70 to 80 per cent. of cases, and though present in other conditions, as in atony, gastritis, and pyloric obstruction, 85 to 90 per cent. of the cases in which it is found are gastric cancer. Boas, who developed this topic, considers it a very early finding; while others, as Riegel, hold that it does not develop until later. The factors involved in its formation are somewhat disputed, but depend to a great degree on (α) the reduced hydrochloric acid; (β) motor insufficiency, stagnation, pyloric obstruction; (γ) slow absorption; in the normal stomach it is absorbed as fast as formed (Sticker); (δ) probably also poor digestion of proteids. As a test meal Boas employs 1.5 per cent. oatmeal to 100 parts of water; the Uffelmann method is employed as modified by Strauss for phosphates, alcohol, and sugar also give the reaction; 5 c.c. of the gastric filtrate are added to 25 c.c. of ether and shaken in a burette, which is provided with a cock at its lowest part; the lower 25 c.c. are drained away and 25 c.c. of distilled water are added to what remains (the etherial extract) in the burette. Two drops of a 10 per cent. solution of ferric chloride and 20 c.c. of water are added; the blue solution turns to canary yellow if lactic acid is present. (*c*) *Microscopically*, the long, thread-like *Oppler-Boas bacilli* (Plate XII) are found, which are probably one, though not the only, source of lactic acid formation; the bacilli stain brown with the Gram stain and are called Gram-positive. They may be confused with other morphologically similar organisms, such as the Leptothrix buccalis, which however stains blue by Gram. The Oppler-Boas bacilli are also found in the fæces in gastric cancer; the bacilli point very strongly to the existence of gastric cancer, but may rarely occur, as in the writer's experience, in gastric ulcer and atrophic gastritis (with hypertrophy of the pyloric musculature). Small particles of tumor are seen in rare cases, showing carcinoma structure and irregular mitoses. Sarcinæ are rarely observed. *Occult hemorrhage* (*v.* HÆMATEMESIS), *i. e.*, blood which can be seen only with the microscope or detected chemically or spectroscopically, has the same significance as in gastric ulcer. (*d*) *Motility*—if the stomach is washed out four to six hours or more after eating, there are evidences of stagnation in the undigested food, particularly in pyloric localization; disturbed motility may appear very early in the disease. The stomach is usually much dilated in patients whose appetites persist and little dilated in those with anorexia. If the stomach is empty after six hours, good motility alone is proven, not good digestion. Every case of stagnation should arouse suspicion of carcinoma.

3. **Other Symptoms and Complications.**—(*a*) *Cachexia* results from malnutrition but is something more than malnutrition; it is probably the result of toxins which break down the proteids. The *body weight* is usually greatly *reduced;* in exceptional cases it may temporarily increase, as in cases of pyloric stenosis; it may increase from hope, as in a case of Keen and Stewart, who gained 70 pounds after operation; or when the intestine digests what the stomach could not. The skin itches and is *anæmic* or clay-colored, somewhat resembling the tint it assumes in pernicious anæmia, though distinguished (i) by its later appearance, (ii) by its association with loss of weight, (iii) by the red cells being seldom lower than two million and the hæmoglobin seldom

PLATE XII.



Oppler-Boas Bacillus in Vomitus. (Musser.)

0 per cent., though an apparently good count sometimes results
ickening of the blood which is due to lack of absorption ; and
the leukocytosis (see PERNICIOUS ANÆMIA). Schneyer has
ed the *absence* of the *digestive leukocytosis;* it occurs in about
cases. As in nephritis or chlorosis (rubra), the face may not
e real anæmia. The wrinkling and wasting of the hands may
nore significant. (*b*) The *urine* is decreased from poor absorp-
d vomiting, the physiological change in the acidity-curve after
is absent, the urea is increased from increased toxæmic catabo-
buminuria is present in 35 per cent. of cases, and indicanuria
mon. (*c*) Several *toxæmic symptoms* may occur. (i) *Fever*
, irregular or intermittent type results from the cancer products,
eptic absorption through the ulcer, or from complications as
or peritonitis. (ii) A coma closely resembling *coma diabeticum*
ult from the cancer toxins or the breaking down of the proteids;
ria has been noted. (iii) Phlebitis and the less common multiple
, tetany, and amaurosis, are probably toxæmic. (*d*) *Metas-*
extension by contiguity may occur. Enlargement of the supra-
lar glands may result from any abdominal carcinoma, but 50
t. are due to cancer of the stomach (Troisier). Liver deposits
pear before any suggestive stomach symptoms. Icterus occurs
er cent. (For other metastases *v.* PATHOLOGY.) (*e*) *Ascites*
(i) from the cachexia and, like the swelling of the ankles, appears
the malady. (ii) Some cases are due to carcinomatous peri-
the fluid often showing blood and cancer cells, with mitoses,
, and (iii) others result from glands or tumor compressing
ta hepatis. (*f*) *Perforation* (*v.* PATHOLOGY) may occur.

er of the cardia presents symptoms more like those of œsoph-
ancer (*q.v.*). The symptoms may be few or absent. The
h is never distended. There may be tenderness under the
. The tumor when advanced may be palpated as well as the
tric lymph nodes.

nosis.—Diagnosis is easy in the typical case outlined in the
clinical picture. No single finding, as achlorhydria, or even
is pathognomonic; only the *grouping of symptoms* and signs is
Single analyses of the stomach contents are often deceptive;
ould be repeated constantly and considered in connection with
indings. Vomiting in middle or advanced life is suspicious
ccompanied by emaciation, anæmia, pruritus, and insomnia.
bsence of hydrochloric acid and the ferments, the findings of
blood, lactic acid, Oppler-Boas bacilli, dilated stomach, and per-
cancer tissue, are positive. The diagnosis may be doubtful, even
presence of pyloric tumor, emaciation, and gastric dilatation,
ydrochloric acid is present; the tumor may then be peptic
ypertrophy of the pyloric musculature, or pyloric spasm due
erchlorhydria; in the last instance the tumor is alternately
d then soft, and often the chyme may be felt or heard as it
ttently squirts through the pylorus. The diagnosis between

beginning cancer, nervous dyspepsia and atrophic gastritis must be reserved for a time. *Atrophic gastritis* is more chronic, often lasting years without emaciation. *Age* is of no diagnostic value. Youth and adolescence do not exclude cancer; in the young, gastric cancer is more rapid in evolution, and hydrops and peritonitis are more common. *Ulcer* (*q. v.*).

The so-called latent, dyspeptic, gastralgic, anasarcous, ascitic, and anæmic forms of cancer, and those causing early metastases (for example, in the liver) and intestinal obstruction, show in what direction error may occur, without due balancing of the individual and combined symptoms.

Differentiation.—(*a*) *Pancreatic* tumors (*q.v.*) are deep, fixed, immobile, and disappear on gaseous inflation of the stomach. They may be accompanied by fatty stools, and by glycosuria. It must be recalled that the normal pancreas is sometimes palpable in meagre subjects. (*b*) *Cancer of the gall-bladder* has respiratory excursion but neither expiratory fixation nor lateral movement; the digestive symptoms, chemism, ectasia, and changes on inflation, of gastric cancer are lacking. (*c*) *Duodenal* carcinoma is hard to separate, though the gastric chemism is usually lacking; passage of blood in the fæces, and icterus, are suggestive. (*d*) *Cancer of the colon* is usually distinguished by the presence of intestinal signs and the absence of stomach symptoms or chemism. (*e*) Enlarged *aortic glands* are distinguished by lack of altered chemism, and abdominal *aneurysm* by absence of cancer chemism and presence of expansile pulsation; (*f*) *omental* and peritoneal tumors are more diffuse. (*g*) *Hypertrophy of the pyloric muscle* (*v. s.*), felt as a tumor, is less common than is usually assumed; it is usually associated with the findings of ulcer or of hyperacidity; (*h*) foreign bodies (hair tumors), subserous lipoma, fibroma, myxoma, and other benign gastric tumors, and palpable round ulcers, are much rarer sources of error. Of sarcoma 59 cases are reported by Lecène and Petit; of these a dozen were successfully operated on.

Prognosis.—The prognosis unhappily is determined by the diagnosis, an error in which is the only hope. The average duration is one or two years (three months to three years). Death results from exhaustion by pain or vomiting, hemorrhage, gangrene, perforation, portal pressure, intercurrent pneumonia, nephritis, and tuberculosis.

Treatment.—**1. Medical or Palliative.**—(*a*) Condurango bark (Friedreich) seems for a time to increase the appetite and strength. (*b*) *Vomiting* is treated as in gastritis, dilatation, and simple ulcer, but with less success; *lavage* is indicated in pyloric stenosis; boric or salicylic acid or resorcin may be added for fermentation; narcotics by rectum, rectal feeding, champagne, and carbolic acid, as outlined before, are also useful. (*c*) Hemorrhage is rarely profuse and usually subsides on rectal feeding; iron, ergot, and lead, should be avoided. (*d*) The achylia is seldom helped by hydrochloric acid or pepsin. (*e*) *Pain* necessitates the use of fomentations, lavage, chloroform (3 to 5 drops) with ice, and as a last resort atropine or opium by rectum.

(*f*) For *constipation*, cathartics are inferior to warm *colonic flushings* or salts, glycerine or olive oil by rectum. (*g*) *Diarrhœa* is lessened by lavage; when due to pyloric insufficiency, salol, beta-naphthol (āā gr. x) and opiates are indicated. (*h*) Stenosis of the cardia may yield to dilatation. (*i*) Motor insufficiency, evidenced by food in the stomach from five to seven hours after a test meal, necessitates lavage, which should be practised before supper. (*j*) The diet should be soft and small in bulk. Meats and fats are digested with difficulty. Vegetables, cereals, small quantities of milk, koumyss, honey, pastries (not too rich), tea, coffee, diluted wine, peptones, albuminoses, baby foods, somatose, and nutrose, may be tolerated, but the stomach finally rebels at all food.

2. Surgical or Radical Treatment.—Cases having good surgical prospects are few, but operation only can cure. When a tumor is found it usually has a rather wide extension; pyloric tumors, for instance, have often reached the pylorus from the lesser curvature (Israel). For resection of the cancer the cases should reach the surgeon, if possible, before the tumor is palpable, a condition possible only in private practice, for hospital cases are always advanced. Metastases, peritonitis and glandular invasion are contra-indications to operation. *Pylorectomy* results in an immediate mortality of 15.6 per cent. as against the original death rate of 56 per cent. in the time of Billroth, who did the first operation (1878). The ultimate outlook is very doubtful, recurrence having been noted after five years. The Mayos, in 1906, reported 100 resections of gastric cancer, with a mortality of 14 per cent.; in one group of 25 cases only one case died; 27.7 per cent. of their cases were living three years after operation, and 22 per cent. were well over three years after resection. The Mayo brothers logically plead for an early diagnosis (by exploratory laparotomy) and for early resection. *Gastrostomy* is indicated in cardiac cancer, and *gastro-enterostomy* for pyloric obstruction.

(G). HÆMATEMESIS.

Definition.—Hæmatemesis is not absolutely synonymous with gastric hemorrhage; it is the vomiting of blood, which may come directly from the stomach or may reach it from the gums, nose, larynx, lungs, œsophagus, or even regurgitate backward from the intestine. Gastrorrhagia designates profuse hemorrhage from the stomach. It may be arterial, venous, or capillary.

Etiology.—(*a*) Various forms of *ulceration*, as simple ulcer, carcinomatous, tuberculous (of which Letorey [1895] found only 21 cases), diphtheritic, typhoid, syphilitic, uræmic, or phlegmonous ulcers, hemorrhagic erosions, and perforations into the stomach from without, as gall-stones, or aneurysm dissecting down from the arch of the aorta (Minkowski), are etiological factors. Parrot described superficial ulcers in the gastritis of the new-born. Dieulafoy describes a pneumococcic hemorrhagic gastritis. Chiari and Murchison, but especially Dieulafoy have described very small simple ulcers causing profuse, often repeate and fatal hemorrhage; their diagnosis seems probable when they develop in perfect health and with no previous stomach symptoms; the are easily overlooked at operation or autopsy because they involv only the mucosa or perhaps some of the muscularis; some regard thi form as the beginning of round ulcer. (b) *Trauma,* such as externa violence, or internal trauma, as from foreign bodies, thermal an chemical injury, and violent vomiting, as sea-sickness and hyperemesis, may be causal. (c) The cause may be *vascular,* such a amyloid degeneration which may sometimes involve the stomach an bowels alone; large or miliary aneurysm, arterial hyperæmia, embolism vicarious congestion from suppression of hemorrhoids or menstruation passive congestion of cardiac or hepatic origin; and œsophageal varix though it should be remembered that when due to cirrhosis of th liver there is in 33 per cent. of cases no other evidence of cirrhosis Welch and Powell have found varices in the floor of peptic ulcers malarial deposits in the liver; acute pylethrombosis, causing profus hemorrhage; and parenchymatous oozing, where no post mortem changes are found. (d) *Nervous* causes are injuries to the centra nervous system, vasomotor influences, and hysteria (aside from simulation and artifices), in which the blood is small in amount and occur usually in the morning vomit; tabetic crises; and general paralysis (e) *Acute infections* may be etiological factors, as malaria, hemorrhagi exanthemata, yellow fever, sepsis, embolism after operations (vo Eiselsberg), appendicitis (Dieulafoy), and genito-urinary sepsis (Guyon) Busse (1905) collected 96 cases following operations; they result fro direct embolism (of which 35 per cent. are in the mesentery or omentum) or retrograde embolism; the hemorrhage occurred in the stomac or bowels; 55 per cent. die. (f) *Blood diseases,* as pernicious anæmia leukæmia, hæmophilia, purpura, scurvy, and splenic anæmia, ma be causal. (g) *Poisons,* which early erode and later ulcerate, as acid and caustics, those causing fatty degeneration of the vessels as arseni and phosphorus, and uræmia or cholæmia, may cause hæmatemesis

Most cases occur in *women* between *fifteen and forty* years of age Of all causes, round ulcer and liver cirrhosis are the first to sugges themselves. At autopsy it may be impossible to find the point of issu of blood.

Symptoms.—The symptoms are (a) those of the *antecedent disease* (b) In some cases no blood may be vomited up, and the incident the remains undiscovered, unless the stools are examined. In *vomitu cruentus* gastric uneasiness is common. The amount vomited varie from microscopic quantities to those sufficiently large to cause death The occurrence of "*occult hemorrhage*" has been particularly emphasized by Boas; blood can be detected microscopically or chemicall when not visible to the eye, in over 25 per cent. of the cases of ulce and cancer; in microscopic examination the diet for days previousl should contain no meat, blood, iron, or chlorophyll, but should consis solely of eggs, fruit, flour, or fat. In profuse gastrorrhagia the blood is bright red, if it remains in the stomach only a short while, as it does in most cases of peptic ulcer. In smaller hemorrhages it is black or rust-colored, as in cancer, uræmia, cholæmia, various poisonings, and perhaps in peptic ulcer from action of the hydrochloric acid; rust-colored blood may become bright on the edges or surface when it has stood for some time. Blood may issue in the fluid form, or as clots, in which case it is suggestive of valvular disease, vascular diseases or the dangerous hæmatemesis which occurs in cirrhosis. In a fatal typhoid hemorrhage, which occurred in one of the author's students, enormous clots were vomited; the author has seen large clots vomited by patients having carcinoma of the stomach. (*c*) *Melæna* may render the stools black, tarry, and offensive. (*d*) *Systemic signs* may occur. A gradual or sudden intense *anæmia* may be noted, with all the usual signs of anæmia (*q.v.*), with vertigo, syncope, tinnitus, spots before the eyes, rapid heart action, and some dyspnœa. Later there may be amaurosis, slight febrile movements, dicrotic pulse, hæmic murmurs, moderate œdema about the ankles, albuminuria, and sometimes hemiplegia or convulsions.

Diagnosis.—Differentiation from hæmoptysis (*q.v.*), from the swallowing of blood from the nipples in nurslings or from the vagina in the new-born, from fracture of the skull, from œsophageal or throat disease, and from simulation of blood in the stools from ingestion of bismuth, coffee, tea, claret, iron, or blueberries, does not need particular elaboration, as the microscopic, chemical and spectroscopic examination determines doubtful cases. *Weber's test* for blood is made by adding to 15 c.c. of the vomitus or fæces, 5 c.c. of glacial acetic acid, and agitating with 20 c.c. of ether; the ether becomes brownish-red in the presence of blood, the brown acetate of hæmatin being formed. *Treatment* is identical with that of the hæmatemesis which occurs in round ulcer.

(H). NEUROSES OF THE STOMACH.

The neuroses of the stomach occur in nervous individuals, either as a part of a neurosis or as a rather isolated nervous symptom. Their relations will also be considered undeŕ neurasthenia and hysteria. They sometimes occur reflexly, as in gall or renal stones, or in uterine disease. Though their varieties will be considered as (I) secretory, (II) motor, and (III) sensory, combinations of symptoms are more frequent than single symptoms.

(I). SECRETORY NEUROSES.

These have been described especially by Reichmann, Rossbach, Sahli, and Riegel.

1. Hyperchlorhydria (Hyperaciditas Hydrochlorica).—This is a qualitative anomaly of gastric secretion in which the free hydrochloric acid is increased; it is not solely a neurosis, though conveniently classified as such; it is attended by no known anatomical lesion; it is an excessive secretion of hydrochloric acid during the height of digestion.

Etiology.—(*a*) *General* etiological factors are as follows: (i) It is most frequent in young and middle-aged persons; (ii) in the better situated classes, (iii) in Polish Jews, and (iv) in brain-workers; (v) it is somewhat more common in women than in men; (vi) a neurotic or psychopathic tendency is a predisposing factor, as is also (vii) chlorosis, in which hyperchlorhydria is found in 85 to 95 per cent. of cases. (*b*) *Local* causes are (i) rapid eating and cold drinks; (ii) condiments; (iii) smoking; (iv) certain foods and coffee; (v) gall-stones, or possibly renal stones, which induce hyperchlorhydria by irritating the stomach; and (vi) habitual constipation.

Symptoms.—Symptoms are quite often absent. The clinical picture varies much, and no single symptom is pathognomonic. (*a*) *Pain* is the chief symptom; it varies from a sense of pressure or discomfort in mild cases to extreme gastralgia in severe cases. It develops at the height of gastric digestion—*i.e.*, one to two hours after eating—and is due to the excess of free hydrochloric acid; it depends on the kind and amount of food ingested; the more food, especially meat, ingested, the later is the pain, for the food engages the free acid longer. Carbohydrates cause increased pain, as they do not engage the acid, and amylolysis is decreased, perhaps suspended; in exceptional cases the patient tolerates small and frequent meals better than larger ones. The patient is often aware of the increased peristalsis and feels a cramping or *spasm of the pylorus.* Pain lasts one-half to several hours. It is *relieved by vomiting,* and unless it has become quite severe is also relieved by ingestion of more food or of alkalies. (*b*) *Belching and pyrosis* are frequent, and the eructated hyperacid fluid causes a burning in the œsophagus—*i.e.*, along the spine. (*c*) *Vomiting* of a sour fluid, which causes a burning sensation, is not frequent, save in severe cases. (*d*) The appetite is usually good, and most patients desire food frequently, though in small amounts; occasionally it is ravenous. (*e*) *Tenderness* over the stomach is *usually diffuse,* moderate in degree and coincident with the pain; it is sometimes pyloric, and is associated with some epigastric distention. (*f*) *Stomach tests* show that the total acidity is increased to 100, even to 150 or 160; the digestive power is shown to be increased by the egg-disk test, the proteids are well subdivided, the amylolysis delayed, motility and absorption normal or increased in uncomplicated cases, and no lactic acid, no fermentation and no gas are present. (*g*) Other symptoms are as follows: The bowels are irregular, *constipation* being more frequent than diarrhœa; *headache* is common, and migraine or neurasthenic symptoms may develop; the gums may retract (Herter); in the *urine* the *chlorides are decreased,* the acidity lessened during digestion, and frequently an alkaline reaction and phosphaturia are present.

Diagnosis.—The diagnosis is determined by the presence of hyperchlorhydria in the vomitus and in the test meal, by the relief of the chemical pain by vomiting or ingestion of more food or alkalies, and by negative physical findings.

Differentiation.—(*a*) *Ulcer* (*q.v.* table) is marked by well-localized tenderness and hæmatemesis or occult bleeding, and its symptoms are much more regular than in hyperchlorhydria, in which bleeding rarely results from erosions. (*b*) *Gastritis* is characterized by mucus formation and decreased secretion, save in gastritis acida, in which the hydrochloric acid rarely exceeds 50. (*c*) *Gastrosuccorrhœa* or *continuous secretion* (*v.i.*). (*d*) *Gastrectasia* and *gastroptosis* are excluded by percussion and by inflation. (*e*) *Gall-stones*, in which pain has no relation to eating, occur at longer irregular intervals and are associated with turgidity and tenderness of the gall-bladder.

Course and Prognosis.—The disease begins slowly and may disappear, to recur after a few days, weeks, or months, from irregular living, nervous strain, excesses, certain articles of diet, or sometimes without obvious cause. The nutrition is usually fairly well maintained. The outlook is generally good if early treatment is instituted; complications, as continuous secretion, gall-stones, or gastric atony, modify the prognosis.

Treatment.—1. PROPHYLAXIS.—Nervous factors, smoking, haste in eating, chlorosis, etc., should receive appropriate treatment.

2. DIET.—(*a*) A *proteid* diet is most beneficial, for it takes up the largest amount of hydrochloric acid, which is not essentially increased by proteids, as has been maintained; veal, beefsteak, mutton, and ham, absorb twice as much hydrochloric acid as equal weights of other proteids (Fleischer); fish fat is particularly to be avoided; milk and some kinds of cheese may be given. (*b*) *Carbohydrates* should be restricted, because the early amylolytic gastric digestion by the ptyalin is delayed or arrested; a slice or two of stale bread, biscuits, toast, and zwieback, or purées of aleuronat meal, which contains much nitrogenous matter, may be given; Strauss allows six to nine ounces of 2 per cent. grape sugar solution, unless there is a complicating motor insufficiency. Potatoes, salads, and most uncooked vegetables, should be interdicted. (*c*) *Fats* may be given in limited quantities, including small amounts of cream or unsalted butter (one ounce); one or two drams of olive oil an hour after meals inhibit the secretion of acid. (*d*) *Condiments*, such as mustard, pepper, vinegar, spices, horseradish, coffee, alcohol, cold foods and drinks, should be forbidden because of their irritating action. Weak tea, cocoa or milk may be given. (*e*) *Thorough mastication* must be insisted upon. (*f*) *Small meals*, given at frequent intervals, are generally most efficacious, though Bouveret's three meals may sometimes be given if the patient's appetite is good.

3. MEDICATION.—(*a*) *Alkalies*, as sodium bicarbonate and magnesia usta, āā gr. xv to xxx, should be given at the height of digestion, to neutralize the hyperchlorhydria and relieve its symptoms; Pawlow maintains that alkalies do not only act chemically but inhibit secretion

by action on the glandular cells. The charged alkaline waters dilute the gastric juice, neutralize the acidity, and are sedative. (*b*) Extract of *belladonna*, ¼ to ½ grain, given at meal-time, lessens secretion and mitigates pain; opiates are seldom indicated and should be avoided (*c*) *Lavage with silver* nitrate (1 to 1,000) solution is useful in obstinate cases.

2. Gastrosuccorrhœa (Hypersecretion, Parasecretion, Continuous Secretion).—This is a quantitative perversion of gastric secretion, in which a fairly abundant secretion is found in the fasting stomach *i.e.*, it occurs without the stimulus of food, and when the stomach should be empty. In the lightest forms it only occurs when food is ingested (alimentary form); in the severest forms it occurs without ingestion of food. It is not denied that some gastric juice may be found in the normal stomach, due to secretion initiated by swallowed saliva or mucus, but if this exceeds one-half to one ounce it is rarely physiological. Two forms are distinguished, the continuous (Reichmann, 1882) and the intermittent form (Sahli, 1885). (*a*) The *intermittent form* occurs in attacks which last a day or two, is often called acute or nervous dyspepsia, and is found in about the same class of patients in which hyperchlorhydria develops; it has been observed during tabetic crises. *Colic-like pain* develops, *usually at night* followed by sour *eructations* and *repeated vomiting*, first of food, then of three ounces or more of yellow-green fluid which contains hydrochloric acid and ferments. The first vomitus may contain considerable hydrochloric acid and no more mucus than the small amount which the normal stomach contains; there is seldom blood. During the attack the pulse is small and rapid, the skin clammy, the urine scanty and alkaline, and headache is frequent. Thirst is great and drinking may give temporary relief, but is followed by increased vomiting Between attacks the acidity is normal, and the appetite and general health are usually good; occasionally there is a constant sense of gastric discomfort. (*b*) The second form is *continuous gastrosuccorrhœa*. Its etiology is similar to that of hyperchlorhydria (with which it may co-exist), but rather less clearly established.

Symptoms.—(*a*) *Pain is more marked* than in hyperchlorhydria develops later after eating, and very often at night (11 P. M. to 2 A. M.) it may occur before meals, for the gastric secretion is constant, or in severe cases it may be present both during the day and night; it is relieved by eating and by vomiting. There may be some local or diffuse tenderness. (*b*) *Vomiting* closely follows the pain, and may occur at its height, or during the night; the quantity of the vomitus varies; it is large when there is dilatation (a frequent complication and a common cause of this type). It contains hydrochloric acid ferments, and a small quantity of mucus; vomiting may be absent in light cases. (*c*) There may be *other digestive symptoms*. The condition of the *tongue* varies, being often *red and clean*, but sometimes coated. The *teeth* are often *carious*. The appetite is usually good, sometimes ravenous, or in advanced cases may be poor. The *thirst is marked*, and is sometimes suggestive of diabetes. The *bowels are sluggish.* (*d*) The results of the *test meal and lavage* show that the total acidity ranges up to 90 or 100, and free hydrochloric acid to 50 or 60. After a test dinner, the fluid, when removed, separates into three layers; the upper one is foamy, the middle one consists of yellowish fluid, and the lower one of sediment of fine undigested carbohydrates; the proteids are found to have been converted into peptones. When the stomach is washed out thoroughly at night to remove all fluid and food, examination on the next morning, of the fasting stomach reveals one to ten ounces of gastric juice (hydrochloric acid and ferments)—*i.e., secretion without the stimulus of food;* if the fasting stomach is washed out one, two or three hours later, gastric juice is again found—*i.e.*, there is continuous secretion, when the stomach should be empty. (*e*) Other symptoms. The urine is the same as in hyperchlorhydria. Old cases show marked *emaciation* which may resemble cachexia, particularly because of the frequently lax muscles and dry, inelastic skin.

Course and Prognosis.—The onset is slow and the course is protracted; it may last for years. The nutrition suffers and death from exhaustion may result in untreated cases. *Complications* are frequent, especially (*a*) dilatation (atony, motor insufficiency), for the stomach is never empty; the hydrochloric acid does not inhibit the growth of sarcinæ or yeast, and gases may generate. The dilatation may be primary but is most often secondary; it is differentiated by thoroughly washing out the stomach at night, and when the tube is introduced the next morning it should show nothing in simple dilatation. Dilatation may also result from (*b*) *pyloric spasm.* Pawlow has shown that in health the pylorus relaxes intermittently and allows the acid chyme to reach the duodenum; the pylorus is reflexly contracted by the acid fluid in the duodenum until this is neutralized, when the pylorus again relaxes. When the secretion is continuous, the passage of acid fluid into the duodenum constantly contracts the pylorus (hypertonic spasm) which sometimes may be felt as a tumor. In this case cancer may be suggested, especially when there is emaciation, or blood in the stomach, until the constant flow of gastric ferments and hydrochloric acid is found. (*c*) There is some tendency toward gastric ulcer. (*d*) Tetany is a rare complication of pyloric obstruction.

Diagnosis.—The diagnostic features are (*a*) the pain at night; (*b*) vomiting, and (*c*) thirst, both of which are more common than in hyperchlorhydria; (*d*) continuous secretion after washing out the stomach and during fasting.

Treatment.—Treatment is in general (*a*) that of hyperchlorhydria as to prophylaxis, diet, mastication, and stimulants. (*b*) *Rectal feeding* is a valuable accessory; water, too, may be given by rectum to quench the raging thirst. (*c*) *Oral feeding* is allowed at long intervals; overloading of the stomach with fluids is carefully to be avoided. (*d*) Lavage relieves pain and hypersecretion. Reichmann employs irrigation with silver nitrate (1 to 500 or 1,000). (*e*) *Olive oil* lessens hyperchlorhydria (Riegel). (*f*) *Alkalies* and belladonna are indicated as in hyperchlorhydria. (*g*) Treatment of *coincident dilatation;* massage or gastro-enterostomy (which, however, is only indicated by pylori obstruction, never for hypersecretion *per se*), may be indicated.

3. Hyposecretion.—Hypacidity (hypochlorhydria or subacidity) an anacidity (achlorhydria) refer respectively to reduction or absence o hydrochloric acid, which may occur from nervous as well as from organic causes (see GASTRITIS; ACHYLIA). When the ferments an hydrochloric acid are absent, achylia gastrica nervosa is a more appropriate term; the condition may be transient or enduring, and Einhorn reported a case in which secretion returned after an absence of fiv years. Suppressed secretion may occur in hysteria, tabetic crises, or less often, in other affections of the cord.

II. Motor Neuroses of the Stomach.

These may be irritative (increased motility) or depressive (decreas motility).

1. Irritative Types.—(*a*) *Hyperkinesis* or hypermotility hurries th food into the intestine, so that it disappears in three to five hours o even less. It exists by itself as a neurosis, but may occur in the hast digestion caused by hyperchlorhydria, and in gastric atrophy, apparently in order that the intestines may earlier digest the unaltere food. (*b*) *Peristaltic unrest* (Kussmaul) or *tormina ventriculi nervos* occurs in functional affections, particularly neurasthenia, and in organic pyloric stenosis. In some cases the unrest may be wholly subjective, the patient experiencing a rolling or cramping sensation. I others, both neurotic and organic, the peristaltic movements from le to right may be seen, as well as antiperistalsis in the opposite direction. The treatment of hyperkinesis and peristaltic unrest is that o the underlying neurasthenia and hysteria. (*c*) *Pyloric spasm* occurs very frequently as a secondary disorder, but very seldom, if ever, as an independent neurosis; it occurs usually in connection with ulcer, hyperacidity, hypersecretion, fissures, erosions, chronic stenosing gastritis, sometimes with cancer of the stomach, and with poor mastication of food and drinking of cold fluids. Its relation to dilatation has been discussed. (*d*) *Spasm of the cardia* occurs in organic disease of the œsophagus and stomach (ulcer, cancer, erosions, hyperchlorhydria), or in neuroses; it is the cause of idiopathic dilatation of the œsophagus (*q.v.*), and the spasm in tetanus and hydrophobia. It makes vomiting difficult and is differentiated from organic stricture by the fact that in cardiospasm a large sound passes more easily than a small one, and that spasm is often—not always—intermittent. The treatment of pyloric spasm is that of the cause (almost invariably gastric); the treatment of cardiospasm is discussed under idiopathic œsophageal dilatation. (*e*) *Nervous eructations* (Bouveret, Mathieu, Follet, Stern) occur in various neuroses and psychoses. They are said to occur especially in sexual neuroses. The odorless *ructus* does not result from fermentation, but from swallowing of air (aërophagia), which constitutes a most intractable habit. The mechanism by which air is swallowed is disputed; Bouveret correctly thinks it is forced down by pharyngeal contractions, but Oser holds that the stomach aspirates air. It is often swallowed unconsciously, or, again, it enters during what Matthieu terms false eructations, in which the patient, in attempting to raise gas, swallows air. The ructus can be caused only by aërophagia, as such large quantities could never generate in the stomach; Bardet estimated that one patient raised twenty quarts in an afternoon. Coincident cardiac and pyloric spasm causes distention (pneumatosis). Treatment lies in the use of the sound, suggestive therapy, and in the difficult task of breaking the habit. (*f*) *Nervous vomiting* occurs (i) only in the neuroses, as hysteria, neurasthenia, migraine, and exophthalmic goitre; but under this caption we may also bring for differential purposes (ii) cerebrospinal conditions, as anæmia of the brain, compression, meningitis, tumor, tabes, general paralysis, disseminated sclerosis and myelitis; toxic conditions, as uræmia or cholæmia, may cause cerebral vomiting. (iii) Most cases are *reflex* in origin; they may originate in the nose, as nausea from disagreeable odors; in the respiratory tract; in pelvic or abdominal conditions, as pregnancy, menstruation, gallstones, renal calculi, and peritonitis. The symptoms may present many incompatible elements; thus the patient may vomit without nausea, almost without effort, so that the vomiting is rather an eructation; it often occurs without pain or without reference to the fulness or emptiness of the stomach; even though it is frequent, the nutrition may be remarkably good, the tongue clean, and the appetite good. Periodical or cyclical vomiting, described by Leyden, has some resemblance to tabetic crises; Edsall considers it an acidosis, for which ʒss to ij of sodium bicarbonate is indicated. Robert's conclusions are as follows:

1. Vomiting of a chronic type, following a gradually developed epigastralgia, from one-half to three hours after the ingestion of food, is attributable to peptic ulcer, gastric or duodenal.
2. Chronic vomiting that occurs from ten to fifteen minutes after food ingestion is attributable to stenosis of the cardia, nervous abnormality, cerebral lesion (occasional rather than chronic), or to protracted acute gastritis.
3. Copious vomiting ten or more hours after food ingestion indicates a muscular insufficiency; frequent repetition indicates a permanent lesion in the nature of a mechanical interference with the exit of food.
4. Vomiting in the night is particularly liable to occur in cholelithiasis, periodic hypersecretion, muscular insufficiency, and nervous abnormality.
5. Vomiting attempts when the stomach is empty indicate a cause other than a gastric lesion, either a reflex cause, a toxæmia, a cerebral lesion, or a nervous abnormality.

6. Morning nausea and retching indicate either a beginning of pregnancy, alcoholism, pharyngitis, nephritis, or a nervous abnormality.

7. Periodic vomiting of clear gastric juice of normal or supernormal acidity in any considerable amounts indicates a secretory neurosis or an ulcerative lesion.

8. Vomiting as a sequel of headache, accompanied by severe nausea, but no gastric or abdominal symptoms, characterizes attacks of migraine; in a majority of cases eyestrain is the underlying cause.

9. Attacks of vomiting of sudden onset, with tinnitus, deafness, and vertigo, are attributable to disturbances of pressure in the internal or middle ear.

10. Periodic attacks of vomiting of sudden onset, associated with more or less severe gastric pain and nausea, retraction of the abdomen, obstinate constipation during but not preceding the attack, and freedom from abdominal tenderness, are suggestive of the spinal crises, idiopathic nervous vomiting, and lead-poisoning.

11. Periodic attacks of vomiting after abdominal colic and constipation, with localized or general tympany, are suggestive of chronic intestinal stenosis.

2. Depressive Motor Neuroses (from Lessened Motility).—There are three types: (*a*) *Atony*, which has been considered under dilatation; (*b*) *pyloric insufficiency* (Séré and Ebstein), which is seldom a neurotic condition, as compared with its frequency in cancer, ulcer, and dilatation of the pylorus from duodenal obstruction. Its presence is determined by inflating the stomach with carbon dioxide, which rapidly passes into the intestine with a gurgling gush. (*c*) The thir type, *insufficiency of the cardia*, causes *regurgitation* of food and flui and rumination (*merycismus*), which is a regurgitation in which th food is again chewed and reswallowed. These are generally involun tary processes, but some subjects can regurgitate at will. The patient are nearly always neurotic males, psychopaths, or idiots. The affectio is either hereditary or (which is more likely) imitative when it occurs i several members of a family. Its mechanism is a matter of theor congenital weakness of the cardia, stimulation of the vagus, an aspiration into the œsophagus by the inspiratory act, are considere causal. It should be treated by giving small meals at frequent interval these should be well masticated; intragastric electrical applicatio and suggestive measures are indicated.

III. Sensory Neuroses of the Stomach.

1. **Hyperæsthesia** may give the sensation of burning in the stomach or a sense of weight; this usually occurs when the organ is full, bu at times also when it is empty. Certain kinds of food, as carbohydrates fats or spices may induce the condition. Silver nitrate, gr. ¼ to ½, ac carbolicum gr. ¼ to j, or extr. gelsemii fl. ♏ij, given on the empty stomach, often afford relief, besides which the basic neurasthenia should receive treatment, and perhaps also the anæmia or constipation.

2. Gastralgia (gastrodynia or cardialgia, which is a poorly selected but widely used term) does not strictly include the gastric pain due to ulcer, hyperacidity, or hypersecretion. Rosenthal's etiological classification is as follows: (i) Cerebrospinal causes, including various brain, cord (tabes), peripheral, and functional nervous affections. (ii) Constitutional causes, including chlorosis and debility. (iii) Toxic causes, resulting from gout, rheumatism, malaria, and from nicotine and constipation. (iv) Reflex causes, from the genito-urinary and alimentary tracts; for example, from floating kidney, intermittent hydronephrosis, splanchnoptosis, uterine or ovarian disease, or masturbation. Fliess holds that many cases of both dysmenorrhœa and gastralgia arise from disease of the lower nasal turbinates. Most cases occur in women between fifteen and forty-five years of age.

Symptoms.—As the term implies, the affection occurs with severe gastric pain which begins suddenly, often at night, lasts for a few minutes to an hour or so, and subsides usually to recur after a variable interval. The pain is epigastric and may radiate widely. It bears no relation to eating, though food sometimes relieves it. Diffuse superficial tenderness or hyperæsthesia over the stomach is not infrequent, but deeper pressure usually affords some relief. Vomiting is rather uncommon. Hiccough, hunger, headache, depression, polyuria, clammy sweats, small rapid pulse, or even collapse, may attend the seizure.

Diagnosis.—A diagnosis is made only by exclusion and usually after the seizure is over. Rheumatic myalgia is more constant. A tabetic crisis is at once recognized by the Argyll-Robertson pupil, and the absent knee-jerks. Intercostal neuralgia gives Valleix's three tender points, which, however, are quite frequent in neurotic subjects. In ulcer (*q. v.*) or circumscribed peritonitis, the pain is associated with localized tenderness. Hyperacidity and hypersecretion should be excluded. Biliary colic (*q.v.*) is more difficult to differentiate, and it is well to remember that icterus is absent in the majority of cases of gall-stones, but a sharp thrust over the gall-bladder during deep inspiration nearly always elicits well-localized tenderness.

Treatment.—Treatment is that of (*a*) the etiology, especially the neurasthenic condition, anæmia, and constipation. Fowler's solution ♏ij before meals is excellent. (*b*) The attack must be treated. Morphine hypodermatically affords the only certain immediate relief, but in these very cases may establish the habit. If given at all, it should be given by mouth without the patient's knowledge. Heat and sinapisms may relieve or aggravate the attack. Silver, carbolic acid, or gelsemium employed in hyperæsthesia often give relief, as may extract of belladonna $\frac{1}{8}$ grain, cocain $\frac{1}{2}$ grain, charged waters, chloroform and Hoffmann's anodyne āā ♏xx to xxx, and galvanization with the anode to the epigastrium and cathode to the spine, though the attack is often over before application is possible.

3. Disturbances may occur in the sense of hunger and appetite. Loss of appetite (*anorexia*) may be purely nervous, though it is more commonly due to gastric affections, fevers, tuberculosis, or toxins. As

certain drugs as quinine, orexin, or even opium. *Acoria* is lo
sense of satiation. These anomalies are central and probab
from organic or functional changes in the bulbar hunger cent
treatment of the neurotic forms is that of the causal neurast
hysteria.

IV. Mixed Neuroses of the Stomach.

Mixed neuroses, dyspepsia nervosa (Leube), neurasthenia
(Ewald), or pseudodyspepsia, is marked by no stereotyped sy
It may occur alone as a species of local neurosis; it is sometim
from other organs, but is much oftener a part of the neu
symptom-complex. Leube's original contention that digesti
is normal must be modified, as the hydrochloric acid may be in
normal, or decreased. In many cases and perhaps in the maj
gastric functions are nearly or quite normal. Two main g
symptoms prevail; (*a*) the *centric*, neurasthenic, or hysteric
irritability, and vertigo; (*b*) the *gastric* eructations, variations
tite, oppression, and diffuse tenderness, which are probably als
(cerebral) in origin. The symptoms vary greatly in degre
change their form with considerable rapidity, and the general
is generally good. Organic disease must be carefully excl
repeated examinations of the secretions and motility of the s
for nervous dyspepsia is too often "a collective term to cover i
diagnoses." The treatment is that of the neurotic basis; rest
tion of worry and strain, self-control, and suggestion, are im

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DISEASES OF THE INTESTINES.

Etiology.—1. Primary Forms.—(*a*) Errors in *diet*, and the ingestion of spoiled food containing ptomaines, unripe fruit, and impure water, are common causes. (*b*) Disturbance in intestinal secretion may possibly occasion acute enteritis, though little is known of the succus entericus. (*c*) Cold, trauma, foreign bodies, particularly fæcal retention, and toxins eliminated from the blood, may initiate inflammation. (*d*) Some cases are *infectious*, though our knowledge of them is still imperfect. Bacteria are undoubtedly an important factor; it is stated that one-third of the weight of the normal stool is caused by bacteria, of which 48 species are normally present; acute inflammation may arise from pathogenic organisms or from virulence attained by some inhabitant of the intestine; the Bacterium coli is the most important, and Gaertner's Bacillus enteridis is probably one of its manifold forms. In a milk diet the Bacterium lactis aërogenes, and in a meat diet the Proteus vulgaris, may be possible factors.

2. Symptomatic Forms.—(*a*) Enteritis is constant in some infections, from the action of microörganisms or their toxins, as in typhoid; it is frequent in others, as in sepsis, or influenza; and rare in others, as in measles, scarlatina, rheumatism, or malaria. (*b*) General diseases, as nephritis; (*c*) burns; (*d*) local intestinal diseases, as intestinal parasites, ulcers, or tumors; and (*e*) metallic or other poisons are etiological factors; (*f*) stasis is usually listed as a factor, but is rarely by itself a potent cause.

Pathology.—The pathology is that of other inflammations of the mucous membranes; *viz.*, redness, swelling, increased mucus secretion, parenchymatous degeneration, and some interstitial infiltration with leukocytes. In the few cases coming to autopsy these changes may not be at all conspicuous, as they regress soon after death. In some cases follicular swelling or even ulceration may be observed as well as punctate ecchymoses, catarrhal erosions, or slight swelling of the mesenteric glands.

Symptoms.—The chief symptom is *diarrhœa*, due to increased peristalsis, increased secretion, or actual inflammatory exudation. The *stools* number two to fifteen daily, contain more or less mucus, and are variously colored; they may be brown-yellow with moderately frequent, or colorless and odorless with very frequent evacuations; their reaction is commonly alkaline or neutral; they contain triple phosphates, calcium phosphate, and oxalate crystals, sometimes cholestearin and Charcot-Leyden's crystals, but rarely leukocytes in any considerable number. There may be concomitant acute gastritis. Thirst, dryness of the mouth, colic pains, borborygmus, visible peristaltic movements, tympany, and slopping or gurgling in the intestine, are frequent. Fever is unusual, though febrile forms with acute splenic tumor are recorded, which may at first resemble typhoid. Albuminuria, cylindruria, or even acute nephritis, may develop in exceptional cases; the urine is scanty in proportion to the amount of fluid lost by the bowel; indican, etherial sulphates, and acetone, may be present. In some few cases herpes or muscle and joint pains occur. Collapse is rare in adults, though cases occur which bear a close resemblance to cholera. In the vast majority of cases convalescence is established in a few days.

Diagnosis.—(*a*) *Duodenitis* can seldom be diagnosticated; pain and tenderness over the duodenum are of most uncertain, and catarrhal icterus is of somewhat presumptive value in diagnosis. (*b*) *Jejuno-ileitis* seldom exists alone; diarrhœa is absent if the colon is not affected; the stools contain intimately mixed mucus, which often appears as small hyaline globules under the microscope (Nothnagel). (*c*) In *enterocolitis*, the bile is decomposed below the small intestine; when it is present in the stools it signifies increased peristalsis which moves the bile along before it can be disintegrated, and indicates acute enterocolitis; bile may be found only in the small globules of mucus and not in the fluid evacuation. Many *undigested food particles* (lientery) are found, and this is rather significant if fever, biliary or pancreatic obstruction, and gastric atrophy, are excluded; they consist of undigested meat, starch granules (which are more significant than muscle fibers), and fat particles. (*d*) *Colitis* is characterized by the absence of bile and lientery, by thin stools, and by more or less mucus, which is obtained especially on washing out the bowel immediately after a movement (Boas). (*e*) *Proctitis* is marked by much tenesmus, dysuria, evacuation of mucus alone, and the passage of bloody mucus covering but not mixed with the solid stools; it is also diagnosticated by the local examination with the finger or speculum.

Treatment.—(*a*) In the early stage, *evacuation* of the causal irritant is indicated, to which end fractional doses of calomel should be given, followed by castor oil; the stronger evacuants, which may of themselves irritate or inflame the intestine, should be avoided. Colonic flushings with pure water, or injections of olive oil, may be added. (*b*) *Absolute rest* in bed is always indicated. (*c*) The *diet* should be restricted to barley water, or no food should be given for a day or two. (*d*) *Heat* should be applied to the abdomen. (*e*) A dram each of *paregoric* and *bismuth* should be given after each bowel movement until their number is controlled. A quarter of a grain of extract of belladonna relieves colic. The following preparation is excellent; the amount for a child being given in the first and the amount for an adult in the second column:

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Phenylis salicylatis (salol)	gr. xx.	ʒj.
Sodii bicarbonatis	ʒss.	ʒj.
Cretæ preparatæ	ʒss.	ʒiss.
Bismuthi subnitratis	ʒj.	ʒj.

M. et divide in pulveres X.
S.—One powder in milk or thin arrowroot solution every three hours.

Lack of tone is relieved by gambir (catechu), colic by belladonna and flatulency by ammonia and ginger:

℞

Tincturæ gambir compositæ	℥j.
Fluidextracti belladonnæ	gtt. xij.
Spiritus ammoniæ aromatici	ʒij.
Tincturæ kino	ʒj.
Syrupi zingiberis	℥ss.
Misturæ cretæ q.s. ad	℥iv.

M. et S.—A tablespoonful every two or three hours.

Other astringents and intestinal antiseptics, as beta-naphthol gr. x p. c. in capsules, or 10 to 15 grains of tannigen (a compound of acetic acid and tannin), every two or three hours, are seldom indicated. (*f*) No laxatives should be given for the constipation resulting from this treatment. (*g*) Opium suppositories (see DYSENTERY) relieve *tenesmus.* (*h*) The diet may be increased in a few days to bouillon with raw egg, hashed beef or chicken, toast, zwieback, roast beef, and milk with equal parts of carbonated water; fruits and vegetables even when cooked should be given still later, and only with extreme caution, because recurrence readily predisposes to chronic enteritis. (*i*) Cramps, collapse, and other choleraic symptoms, are treated as in cholera (*q.v.*).

(B). CHRONIC ENTERITIS.

Etiology and Pathology.—The etiology is essentially that of acute enteritis, from repeated attacks of which, or of "acute indigestion," the chronic type may develop.

Chronic enteritis always suggests a causative chronic gastritis, though both affections may result from a common cause, particularly overeating and drinking. Chrónic enteritis may follow chronic gastritis, achylia gastrica, and other gastric conditions; or intestinal catarrh may cause anorexia, eructations, abdominal distention, and kindred symptoms. Hyperchlorhydria may disturb the intestines by the escape into them of more acid than they can neutralize; on the other hand hypo- or achlorhydria readily initiates bowel trouble because hydrochloric acid bears an important relation to the intestinal and pancreatic secretions.

The pylorus remains closed until the food in the stomach is ready for the intestinal juices and then it relaxes; the hydrochloric acid, reaching the duodenum, provokes a flow of bile (which arrests the action of the pepsin), and the acid also provokes the pancreatic secretion (which neutralizes the hydrochloric acid) by acting upon the duodenal mucosa, which gives off a substance known as "secretin," into the blood; secretin acting upon the pancreas causes it to secrete (Bayliss and Starling). When these ends are accomplished, the pylorus again relaxes. When the pancreatic juice is poured out, it contains no active proteolytic ferment, but in the succus entericus there is an "activating" ferment (enterokinase) which converts trypsinogen into trypsin; *i.e.*, the intestinal juice "activates" the trypsinogen (Pawlow).

The bile doubtless also increases the activity of the pancreatic enzymes or ferments; the action of the fat-splitting pancreatic ferment is enlivened threefold.

The pathology of chronic enteritis is also similar to that of the acute type. In color the mucous membranes is brownish-red or slaty-gray. The mucosa and submucosa are often thickened, and the muscularis is sometimes hypertrophied. Polypoid hyperplasia and cystic degeneration of the intestinal glands as well as atrophic changes may result. Catarrhal erosions and follicular ulcers may occur as described under the last type of dysentery (*q.v.*).

Symptoms.—Generally speaking, *constipation* is more frequent than diarrhœa; in comparatively few cases, a single soft morning evacuation is noted, or perhaps several; in others constipation is observed most of the time, broken occasionally by short attacks of colic and diarrhœa; continuous diarrhœa indicates inflammation of both the large and small gut. Constipation is due to alteration of the nervous fibers in the gut or to inflammation in the muscular coat. A sense of abdominal tension or colic may be present and is distinguished from gastric discomfort and pyloric spasm in that it occurs independently of ingestion of food. Diarrhœa results from decomposition and fæcal irritation. There may be pain, flatulency, borborygmus, or occasionally tenderness over the bowels, which may occur together or at intervals; but the sole diagnostic criterion is the presence of *mucus* in the passages; an intimate mixture with the stool indicates inflammation in the small gut; a mere coating of the passages with mucus indicates colitis, and evacuations of mucopus alone, proctitis. Mucus may be seen as small sago-like globules, which are often vegetable matter. These globules when bile-stained indicate inflammation of the small gut, as do shreds of bile-stained mucus. Small homogeneous denucleated epithelial cells may be found in the mucus adhering to scybala. Pus cells are rare except in proctitis. The presence of *mucus is always indicative of enteritis* and never occurs from ulceration or carcinoma alone (a slight coating of mucus over hard fæces is not significant). *Blood always indicates a complication*, such as piles, ulceration, or cancer. The stools may contain much *undigested meat, starch, or fat.*

Schmidt and Strasburger have proposed the *functional examination* of the fæces; while their method does not give us as exact information as does the examination of the stomach, valuable facts may be determined. Schmidt's test diet includes 1,500 c.c. (3 pints) of milk, 100 gm. (3 ounces) of zwieback or toast, two eggs, 50 gm. (1.6 ounces) of butter, 125 gm. (one quarter pound) of rare tender steak, 190 gm. (6 ounces) of boiled potatoes, 60 gm. (2 ounces) of oat-meal, and 20 gm. (two-thirds of an ounce) of sugar; this total is variously distributed among the three meals. With the first meal a capsule of five grains of charcoal or carmine is given, and the first examination is not made until the stools become colored black or red. In this way (*i*) the "period of passage" is established, the normal time being ten to twenty hours. (*ii*) Mucus is detected, microscopically, by mixing a piece of fæces as large as a walnut with a small amount of water; it appears as translucent, sago-like, and often bile-stained flakes, which must not be confused with starch particles. (*iii*) Much connective tissue, recognized by its yellow-white color and its toughness, indicates reduction of the hydrochloric acid. (*iv*) Muscle fibers, appearing as reddish threads or lumps, may indicate achylia gastrica, lack of trypsin, lack of the activating enterokinase, or very active peristalsis. (*v*) Free starch granules always indicate intestinal catarrh; normally only starch enveloped in cellulose is present. (*vi*) The normal stool shows hydrobilirubin, detected by mixing a small amount of the fluid fæces with an equal amount of a 5 per cent. solution of bichloride of mercury, which mixture turns yellow-red after twenty-four hours; bilirubin, recognized by a green color in the above test, always indicates catarrh of the small bowel (except in young children). (*vii*) The fermentation test is made by mixing a dram of the fæces with sterile water in a bottle which is connected above with a tube containing water; almost no gas results in normal cases; if the reaction becomes acid with a pathological fermentation, it is due to carbohydrate fermentation; if it becomes alkaline and foul in odor, it is due to proteid fermentation. (*viii*) With a little experience with the amount of fat normally found it can be readily determined whether the fat is pathologically increased.

The appetite is usually good. The general nutrition is good in some cases, though in others, pallor, emaciation, slow pulse, and cold extremities, may be noted. Attacks of diarrhœa, together with undigested food in the stools, should lead the physician to look for achylia gastrica (Einhorn, Oppler).

Prognosis.—The prognosis, save in either extreme of age, is good as to life itself, but is always doubtful at any age as to complete recovery. Improvement is at the best tardy and too often transient. Intestinal atrophy may develop, though this is less frequent than in puerile forms.

Treatment.—(*a*) Moderate exercise and an *hygienic* conduct of life are most essential. A flannel band should be worn over the abdomen. (*b*) The *diet* must be carefully supervised. Though it is impossible to give here a complete dietary, salads, beer, wine, fruits and vegetables of nearly every description, either raw or cooked, sweets, pickles, and acids, are among the forbidden foods, and the articles which may be given include eggs, fresh butter, lean meats, stale bread, rusks, thoroughly cooked rice or sago, and potato purée; *i. e., simple foods*, in *small amounts* and at *frequent intervals*, controlled by frequent examination of the fæces. (*c*) Regulation of the *bowels* is necessary. For constipation, purgatives should be avoided and the bowels moved by *enemata of olive oil*. Constipation is often due to the careful diet, which does not provoke peristalsis. For diarrhœa, bismuth in dram doses and tannigen or tannalbin (5 to 15 grains) are most efficacious (see ACUTE ENTERITIS). Calcium carbonate and salicylate are recommended by Boas and Javorski (āā ℥ss to a quart of charged water; one-half glass q. i. d.). (*d*) *Flatulency*—Some flatus is not pathological. As it results not from fæcal stagnation alone, but from stagnation plus catarrh (chiefly in the ileum, cæcum, and ascending colon), the first indication is that of the catarrhal enteritis; the second is the restriction of foods which ferment (*v. s.*); and the third is the administration of aromatic remedies (see Therapy of Acute Enteritis).

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Menthol	ʒss.
Phenylis salicylatis	ʒiij.
Extracti belladonnæ	gr. iv.

M. et ft. capsulæ XX.
S.—One after meals.

(*e*) *Colitis* may be treated by medicated rectal flushings, 1 to 1,000 or 2,000 silver nitrate, borax, salicylic acid, or ichthyol. (See Treatment of Dysentery.)

(C). ENTERITIS (COLITIS) MUCOSA OR MEMBRANACEA.

This disease was first described by Mason Good (1825), though the first classical description was Da Costa's in 1871. The name implies that the malady is an inflammation, but in reality most cases are neither enteritis nor colitis. Nothnagel proposed the name *colica mucosa* for the non-inflammatory variety, which is a secretory neurosis.

Etiology.—(*a*) Eighty to 90 per cent. occurs in *nervous* or *hysterical women* between twenty and forty years old, 40 per cent. of whom have uterine troubles; males who are affected are usually nervous or hypochondriacal; a few cases have been seen in nervous children. (*b*) Nearly all cases suffer from years of previous *constipation*. Its pathology is unknown; in some autopsies no anatomical alteration is noted, in others that of enteritis.

Symptoms.—(*a*) *Mucus casts* of the bowels, shreds or lumps, are voided periodically; the mucus may resemble a huge diphtheritic cast, is usually evacuated in large amounts, which often constitutes the entire bowel movement, and contains very few leukocytes or other signs of inflammation; it is rarely simulated by curds of milk. (*b*) There is usually, but not always, *colic* or pain and tenderness with the attack, which frequently occurs over the splenic flexure of the colon, is sometimes diffuse, and occasionally radiates, as into the leg. If the mucus is firmly adherent to the bowel wall the pain is particularly severe, and obstruction of the bowels may be simulated very closely. *Tenesmus* in voiding the mucus is usual, as are formation of gas, and *increase* of the original *nervous symptoms*, as palpitation, tremor, or vertigo. There may also be coincident diarrhœa, achylia gastrica, and a periodic sand formation (Dieulafoy). (*c*) *Constipation*, abdominal distention or pressure and anorexia exist between the paroxysms, which last a day or even a week, and are separated by intervals of weeks or months. It is said that death may occur during an attack.

Treatment.—Treatment concerns (*a*) the *fundamental neurosis*. As a rule, the less the neurotic symptoms are in evidence, the better are the results of therapy; education, the rest cure, and other measures outlined in the treatment of hysteria and neurasthenia (*q.v.*), are indicated. (*b*) *Constipation* (*v. i.*) is treated in the usual way, and an appropriate diet is essential. It has been maintained by von Noorden that the diet usually given in spastic constipation is not appropriate; he advises the use of graham bread, leguminous vegetables, including the husks, vegetables with much cellulose—which seems to be the most important element—fruits with small seeds and thick skins, as gooseberries, currants, or grapes, large amounts of cream, butter and bacon, and olive oil. Von Noorden's results with this diet are: 79 per cent. cured (15.8 per cent. partially cured, 50 per cent. permanently cured, 13 per cent. relapsed), 15.8 per cent. unknown result, and 5.2 per cent. failure. In White's series 50 per cent. recovered, 13 per cent. improved, and 37 per cent. were not improved. (*c*) Local measures include *colonic flushings* with normal salt solution, or half a pint of olive oil left in the rectum over night (Kussmaul). (*d*) *Narcotics, purgatives*, irrigation with alum, tannin, and silver solutions, and operations are distinctly *contra-indicated*. The morphine habit may be readily contracted; if given at all, morphine is exhibited with belladonna as a suppository. The patient in an attack is kept in bed and heat is applied to the abdomen.

(D). DIPHTHERITIC, CROUPOUS, AND PHLEGMONOUS ENTERITIS.

True diphtheria, due to the Klebs-Loeffler bacillus, is very rarely encountered, and practically all membranous enteritis is *diphtheroid;* it develops in various acute infections, in uræmia, and in metallic poisoning, and has been described under the third form of dysentery.

Phlegmonous or *purulent enteritis* is likewise extremely rare and chiefly of anatomical interest. Most cases develop in the duodenum. The affection may be primary, but more often is secondary to intestinal ulceration, carcinoma, or intussusception. Maragliano described a form probably due to the colon bacillus, in which multiple purulent foci are found in the intestinal wall, which frequently cause perforation.

(E). INTESTINAL ULCERATION.

I. Duodenal Peptic Ulcer.

This is almost identical in etiology, pathology, and treatment, with the peptic gastric ulcer.

Etiology.—(*a*) *Gastric ulcer* is 10 to 40 times as frequent as duodenal ulcer, whose frequency Kinnicutt estimates at $\frac{1}{10}$ per cent. in 30,000 autopsies. In 121 duodenal ulcers collected by Oppenheimer, gastric ulcer was present in about 6 per cent., and in Lebert's series of 237 stomach ulcers, duodenal ulcer was present in about 10 per cent. Moynihan found gastric ulcer in about 50 per cent. and Mayo in 22 per cent. Thirty-one cases of intestinal ulcer are reported after gastro-enterostomy for round ulcer of the stomach (*q. v.*); ulceration may be obviated by alkalies after operation. (*b*) Seventy-nine per cent. occurs in *males* as a result of alcohol and tobacco. (*c*) In Collin's series of 279 cases, two-thirds occurred between twenty and sixty years of age, being about equally frequent in each decade. (*d*) Other etiological factors are: (i) in extensive *burns* (Curling, 1842) acute ulcers develop in one to two weeks, rarely earlier or later; Holmes found them in 13 per cent. of 125 fatal burns; their cause is probably vascular obstruction, though the precise factors are not yet established (fibrin-ferment, toxins absorbed from the skin, aggregation of blood plaques). (ii) *Nephritis*, which occurs in 17 per cent. of the cases, is suggested as a cause by Perry and Shaw. (iii) It may result from *embolism* and thrombosis. Endocarditis and sepsis occasionally cause duodenal ulcer. Thrombosis of the *umbilical* and other veins produces the cases seen in the new-born (Landau). It has also followed trauma, multiple neuritis, periarteritis nodosa, and foreign bodies such as gall-stones. *Pathologically* the resemblance to gastric ulcer is marked in all details. It is *single* in 84 per cent., is located in the *upper horizontal part* in 87 per cent. of cases, and is almost always above the papilla.

Symptoms.—Symptoms are absent in 30 to 60 per cent., at least until hemorrhage or perforation occurs. This *clinical latency* is explained by absence of gastric hyperacidity, motor insufficiency, and peritonitis. (*a*) It is generally stated that *gastric symptoms are absent* in the great majority of cases. C. Graham, in an analysis of 46 cases operated by Mayo, found stomach symptoms, especially vomiting and gas formation, in about 70 per cent. The appetite is usually good. It is usually stated that the hydrochloric acid is not increased; Graham found superacidity in about half the cases. It must be remembered that associated gastric ulcer is quite frequent. The writer's views regarding the value of hyperacidity in gastric ulcer (*q.v.*) have been given. In one of his cases of gastric ulcer in which there was anacidity, Murphy found at operation a gastric ulcer and also a greatly dilated duodenum; in the jejunum there was marked stenosis due to a healed round ulcer, as shown by microscopic examination. This case is, as far as we know, quite unique; Tobiesen reports a jejunal ulcer following burns. (*b*) *Pain* often occurs three to six hours after eating, and is located over the duodenum, and is sometimes accompanied by *tenderness*. Both pain and tenderness may be peritonitic. (*c*) *Hemorrhage* from the bowels occurs in 33 per cent. of cases. Darras estimates it at 75 per cent. The stools are usually tarry, but are sometimes bright red from copious bleeding. "Occult" hemorrhage (small or microscopic hemorrhage) is even more common, and is as important as in gastric ulcer or cancer. Intestinal hemorrhage is often fatal from erosion of the pancreatico- or gastro-duodenal arteries, less often of the aorta or cava. Hæmatemesis is infrequent, though it has been said to occur in a quarter of the cases where there is intestinal hemorrhage; hæmatemesis usually indicates duodenal stenosis, in which the blood passes more readily upward than downward. Constipation is frequent.

Complications and Issue.—(i) Spontaneous *healing* is infrequent, and the clinical course is often long (five to thirty years). (ii) *Perforation*, total with peritonitis, or partial (chronic) with gall-bladder or pancreas symptoms, is very frequent (50 per cent., Perry and Shaw), and is even thought to be as high as 69 per cent. (iii) *Gastric dilatation* occurs in 7 per cent. and duodenal dilatation in nearly 2 per cent.; both result from duodenal stenosis. (iv) *Icterus* occurs in less than 3 per cent.; (v) duodenal cancer is an infrequent sequel.

Diagnosis.—Leube holds that a certain differentiation from gastric ulcer is never possible, and one is impressed with the frequency of symptoms of gastric ulcer in many reported cases. Von Wyl holds that differentiation from gastric ulcer is possible in but 10 per cent. of cases. The other chief cause of error is disease of the gall-bladder. As Graham states, "There will always remain a certain proportion of cases that will mislead the careful physician. Those cases of gall-stones in which the stomach symptoms of gas, distress, sour belching and dilatation predominate, and pain is only of a dull character, will usually be diagnosticated as ulcer; but the duodenal case, of which the chief symptom is the sudden, sharp, intense pain of perforative peritonitis, and in which, with no obstruction or hyperacidity, the other stomach symptoms are in abeyance, will be diagnosticated cholelithiasis. Both conditions are purely surgical, and the differentiation in many cases must be made on the operating table." Acute pancreatitis (*q. v.*), intestinal obstruction, and appendicitis, must be considered in the diagnosis.

Treatment.—The treatment is that of peptic ulcer of the stomach (*q. v.*), including its surgical indications. The operative mortality ranges from 70 to 85 per cent. Moynihan in 52 operations (1905) obtained these results: in 7 perforations, 71 per cent. recovered; in 22 chronic ulcers, 95.5 per cent. recovered; in 22 ulcers associated with gastric ulcer, 95.5 per cent. recovered.

II. Other Intestinal Ulcers.

Ulceration in acute infections, as typhoid, dysentery, anthrax, and diphtheria, has been described. In exceptional cases ulceration may occur in erysipelas, variola, and sepsis. *Chronic infections* may cause ulcers, as syphilitic, tuberculous, and actinomycotic lesions; these are considered under general infections. Very rare cases of ulceration have been observed from *favus* and *mucor corymbifer*. *Inflammatory ulcers*, as the catarrhal and follicular, usually occur in the colon; they may be simple or (so-called) dysenteric (*q. v.*) and may develop from the epithelial layer; they are seen in enteritis, infantile diarrhœas, and fevers. *Stercoral* (*decubital*) *ulcers* develop from the pressure of retained hard fæces or foreign bodies, and occur in the dilated pouches of the colon or above an intestinal stenosis. *Constitutional affections* are occasionally causes, as leukæmia, scurvy, gout, and amyloidosis. *Toxic* ulcers may develop in uræmia (*q. v.*), and in mercurial and arsenical poisoning.

Symptoms.—The symptoms are rarely distinctive without suggestive etiological factors; most ulcerations occur without symptoms or with ambiguous symptoms. (*a*) *Diarrhœa* is frequent, particularly in ulceration of the large gut; it is often absent with lesions in the small gut or in the upper colon. Diarrhœa results from decreased absorption and from increased peristalsis, due to exposure of the intestinal nerves. (*b*) *Blood* in the stools is mixed with them in lesions of the small gut, sometimes covers them in lesions of the large gut, and is voided independently of the fæces in rectal lesions. It is more often dark and tarry, especially in tuberculosis, than bright, except in low-situated lesions and in some profuse typhoid, dysenteric, or duodenal hemorrhages. It is not common in nontoxic enteritis. Blood is common in other diseases than ulceration, as in cancer, piles, stasis, and blood diseases. (*c*) *Pus* is seen more frequently in colonic than in ileac ulceration; it is common in cancer, proctitis, and croupous enteritis. Pure pus in considerable amounts is rather indicative of rupture of extra-intestinal pockets into the intestine, as appendicitic and salpingitic abscesses. (*d*) The presence of *mucus* has no special significance, as it indicates enteritis only, which of course may complicate ulceration. (*e*) *Shreds of tissue* are very rare, save in dysentery. (*f*) *Local symptoms* are likewise ambiguous, as tenderness, pain, and tympanites. (*g*) *General symptoms* depend on the basic disease; fever is most frequent in cancerous, dysenteric, or tuberculous ulcer. (*h*) *Rupture* causes peritonitic adhesions, diffuse peritonitis, retroperitoneal cellulitis, or subphrenic pneumopyothorax.

Treatment.—Treatment is that of enteritis or colitis. Of chronic ulcerative colitis Mehrkorn has published 38 cases in which colostomy resulted favorably.

(F). INTESTINAL DISORDERS IN INFANTS.

Etiology.—Classification of the etiological factors is extremely difficult because of the numerous species of bacteria in the intestine and the impossibility of drawing any absolute line between chemical and bacterial factors and between functional and organic changes. Most cases occur in children between *six and eighteen months old*, in the *summer months*, July especially, and in the *poorer classes*. It occurs largely in infants who are *not fed at the breast*, and for two main reasons; (*a*) cow's milk is prone to disturb digestion (see TREATMENT), and (*b*) there is a greater chance for bacterial infection. Bacteria operate in several ways. Vaughan and Fluegge have demonstrated that toxins may form in milk. This factor is especially potent when milk is long in transit, particularly in hot weather. Bacteria may cause inflammation of the intestines; the *Bacterium lactis aërogenes* causes inflammation in the small and the *Bacterium coli* in the large intestine; the proteus, pyocyaneus, pyogenic cocci, and, since 1897, Shiga's Bacillus dysenteriæ, are also factors, but their relative importance is not yet clearly defined. Escherich, Booker, and Favel describe a streptococcus enteritis, which, however, can hardly be considered as a distinct type; Baginsky considers the saprophytes of the intestine and of cow's milk causative, and Czerny holds that toxins may develop in the liver. The distinction between intoxication and infection is maintained with difficulty; *intoxication* causes such symptoms as erythema, hemorrhagic diathesis, anæmia, nervous disturbances, and dyspnœa; and *infection* produces certain complications as lobular pneumonia, nephritis, encephalitis, phlegmon, arthritis, purulent pleurisy, and necroses in the liver. In some institutional epidemics, infection may be carried from one individual to another by fæcal contaminations. Insects may also convey infection.

Symptoms.—(1) The symptoms of *the acute dyspeptic or fermental form* cannot be sharply distinguished from those of the other severer forms, though possibly caused by the same microörganisms. The pathological findings are often, as in adult forms, remarkably insignificant. The *temperature* usually rises suddenly to 103 or 104 degrees, remains high for a few days, and falls rather suddenly. The tongue is dry, the face red, the pulse rapid, and the breathing sometimes dyspnœic (Czerny). There is considerable restlessness—sometimes initial convulsions—anorexia, and vomiting. The *stools* in infants are normally yellow from bilirubin, which is not decomposed, are thick, and slightly acid in reaction; they become greenish from bacterial action or reduction of the bilirubin into biliverdin, react alkaline from albuminous decomposition or acid from acid fermentation, and become thin and offensive; the evacuations become frequent (6 to 10 daily). The microscopic findings are those of acute enteritis in adults, as mucus, leukocytes, triple phosphates in the alkaline stools, or cholestearin in the acid stools; blood in any quantity is uncommon. Involvement of the small intestine is attended by considerable formation of gas, some cramping, large movements, and slight tenderness and tenesmus. In severe cases the nervous symptoms may become conspicuous and considerable emaciation may result. Albuminuria occurs in about 20 per cent. and in many cases is attended by cylindruria. The author has seen two cases of extensive œdema follow dyspeptic diarrhœa without albumin or casts. Recovery is usual and prompt after thorough intestinal evacuation, though some few cases run a subacute or chronic course.

2. *Cholera infantum* occurs in the severest cases and usually in children already suffering from some intestinal disorder. According to Holt, it constitutes but 2 to 3 per cent. of summer diarrhœas. The *bowel movements are copious*, alkaline, and contain flocculi; though at first they contain food and are perhaps offensive, they become watery

and odorless. Both the stools and the general clinical aspect resemble the picture of *cholera Asiatica* and *cholera nostras* (*q. v.*). Though there is *fever*, the *extremities* are *algid and cyanotic*, the *pulse small*, the heart tones weak, and all the symptoms of *collapse* are present; the eyes are sunken, and there is the pinched pallid *facies cholerica*, the *thirst*, and the incoercible *vomiting*. The skin is rigid, as though the fatty tissue had hardened (*sclerœdema* or *sclerema adiposum*), and nephritis is almost invariably present. These symptoms result not only from loss of great quantities of fluid by purging and vomiting, but also from intoxication which produces the convulsions, irregular and Cheyne-Stokes breathing, retraction of the neck, stupor, and other symptoms grouped by Marshall Hall under the term "hydrencephalic state," and explained by him as due to œdema of the brain (*q. v.*), though better explained by intoxication. Oestreich thinks that hypostasis, resulting from compression of the lungs by the tympany, causes more deaths than does the toxæmia.

3. *Acute enterocolitis* (ileocolitis) may follow the dyspeptic form or the various specific infections in children, or may begin as an independent form. Pathologically it includes types which cannot be separated clinically, as (*a*) simple enterocolitis, (*b*) follicular ulceration, and (*c*) diphtheroid enteritis. The growing importance of Shiga's Bacillus dysenteriæ in infantile enterocolitis is established by the recent work in this country of Vedder, Duval, Flexner, Bassett, Knox, Park, Zahorsky, Dunham, Wollstein, and Rotch. In some epidemics the bacillus which is found produces acid in mannit media, which Shiga's bacillus does not produce. In Rotch's cases of infantile diarrhœa the Bacillus dysenteriæ was found in 16 per cent.; it was sometimes found in the dyspeptic or fermental type, but most often in the ileocolitic type (78 per cent.). In one collection of 412 cases Shiga's bacillus was found in 63 per cent.; Geo. Weaver never found it, though the Flexner-Harris type occurred in 25 per cent. (*v.* BACILLARY DYSENTERY).

The *symptoms* rather closely resemble those of dysentery in the adult. The *stools* are small, yellow, green, or brown, are very frequent, are attended by tenesmus and void mucus, blood, pus, and sometimes shreds of tissue or membrane. Blood may be present from congestion or acute ulceration. Rotch tabulates the differential features as follows:

Acute Fermental Diarrhœa.	Acute Ileocolitis.
(*a*) Small intestine, mostly.	Large intestine, mostly.
(*b*) 10 to 12 discharges; often large.	10, 15, to 50 discharges, usually small.
(*c*) No or little blood.	Blood and sometimes shreds of tissue or mem brane.
(*d*) Mucus.	Mucus.
(*e*) No tenesmus.	Tenesmus.
(*f*) Not much abdominal tenderness and pain.	Abdominal tenderness and pain.
(*g*) No or slight lesions.	Lesions marked.
(*h*) Temperature high—104° F. for one or two days, and then falling sharply by crisis.	Temperature may be high at first (103° F. t 104° F.) but usually soon moderates (99° t 101° F.) and falls gradually by lysis to norm after some weeks.

The severity of the symptoms varies greatly; the catarrhal form may cause mild symptoms or result in death, with or without gastric symptoms. The ulcerative and diphtheroid types are less often attended by vomiting, but run a fatal course of days or weeks.

4. *Chronic dyspeptic disturbances* often occur with gastric dilatation. The onset is gradual or it may directly follow the acute forms. The stools are green or gray, contain mucus, gas, and sometimes pus and tissue shreds. Food and fluid are not absorbed, or absorbed in part only; undigested food particles and fatty stools are observed; mucus is not common in marked cases of intestinal atrophy. Vomiting is rather frequent. There is usually but little general disturbance, no fever, and little or no colic. The scales show marked loss in weight and in some cases the emaciation may become extreme (pædiatrophy, athrepsia), the muscles becoming weak, the voice faint, the eyes lusterless, the face of the "Voltaire type," and the abdomen distended. Death results from marasmus or intercurrent infections.

Preventive Treatment.—Children should if possible be taken to the country in the hot months. Excessive swaddling is to be avoided.

Diet.—(*a*) *Human milk* is superior to all others, and if the mother cannot nurse her child, a wet-nurse should be procured if possible, as few cases of stomach and bowel disease occur in breast-fed infants. The wet-nurse must be carefully examined for syphilis and tuberculosis. The milk from the breast should spurt freely in several streams. A weak child may become dyspeptic even at the breast, and a fraction of a grain of calomel and magnesium carbonate give the most rapid relief. Other children thrive, even when the stools are green, for which Heubner blames the digestion of the infant. Sometimes colic and dyspepsia in the child are due to menstruation, beginning of pregnancy, excitement, lack of exercise, or alcoholic indulgence in the nurse or mother. If a wet-nurse cannot be obtained, (*b*) *cow's milk* or some *prepared food* must be used, but most of the latter are objectionable because their carbohydrates are not dextrinized, as shown by the iodine test. Cow's milk differs from human milk in many details; its contamination is far more likely, whence the importance of pure milk and sterilized nipples and bottles. Human milk contains about 1 per cent. of proteids, 4 per cent. fats, and 7 per cent. of milk sugar, but in cow's milk the percentages are respectively, about 3.5, 3.5 and 5 per cent.; in cow's milk the casein constitutes 80 per cent. of the proteids, which are physically and chemically different in mother's milk; *e.g.*, they contain more whey proteid and more of certain relatively uninvestigated substances, as nucleon. In cow's milk the ferments and fats are different and the mineral salts, especially phosphates, are increased which, as Blauberg has shown, increases the difficulty of digestion. The first objection is the high percentage of proteid (casein), which must be predigested, or otherwise the milk must be diluted. Simple dilution, however, necessitates giving too much water or reducing the fat and milk-sugar, fat being essential not only as a food but as an energizer, and milk-sugar being important because the Bacillus lactis aërogenes, operating upon it, produces lactic acid, which is inimical to pathogenic bacteria in the bowel. In modification of milk or in *substitute feeding*, then, the *proteid of cow's milk must be reduced, fat added in the form of cream*, and *milk-sugar added* to attain the normal percentage. The milk may be diluted with thin oatmeal water, as a diastase ferment is present earlier and in larger amounts than previously admitted, though nature doubtless has her designs in withholding carbohydrates from mother's milk. The fats of Holstein, Durham, Devon, and common breeds, can be better emulsified than those of Jerseys and Guernseys, which contain more volatile glycerides.

THEORETICAL BASIS FOR FEEDING A HEALTHY INFANT.
(According to Rotch).

AGE.	Fat.	Sugar.	Proteids.	Proteids if split.		Amount at each feeding, in—		Intervals between feedings, in hours.	Number of feedings, in 24 hours.
				Whey proteids.	Caseinogen.	Ounces.	C. c.		
Premature	1.00	4.00	0.25	0.25	0.25	⅛–¾	4-25	1-1¼	24-18
Premature	1.50	4.50	0.25	0.50	0.25	⅛–¾	4-25	1-1¼	24-18
At term	2.00	5.00	0.50	0.50	0.25	1	30	2	10
End of second week	2.50	5.50	0.50	0.50	0.25	1½	45	2	10
End of third week	3.00	6.00	0.75	0.75	0.25	2	60	2	9
End of fourth week	3.50	6.50	1.00	0.75	0.50	2½	75	2	8
End of sixth week	4.00	7.00	1.00	0.90	0.60	3	90	2½	8
End of eighth week	4.00	7.00	1.25	0.90	0.75	3½	105	2½	7
End of twelfth week	4.00	7.00	1.50	0.90	1.00	4	120	2½	7
End of four months	4.00	7.00	1.50	0.75	1.25	4½	135	2½	6
End of five months	4.00	7.00	1.75	....	....	5½	165	3	6
End of six months	4.00	7.00	2.00	....	....	6	180	3	6
End of eight months	4.00	7.00	2.50	....	....	7	210	3	6
End of nine months	4.00	7.00	3.00	....	....	8	240	3	6
End of ten months	4.00	6.00	3.00	....	....	8	240	3	6
End of eleven months	4.00	5.00	3.00	....	....	10	300	3	5
End of twelve months	4.00	4.75	3.50	....	....	10	300	3	5

"The data given above are simply a guide to the rules for the feeding of the average healthy baby; they are only approximate, and not intended to be followed in cases of difficult feeding without due consideration to the individual requirements."

Quite low dilutions should be given at first, and they should be gra ually increased. Attention should be given not only to digestion b to *nutrition*, the proper estimate of which is determined by regul weighing. Sterilization of milk destroys certain ferments, as the renn enzyme, besides protective substances (Ehrlich).

Treatment of Acute Dyspepsia.—(*a*) The *stomach* should *washed out*, if toxic symptoms are at all marked (Heubner); pla water or a 1 to 3,000 resorcin solution should be used. (*b*) *Calom* gr. ss and magnesium carbonate gr. j every three hours for abo three doses, or one dose of castor oil (℥ss to j) should be administere (*c*) The *bowel should be flushed* with warm water; one pint is sufficie in children of one year, or somewhat larger quantities in children t years old. (*d*) *Food should be withheld* (Czerny) for one or two days, and plenty of boiled water only should be given. Albumin water, which consists of the whites of two eggs beaten in a pint of water, may then be given, and thin arrow-root water, beef juice, and peptonized milk, may gradually be administered until the green color of the stools changes to a normal yellow-brown. (*e*) *Other medication* is usually superfluous. Lesage recommends that lactic acid be given for alkaline green stools, ʒj of a two per cent. solution (p.c.). Resorcin $\frac{1}{3}$ grain and dilute hydrochloric acid ♏ v may be given after meals.

Treatment of (Dominating) Enteritis.—If the case is seen in the early stages, a *calomel* purge should be administered, and the *stomach washed* out; and in all cases *feeding must be suspended* as outlined above. The child should have plenty of boiled water, and cool applications to the skin are indicated unless the extremities are blue and cool. *Tannigen* or tannalbin may be given in relatively large doses, 10 to 15 grains, several times daily, even in the very young, and may often be combined with minute doses of calomel (gr. $\frac{1}{80}$). As in enteritis of adults, bismuth subnitrate is given in large doses, ʒss. Bismuth salicylate, in doses of $\frac{1}{3}$ to 1 grain, is also given. If these are ineffective, opium ($\frac{1}{30}$ grain) may be administered for several doses.

Treatment of Cholera Infantum.—Food should be withheld. Lavage of the stomach and bowels is ineffective, unless it is done in the earliest stages. There are *three main indications:* (*a*) *fluid must be restored* to the tissues; this is possible only by subcutaneous saline transfusions which support the heart, fill the vessels, stimulate the kidneys, and eliminate the toxins; (*b*) *cardiac stimulation*, by hypodermics of strychnine $\frac{1}{300}$ to $\frac{1}{500}$ grain, and by camphor and local heat; champagne may be given to quiet the vomiting and support the heart; brandy and whiskey are given in doses like those employed in bronchopneumonia (*q. v.*), though Heubner believes that alcohol further injures the intestinal epithelium; and (*c*) *secretion must be checked* by administration of 5 minims of paregoric every four hours, of small enemas of starch and laudanum (10 minims), or, which is more effectual, of morphine ($\frac{1}{100}$ grain for a child one year old). In the fortunate cases which rally, nourishment can be resumed only with the greatest caution. The serum treatment is of uncertain value.

The Treatment of (Dominating) Colitis.—This is much like that of dysentery, and includes the measures above indicated. Flushings with normal salt solution, 1 per cent. tannin or 1 to 1,000 salicylic acid may be employed. Tannigen and bismuth should be given freely.

The Treatment of Chronic Dyspepsia.—Treatment in institutional cases begins with nursing; one wet-nurse can nurse several children. Lavage of the stomach with two to three ounces of plain warm water, normal salt solution, or 1 per cent. Carlsbad salts solution, increases the functional activity of the stomach. The extract of nux vomica stimulates secretion (gr. $\frac{1}{10}$ or according to the child's weight). The food must be given in the smallest amounts compatible with maintenance of weight and energy.

(G). APPENDICITIS.

Definition.—Appendicitis, the chief abdominal malady, is an inflammation of the vermiform appendix.

History.—Fitz in 1886 drew attention to the subject, and coined th name. Appendicitis was previously confused with typhlitis, or inflammation of the cæcum; though typhlitis does exist, it is rare, and th term "perityphlitis" should be entirely discarded. Our presen knowledge of appendicitis is largely due to the work of American surgeons, Willard Parker, who first (1867) advocated operation, Sands Bull, McBurney, Keen, Senn, Murphy, Weir, Morton, White, Deaver and many others.

Etiology.—1. **Predisposing Factors.**—(*a*) The appendix, being functionless vestige of the cæcum, is inherently *less resistant.* (*b*) It *lumen* is *smallest at its cæcal end,* which favors retention of foreig bodies, promotes microbic growth, and decreases or suspends drainag into the bowel. (*c*) The *meso-appendix,* which conveys the blood t the appendix, is *shorter than the appendix,* which therefore curls; th blood supply is then easily altered by appendiceal œdema and infiltration. (*d*) *Foreign bodies* and fæcal concretions may produce erosion which apparently promote microbic access and therefore inflammation. In J. B. Murphy's series of 2,140 cases, foreign bodies wer found in about 20 per cent. and fæcal concretions in 38 per cent. J. F Mitchell found foreign bodies in 7 per cent. of 1,400 cases of appendicitis, and Louis J. Mitchell in 1,600 autopsies found them in abou 1 per cent. Foreign bodies include seeds, capsules, gall-stones, pins nails, shot, fish-bones, etc. (*e*) *Age*—Over 50 per cent. of the case occur in persons under twenty years of age, and 30 per cent. in thos between twenty and thirty. It is rare in infancy, and with advancin years obliteration and atrophy lessen its frequency. (*f*) *Sex*—Male constitute about 80 per cent. of cases; in women it is thought tha an extra blood supply from the ovary lessens the results of infection though this supposition is disputed, and Murphy's large series show but a slight preponderance in males. (*g*) Other factors are traum and severe exertion, which are causal in a small number of cases exposure, which is a frequently alleged factor; and enteritis, whic is seldom a conspicuous prodrome, whence errors in diet are of n etiological importance.

2. **Bacteriology.**—The colon bacillus is the most frequent cause and is found in 80 per cent. of cases, though probably in some of them it has overgrown some other causal organism. Next is the staphylococcus, the virulent streptococcus, then the pneumococcus, proteu and pyocyaneus. Less frequently the typhoid, tubercle, influenza, o actinomycosis organisms are found; Fitz found 5 typhoid perforation of the appendix, and Fenwick in a collection of 2,000 cases of phthisi recorded 56 per cent. of tuberculous ulcerations in the bowel, of whic but 0.8 per cent. were in the appendix.

Symptomatology and Pathology.—1. The Early Clinical Course.—The *onset is precipitate,* is without prodromes in most cases, and is characterized by the *four main symptoms* upon the sequence of which Murphy has placed especial stress. (*a*) *Sudden severe abdominal pain,* the intensity of which ranges between colic and agony, is nearly always due to acute retention within the appendix caused by the occlusion of the cæcal end, and resembles the pain of retention in acute otitis media. It is felt over the *right lower abdominal quadrant* or over the pit of the stomach, and may even radiate along the genitocrural nerve to the testis, which is sometimes retracted. Reaching its greatest severity in a few hours, the pain persists until the pressure of the pus is relieved, which is accomplished by drainage into the bowel, perforation, or gangrene. Sudden relief from pain is most ominous, as it often indicates gangrene. (*b*) *Nausea and vomiting* result reflexly from occlusion and follow within a few hours after the pain. (*c*) *Abdominal tenderness* soon develops, is rather *general at first,* and is often associated with a cutaneous hyperæsthesia (Sherrin), which covers like half a girdle the right lower abdomen from the spine to the mid-line. The right rectus muscle resists palpation. A little *later* the general tenderness disappears, leaving *well-localized tenderness* over the appendix; this point is almost always in the right lower quadrant of the abdomen, at *McBurney's point* (about two inches from the anterior superior iliac spine in a line drawn between it and the navel), or a little below it in adults or above it in children; less frequently the tender point may be found in the loin, in the pelvis (5 per cent.), near the bladder, rectum, or uterus on rectal or vaginal examination, near the gall-bladder, or very exceptionally on the left side of the abdomen, or in hernial sacs, all of which correspond to possible variations in appendicular location. Pressure with the hand on the left half of the abdomen causes pain in the right side when the pressure is relieved. Palpation must be made gently, lest rupture occur. In the early stage of appendicitis it sometimes seems as though a tumor could be felt, though it is usually only the contraction of the abdominal muscle. (*d*) *Fever is almost invariably* present in the early stage of acute forms, and develops in a few hours to a day, according to the severity of the infection. An initial chill is rare. The temperature rises to 101°-102° and even higher in some virulent types or in children. It signifies septic absorption, and may cease suddenly in gangrene or gradually in rupture, and may slowly recur from extension. It does not signify cessation or absence of pus formation, as abscess or peritonitis may co-exist with low temperature. There may be other early symptoms. The *pulse* usually follows the temperature curve, though the author has seen it remain normal in cases of extensive early gangrene. The leukocyte count runs parallel with the fever, and often—though not always—measures the sepsis; *leukocytosis* may amount to 12,000, 25,000, or even 50,000, but no confident reliance can be placed on its absence as an assuring sign nor on its presence as an indication for operation. Constipation is present in most cases. The urine is often febrile, scanty, and usually contains indican and sometimes albumin; dysuria may result from appendicitis in the pelvis. Profuse bowel hemorrhage has been recorded. In Dieulafoy's toxæmic type toxæmia may obscure the local symptoms.

Fig. 37.

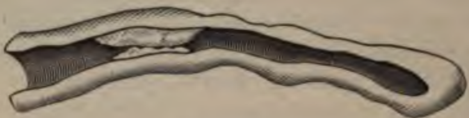


Fig. 38.

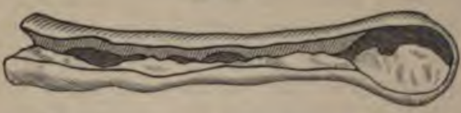


Fig. 39.

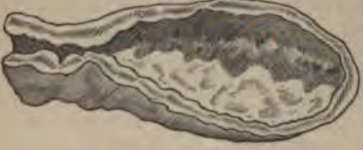


Fig. 40.

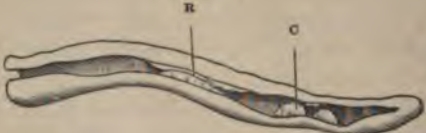


Fig. 41.

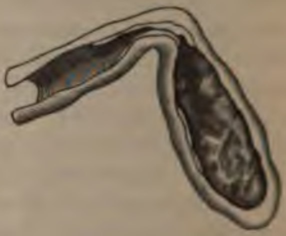


Fig. 42.

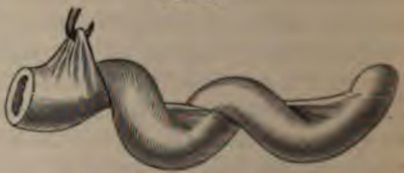


Fig. 43.

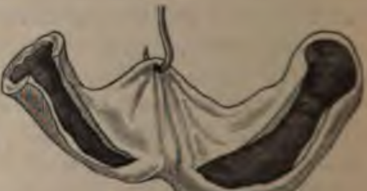
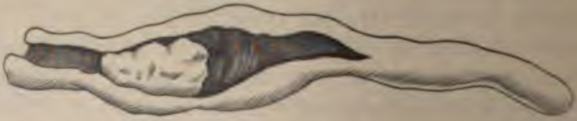


Fig. 44.



Figs. 37 and 38. Obliterating appendicitis. Fig. 39. Obliterating appendicitis, with distal enlargement. Fig. 40. Obliterating appendicitis (at R), with calculi (C). Fig. 41. Appendicitis with bending, obliteration and distal distention. Fig. 42. Appendicitis with curling around its mesentery. Fig. 43. Strangulation by a band. Fig. 44. Calculous obstruction. (Dieulafoy.)

2. The Later Clinical Course.—(*a*) The *inflammation subsides* gradually by drainage into the cæcum. Though the inflammation may be very circumscribed, the appendix rarely returns to a completely normal condition; it suffers necrosis and ulceration from microbes, from fæcal concretions, or both, which leave the appendix infiltrated, thickened, rigid and more or less constricted at one or more points. The mucous membrane may be wholly substituted by granulation tissue. Sometimes the lumen is entirely obliterated (appendicitis obliterans). Occlusion at its intestinal end causes a cyst-like swelling as large as the thumb or even as a sausage. Adhesions to the abdominal wall and gut are common. (*b*) After a few days a localized peritonitic *abscess* very frequently forms, usually over the psoas muscle at the angle between the ileum and cæcum, but occasionally over the unusual localization of the appendix (*v.s.* under PAIN). The appendix perforates, generally near its middle point at the mesenteric attachment, from necrosis or gangrene, and a localized abscess of yellow, thick, offensive pus forms; when the abscess is well demarked, the pain, fever, and pulse rate decrease. A *well-defined tumor* may develop, over which no peristalsis occurs; dulness or tympany are elicited according to the depth of the tumor or its fluid or gaseous contents; fluctuation is present only in very large abscesses; the tumor in infections due to the colon bacillus consists more largely of infiltration than of pus. In the incised abscess the green or black and totally gangrenous appendix may be seen, or it may be difficult to find. The abscess may form without any symptoms other than those of the onset. The *result of the abscess* may vary greatly: (i) it may rupture into the intestine which forms its wall (2 per cent.), and may possibly carry the appendix with it; (ii) it may rupture into the urinary tract, of which cases nearly one-half are fatal; (iii) it may cause thrombophlebitis purulenta, and any or all of the manifestations of septicopyæmia may follow, as secondary fever, suppurative pylephlebitis, liver abscess, subphrenic abscess, and vomiting of blood; (iv) it may rupture into the retroperitoneal tissue, which is followed by high temperature; and all possible extensions may occur, as into the groin, the scrotum with external rupture, the pelvis, the pararenal tissue, the pleura (84 per cent. are right-sided), the lung, gluteal region, back, hip joint, etc.; and (v) it may rupture into the free peritoneal cavity. (*c*) *The third cardinal issue* is diffuse septic *peritonitis*, which is recognized by all its unfortunate and unmistakable symptoms; these are a sudden fall of temperature followed by a secondary fever, diffuse replacing localized tenderness and rigidity, sudden hyperleukocytosis, persistent projectile vomiting, rapid pulse, Hippocratic facies, and collapse from profound intoxication. It most often follows the localized abscess, but may occur directly after a gangrene which is so sudden as to allow of no protective adhesions, or after perforation from a foreign body in the appendix without infection; the latter occurs in about 1 per cent. of cases, according to Murphy. This writer distinguishes variations due to the type of infecting organism: (i) The staphylococcic peritonitis which produces generally but little pus, or, if any, a seropurulent fluid; it forms a plastic lymph which prevents absorption for a short time, but, as the peritoneum becomes abraded, leads to intoxication; this is evidenced by collapse, an anxious expression, and by diarrhœa. (ii) The streptococcic form, which produces no pus, but causes a granular, blistered, dry peritonitis, which is quickly followed by rapid pulse, talkative delirium, and tympany. (iii) Colon infection, which produces an abundant, creamy, thick, offensive pus, and slowly causes death after great depression, low, muttering delirium, somnolence, and low arterial pressure.

3. **The More Remote Sequences.**—Intestinal strangulation by adhesions, deformity, and traction from chronic changes in the appendix, and intractable intestinal, neurasthenic, and nutritional disturbances, may result from chronic appendicitis.

In *children* the onset is usually very abrupt; the vomiting is repeated, the pain is greater, the appendix is more often palpable, diarrhœa is more common, and the percentage is greater (than in adults) of diffuse peritonitis, probably because the omentum is less able to envelop the appendix. In *aged persons* gangrene is more frequent than it is in youth or middle life.

Diagnosis.—The diagnosis depends on the four cardinal symptoms of onset, in the sequence named; *viz.*, pain, nausea or vomiting, abdominal tenderness, and temperature (Murphy), followed by leukocytosis and the other secondary manifestations, and by the developments of the later course, as circumscribed abscess or diffuse peritonitis. If all these symptoms are considered separately, an analysis is necessary of every abdominal lesion causing pain alone, vomiting alone, temperature, sensitiveness, abscess, tumor, disease in the right lower abdominal quadrangle, perforation, or peritonitis. While the existence of an acute typhlitis cannot be questioned, it is very rare and should seldom receive any consideration.

Pain occurs in gall-stones, renal calculi, tabetic crises, internal incarcerated hernias, epigastric hernia, intussusception, gastric or duodenal ulcer, tuberculous peritonitis, stone or torsion of the ureter (Dietl's crisis in floating kidney), uræmia, colica mucosa, acute pancreatitis, the crises of angioneurotic œdema, pelvic peritonitis, rupture of extra-uterine pregnancy, twisted pedicles (ovarian tumors), pyosalpinx, dysmenorrhœa, and the reflected pain of vertebral disease, pleurisy, and pneumonia. *Fever* in typhoid precedes the local symptoms. These affections, as well as gravitating abscesses, infected urachus, lesions of the abdominal wall, cancer, hysteria, and hypochondriacal fear of appendicitis, are almost invariably excluded by regarding the sequence of symptoms and by a careful general examination. In *appendicitis larvata* (Ewald), pain is slight or absent, and there may be diarrhœa; the thickened sensitive appendix is often palpable.

Exploratory puncture is absolutely contra-indicated.

Prognosis.—The prognosis is most uncertain, because (*a*) there is no safe criterion for early diagnosis of the type; (*b*) the symptoms and signs are not proportionate to the actual lesions—*e. g.*, light cases clinically may prove to be the most severe anatomically; (*c*) apparent improvement to the experienced observer is often only the onset of alarming complications, which are in reality less complications than essential parts of the disease; Dieulafoy well calls them "treacherous lulls" (*accalmies traitresses*); (*d*) though appendicitis often subsides clinically, recovery is seldom complete anatomically, whence recurrence (chronic, relapsing, or residual appendicitis) often occurs in a more severe form in 24 per cent. (Hawkins) to 44 per cent. of cases (Fitz).

Treatment.—Cases of appendicitis may recover, temporarily (70 to 80 per cent. or perhaps more), but it is impossible to predict which ones will do so. Personal prejudice was formerly against early, frequent, or universal operative treatment as advocated by many, as Deaver, Murphy, Holmes, and others. After testing and seeing tested the so-called medical treatment, consisting of rest, starvation, laxatives, and other placebos, and seeing gangrene, peritonitis with low temperature, and even death, occur in "mild" cases, the conviction is inevitable that *appendicitis is always surgical,* regardless of type, time, or tendencies, and a surgeon should at once be associated with the internist or practitioner in every case. Medical treatment is out of date and recovery under it is due to good fortune alone. The so-called indications for operation are too often signs of irreparable damage. Of course the results of operation depend upon the experience of the operator. Though this is not the place for discussion of technique, or of the *indication* for simple drainage or appendicectomy, the following points may be given: (*a*) Early operation *within one to two days* of onset is attended by the least danger. (*b*) Many operators dislike to operate between the *second* and *eighth days* or other arbitrary limits, but this position implies that the clinician can diagnosticate the anatomical changes which are present; gangrene, thrombophlebitis, imminent rupture, etc., cannot be excluded; in this period simple drainage is indicated—possibly, too, appendicectomy, if the appendix obtrudes in the field of incision; lavage, exploration, and protracted operation, are dangerous. (*c*) *In later cases,* when there is apparent regression, treatment may be more conservative; complete demarkation may be waited for, but even in such cases thrombophlebitis or rupture into the general peritoneum may intervene. (*d*) In cases of *diffuse peritonitis* incision and simple drainage are indicated unless the patient is moribund. The patient is placed in the semi-sitting posture (Fowler) and given frequent or constant normal salt enemas (Murphy). Many cases recover (see PERITONITIS). (*e*) *After the attack*—Murphy's reasons for operation are: "First, after an attack of appendicitis, the patient is prone to recurrence; second, upward of 60 per cent. of the cases operated on by me in the intermediate stage had had more than one attack; third, in the large proportion of the cases in which there was only incision and drainage of the abscess (which was a procedure frequently recorded by me in my first three hundred cases) the attacks recurred (in one patient I drained a peri-appendical abscess on three different occasions, always with severe sepsis, before I succeeded in inducing the patient to come in between attacks to have the appendix removed); fourth, the pathological changes, such as adhesions, flexions, and stenosis, following a primary attack, very much favor a recurrence; fifth, even if there were no danger of recurrence of the inflammatory conditions, the adhesions, fixations, stenoses, flexions, and retentions in the appendix,

produce sufficient disturbance of the digestive tract to necessitate
operation for their prevention; sixth, recurrent infections are practic
as dangerous as primary attacks (one patient died in the seventh attac
seventh, the appendix can be removed without danger." Dennis st
that under medical treatment the mortality is 16 per cent., relap
occur in 30 per cent., and diffuse peritonitis causes 100 per cent., mor
ity; under surgical treatment 4 per cent. die, there are no relapses,
30 per cent. of cases of peritonitis recover.

(H). INTESTINAL OBSTRUCTION.

Etiology.—Obstruction of the bowels may be due: (i) to in
susception, the most frequent cause, which is due to invagination of
part of the bowel into another; (ii) to strangulation, which ranks n
(iii) to twists (volvulus) and knots; (iv) to strictures; (v) to tum
(vi) to foreign bodies; (vii) to dynamic (paralytic and spas
obstruction. The figures quoted below are from Fitz's series of
cases, Leichtenstern's 1,134 cases and Treves' series.

I. Intussusception. (Invagination.)

This is the most frequent form (30 to 40 per cent.) of acute obstr
tion. The external part (the intussuscipiens or vagina) receives int
the intestine just above (the intussusceptum or invaginatum). T
actual *cause* is unknown; it develops in perfect health, in various ac
and chronic affections, in tumors and strictures of the gut, in diarrh
and other functional and intestinal disorders, and in contusion. T
theories are advanced: (*a*) that the gut is spastically contrac
(Nothnagel) and therefore enters the gut below, and (*b*) that the
below is paretic (Leichtenstern). The author saw a marked case
lead-colic, which recovered under operation by Van Hook. In ac
intussusception 50 per cent. occurs under ten years of age (Leicht
stern and Fitz); Wiggin in 103 cases found 50 per cent. in the fou
fifth, and sixth month of life. In chronic intussusception 50 per ce
occurs in persons between twenty and forty years of age; 66 per ce
occurs in males. *Pathologically* only the vital or inflammatory fo
is of clinical importance, in which the mesentery and its vessels are
invaginated; the "agonal" form occurs during the death ago
chiefly in children, in the small gut, is often multiple, and does
involve the mesentery. The vital form is usually single, descend
(in 99 per cent.), and complete; incomplete forms may result fr
polyps or other tumors, usually benign, which drag down the
There are three main forms: (*a*) the enteric, the small gut invaginat
into the small gut, 30 per cent.; (*b*) the ileocæcal, the small gut ent
ing the large gut, 52 per cent. Leichtenstern (75 per cent. Fitz and
per cent. Wiggin); and (*c*) the colonic, the large gut invaginating i

the large gut (18 per cent.), which includes the colon invaginating into the rectum; in rare cases a Meckel's diverticulum may invaginate into the small gut.

Symptoms.—(*a*) There is sudden, severe, *reflex pain,* which is usually umbilical in children but sometimes occurs elsewhere in adults, is continuous or intermittent, or paroxysmal in chronic forms of intussusception. (*b*) Early *reflex vomiting* may occur in children, though in adults it is less frequent than in other forms of obstruction; the later vomiting is seldom fæcal (13 per cent., Fitz); vomiting is more marked in high than in low invagination and depends considerably on the degree of mesenteric invagination and its necessary vascular disturbance. (*c*) The *stools are characteristic,* showing blood and mucus in 80 per cent. of acute and in 50 per cent. of chronic cases; in 108 cases collected by Hare and Martin, blood was present in the stools in 104; the movements may not stop at once but only after several evacuations. Their evacuation is attended by severe tenesmus (50 per cent.), especially in the young. (*d*) A *sausage- or egg-like movable firm tumor* (50 per cent. Leichtenstern, 61 per cent. Fitz) may be palpated, most often over the ascending or transverse colon, sometimes by the rectum or vagina, and is occasionally visible at the anus, where it has been mistaken for prolapse or polyp and has been extirpated. It is most common in children, and in the ileocæcal form. According to Treves it is nearly always to be felt during paroxysms of pain. (*e*) Meteorism and a depression in the right flank or ileocæcal region are infrequent.

Issues.—Peritonitis results in 50 per cent. of acute and nearly 100 per cent. of chronic cases; perforation occurs in chronic (15 per cent.) more often than in acute cases; sloughing off of the gangrenous bowel occurs in 42 per cent. of adult cases, and seldom in children; in Cruveilhier's case three yards of dead gut were discharged. Spontaneous reposition and fistula are very uncommon.

II. Strangulation.

This causes 35 to 38 per cent. of intestinal obstructions. Its *mechanisms* are innumerable; it is generally due to *bands,* or isolated peritonitic adhesions; Meckel's diverticulum, the remains of the omphalomesenteric duct, which lies within a yard of the ileocæcal valve and is sometimes continuous as a cord to the navel, has caused strangulation in 183 cases (Hilgenreiner, 1902); adhesions from previous peritonitis, appendicitis, or salpingitis; omental strands forming bands under which the gut is caught; holding of the gut in abnormal positions; and binding of coils to each other. Incarceration may occur when there are congenital or acquired *orifices and clefts* in the omentum or mesentery or under the suspensory ligament of the liver, ligamentum teres, or ligamentum latum uteri. *Hernias* include strangulations in the more common locations (femoral, inguinal, umbilical), or in the less frequent types as the obturator, sciatic, lumbar, intersigmoid, retrocæcal (Rieux), foramen of Winslow, duodenojejunal (Treitz's retroperitoneal form), or diaphragmatic. Seventy per cent. of cases occurs in males, 40 per cent. between fifteen and thirty years of age, 90 per cent. in the small intestine, 67 per cent. in the right iliac fossa, and 83 per cent. in the lower abdomen (Fitz).

Symptoms.—The symptoms are usually acute and urgent. (*a*) Early reflex *vomiting*, from shock, voiding in succession the gastric contents, bile, and fæcal matter which usually consists of yellow, brown, or black fluid or very rarely solid fæces, is strongly indicative of organic strangulation; vomiting usually lasts from the beginning to the end of the disease; retching results from nervous reflex irritation. (*b*) There is early severe reflex *pain*, which is greatest at the navel and lasts until collapse, peritonitis, or paralysis of the gut develops. (*c*) *Collapse,* with Hippocratic expression, weak pulse, and hurried breathing, which resembles the condition in cholera, results reflexly and may occur early from the disturbed local circulation in the gut, or later from peritonitis. (*d*) *Complete sudden stoppage of the bowels* may result in which no fæces nor flatus can pass; some fæces from the lower gut may pass after enemata, but no gas. Meteorism of two kinds occurs—(i) the static variety in which gas accumulates in loops above the obstruction, and (ii) the local variety, in which the strangulated loop becomes distended, chiefly because its circulation is impaired; it may be seen or felt as an *immovable tense mass,* showing no peristalsis and corresponding to the thick, œdematous, hemorrhagically infiltrated and finally gangrenous gut. (*e*) The *urine* is decreased, because of the vomiting, lack of food, and nervous collapse; it contains indican if the obstruction lies in the small gut, and is tested by treating with Obermeyer's solution (2 pro mille solution of ferric chloride in concentrated hydrochloric acid) and shaking with chloroform. Sometimes casts, albumin, or even hemorrhagic nephritis, are observed. (*f*) Strangulation in some cases may be found in the usual or accessible hernia locations.

Diaphragmatic hernia was first diagnosticated by Leichtenstern (1874); it is left-sided in 88 per cent., is congenital or acquired (from trauma especially), and is recognized in but 3 per cent. of the reported cases; its signs closely resemble those of pneumothorax (see DIAGNOSIS OF PNEUMOTHORAX).

In strangulation and other acute obstructions (*v.i.*) the mucosa in the occluded area may become eroded, more easily penetrated by bacteria, ulcerated, and hemorrhagically infarcted, leading to necrosis, perforation, and peritonitis.

III. Twists (Volvulus) and Knots.

Volvulus is a twisting of the intestine, which in nearly all cases occurs around the mesenteric axis; twisting of the gut on itself is rare; volvulus occurs in 14 per cent. of obstructions (Fitz); 68 per cent. of the cases are males, usually between forty and sixty years old; it occurs in the sigmoid flexure in 66 per cent. (Leichtenstern), less frequently in the cæcum (75 cases collected in 1905 by Corner and Sargent), colon ascendens, and small gut, and then especially when the large and small intestines have a small mesentery; only three cases of volvulus of the stomach are recorded. The predisposing causes of volvulus are a vegetable diet (said to explain its frequency in Russian peasants), persistent constipation, a long sigmoid flexure with a long but narrow mesentery, and mesenteritis. The twist may occur through 180 to 360 degrees or more.

Symptoms.—The symptoms closely resemble those of the forms described, especially when knots form between two coils of the small intestine. In volvulus of the sigmoid there is pain and vomiting, which is less marked than in strangulation or invagination and is seldom fæcal; the *local meteorism* is *more pronounced* than in any other type of obstruction, for the huge coil of the obstructed gut passes upward, possibly to the right, in extreme cases pushes up the diaphragm to the third or fourth ribs, and seldom shows any peristalsis, which its disturbed circulation prevents. But little water can be injected into the rectum. Hemorrhagic peritoneal exudate is common.

IV. Strictures (Enterostenosis).

Strictures more often cause chronic than acute obstruction. (*a*) *Congenital* stricture is even rarer than the rare congenital atresia (imperforate anus, ileocæcal, duodenal, or sigmoid atresia). (*b*) *Cicatricial* stricture results from healing of tuberculous, syphilitic, dysenteric, duodenal, and, very rarely, typhoid ulcers. In 1904 Arbuson collated 129 cases of tuberculous stricture. (*c*) Annular *cancer,* (*d*) compression by various organs or *tumors* (80 per cent. of which are pelvic), and (*e*) *traction,* are other causes. (*f*) Inflammatory hypertrophy of the mucosa and muscularis is exceedingly uncommon.

Symptoms.—The symptoms are those of chronic obstruction, and vary with the location of the stenosis. (I) *Stenosis of the small intestine* may occur (*a*) above the duodenal papilla, as a result of gallstones or duodenal ulcer, and resembles pyloric stenosis, from which differentiation is practically impossible; it may occur (*b*) in the duodenum below the papilla, as a result of gall-bladder adhesions, duodenal ulcer, tumors, or pancreatic disease (acute pancreatitis, tumor, cyst); icterus is frequent, as well as vomiting of bile and pancreatic juice (which digests starch and fibrin in an alkaline medium); other symptoms are dilatation of the stomach, pale stools, very slight abdominal meteorism except over the epigastrium, and the absence of indicanuria. (*c*) Jejuno-ileac stricture may result from healed ulcers, inflammation in replaced hernias, and adhesions to the internal genitalia; the ulcer stricture may be multiple, as many as twelve being recorded in one case; it is remarkable that extreme stenosis may cause no symptoms. There is constipation, which sometimes alternates wi diarrhœa, colic, meteorism, and visible palpable peristalsis, causi "intestinal stiffness" (Nothnagel) until the obstruction is finally ove come by the hypertrophied muscle above the stricture, when a gurgli sound is sometimes heard. The colon is collapsed. (II) *Stricture the large gut* is marked by *constipation,* which is significant when develops rapidly in a person not previously constipated or whe cathartics have no effect in constipated individuals; by paroxysmal *colicky pain,* even more than in stricture of the small gut, because th stools are harder; eventually by nausea and vomiting, which are su cessively gastric, bilious, and stercoraceous; by *meteorism* along th colon and in the lumbar region behind, which it is usually dull; b increased, even tetanic, *peristalsis* in the intestinal coils above th stricture, which possesses a distinct localizing value; and by th evacuations which may be *ribbon-like* or *sheep-stools,* and which ar often diagnostically overestimated, for they may occur in tumor starvation, colica mucosa, and spastic constipation; they are positiv neither by their absence nor presence.

V. Tumors (see page 677).

VI. Foreign Bodies.

Foreign bodies caused obstruction in 12 per cent. of Fitz's serie (*a*) *Gall-stones*—of which 250 obstruction cases are on record—lod chiefly in the upper duodenum or lower ileum, ulcerate through the gal bladder into the duodenum or less often through the colon, or co press the duodenum through a dilated pouch of the cystic duct. In a surprisingly small per cent. is there a history of colic (19 per cent.) or of icterus. Collapse is usually not marked, a tumor is seldom fel t, and the constipation is often but relative; indeed, diarrhœa ma y occur. (*b*) *Fæcal masses* are far more common than statistics sho , and form the chief obstruction of the large gut. The writer saw a case of acute absolute obstruction develop after a Thanksgiving di - ner; there was fæcal vomiting and severe collapse; a mass of no t particularly hard food, measuring two inches in diameter, was found; recovery occurred, though the bowel was partly necrotic. They ar found especially in the sigmoid (see CONSTIPATION). (*c*) *Enteroliths* are not common. They form slowly of phosphates, usually with an organic nucleus; sometimes they are constituted of bismuth, chalk, iron, or magnesia. They measure an inch to nine inches in diameter, and in one instance an enterolith weighed four pounds. Though most common in the large intestine, they may develop in diverticula of the small intestine. (*d*) Hair tumors, twisted masses of ascarides, fruit seeds, oatmeal husks (especially in Scotland), stones, and foreign bodies (in children or insane subjects), are possible causes.

VII. Dynamic Ileus.

The above causes are mechanical. Another form of acute obstruction is distinguished, namely, dynamic ileus (paralytic or spastic obstruction). Under this topic may be included obstruction following mesenteric embolism, trauma, abdominal operations, or inflammation in an undescended testicle; they are largely but not absolutely associated with acute peritonitis. Spasmodic obstruction may develop in rare cases without cause, or in neurotics, in gall-stone occlusion, ulceration, and intestinal crisis of tabes. It rarely produces fæcal vomiting; Bregmann (1901) found in the literature 20 cases of hysterical ileus.

Diagnosis.—1. Of the Fact of Obstruction.—The symptoms are generally unmistakable, and consist of constipation, cessation of flatus, distention (particularly the local form), compensatory peristalsis (hypertrophy), and frequently general reaction (collapse). The hernial sites, the rectum, and the vagina, must always be carefully examined.

2. Of the Location.—The uncertainties of diagnosis of location are manifold, but the following table brings out some of the leading criteria:

OBSTRUCTION OF THE SMALL.——*vs.*	——OF THE LARGE INTESTINE.
General Condition—Earlier and more profoundly impressed (reflexly) by toxæmia and peritonitis.	Later and less impressed.
Symptoms more marked, more stormy onset, more vomiting (both reflex and fæcal), more pain.	*Signs more marked*, as meteorism along colon (later becoming general), less water can be injected, more peristaltic effort and palpable rigid coils, more results by rectal or vaginal examination, more tenesmus.
Indicanuria—Develops early, and to marked degree.	Develops later or not at all (also present in cancer and peritonitis).
Urine—Much decreased.	Little decreased.

3. Of Its Nature.—Only the broadest probabilities may be considered. (*a*) It is practically impossible to recognize obstruction by *bands, adhesions*, Meckel's diverticulum, clefts, and internal hernias (except diaphragmatic). (*b*) *Invagination* is not only the most frequent but also the most characteristic form, because of its sudden onset (in children), severe (intermittent) pain, tumor, bloody stool (very rare in hernias replaced by operation or taxis, in strangulation, volvulus or gall-stones), tenesmus, results of rectal palpation, and the exfoliation of dead intestine. (*c*) *Strangulation* is next in frequency, and is differentiated from coprostasis, stone occlusion, compression, stenosis, and tumors, by its more marked and earlier collapse, its more severe initial pain and vomiting, its greater local meteorism, and more frequent hemorrhagic peritoneal effusion. (*d*) *Volvulus* is suggested by the advanced age of the patient, constipation, relatively slower onset and course, infrequent fæcal vomiting, and the meteorism, which is greater than in any other form and is accompanied by little peristalsis. The small amount of fluid which can be injected, the frequent hemorrhagic peritoneal fluid, the tenesmus, and the slightness, lateness or absence of indicanuria, are also suggestive. (*e*) *Gall-stones* often give no positive symptoms; in some instances the lack of shock, of increased pulse, and of meteorism, and the paradoxical intermittent release of flatus and fæces, may be suggestive. In no other type is fæcal vomiting, combined with the passage of flatus and fæces, so common.

4. Differentiation.—Confusion with flatulent colic, gall- or kidney-stones, and incarceration of the kidney, uterus, or testis, is avoided because in intestinal obstruction there is rapid stoppage of the bowels; the author saw one case of lead-colic with intussusception. The possibility of acute pancreatitis must be considered. Peritonitis is characterized by early fever, early diffuse tenderness, meteorism, cessation of peristalsis, board-like abdominal rigidity, often by fluid in the peritoneum, late collapse, late fæcal vomiting, later and less complete obstruction, and by frequent singultus.

Treatment.—In *stenosis*, which is typified by ulcer cicatrices, a concentrated fluid or semi-fluid *diet* should be given, and thorough mastication of solid food and avoidance of coarse food, or food with chaff, skins, stone, or gristle, should be insisted on. *Cathartics* are to be avoided, and the bowels should be moved with enemata of oil or water.

Thiosinamin has been recommended to dissolve cicatricial stenosis:

℞
Thiosinamin . . . . ʒj.
Glycerini . . . . ʒiij.
Acoholis diluti . . . . ʒvj.
M. et S.—Inject 8 to 15 drops subcutaneously once daily.

Operative measures may be instituted for foreign bodies or for malignancy, in which case the outlook is best in lesions of the colon and ileum.

1. Medical Treatment of Obstruction.—In every case the physician should at once divide the responsibility with a surgeon. In acute cases, especially in the small intestines, food causes aggravation. Early gastric lavage affords some, though transitory, relief. The possibility of chronic *fæcal obstruction* should be first eliminated by colonic flushings, given with the patient's head down or body inverted; hard desiccated fæces must be dug out with the finger, with frequent enemata to bring away fragments; in colonic injections a soft tube cannot be passed easily above the sigmoid, and hard tubes may cause perforation. *High enemata* and *colonic inflation* with air occasionally reduce volvulus and intussusception. Laxatives, massage, and puncture of the gut with an exploring needle, are always contra-indicated. *Opium* is useful in the early stage to relieve shock; in moderate dosage it does not produce intestinal paresis. *Stimulants* hypodermatically are frequently indicated. The use of *atropine* (Theden, 1788) has been recently revived especially by Batsch (1900); some cases are relieved by it, but dangerously large doses are necessary.

2. Operative Treatment.—Medical treatment of strangulation, intussusception, or volvulus, is of little value. A most convincing statistical argument for *early operation* is Naunyn's series of 288 cases, in which 75 per cent. of recoveries was obtained by operation in the first two days, while each successive day lowered the patient's chances to 40 per cent., 30 per cent., etc. Obalinski (110 cases) reports 35 per cent. recoveries, Treves (155 cases) 54 per cent., and Kocher in his earlier experience 38 per cent. and later 70 per cent. (96 cases of his own). Much depends on the physiological resistance of the patient. In intussusception Gibson's figures (239 cases) give 53 per cent. as the average mortality from operation, being 39 per cent. for operations in the first two days, and increasing with each day of postponement; it was 82 per cent. in children under three months of age; Rydygier's mortality in chronic forms was but 24 per cent. Forty per cent. of adults and 80 per cent. of children die under medical treatment (Leichtenstern). In 100 cases of intussusception in children 63 per cent. of operated recoveries are reported by Clubbe (1905). Fifty-two per cent. of obstruction by gall-stones recover under expectant and 33 per cent. under surgical treatment. For the indications and technique of resection, anastomosis, enterostomy, colostomy, etc., reference should be made to surgical treatises.

(I). INTESTINAL TUMORS.

Carcinoma is the only neoplasm of clinical importance. It is estimated that it constitutes 22 per cent. of all cancers. Its *etiology* is as obscure as that of all cancers. Trauma is seemingly an occasional factor and previous ulceration is a predisposing cause. It is most frequent in males over forty years of age; according to Maydl, 17 per cent. occurs in persons between thirty and forty and 14 per cent. in those under thirty years of age.

Pathology.—According to Leube, 80 per cent. occurs in the rectum, 15 per cent. in the cæcum and colon, and 5 per cent. in the small intestine. Haussmann, in 268 cancers of the bowel (excluding those of the rectum), found 91 per cent. in the large and 9 per cent. in the small bowel; if figured more in detail, 37 per cent. occurs in the sigmoid, 18 per cent. in the cæcum, 11 in the descending colon, 9 in the transverse and 8 per cent. in the ascending colon, 8 per cent. in the ileum, 4 in the splenic and 3 in the hepatic flexure, and 2 per cent. in the jejunum. Kanzler (1906) found in the literature, only twenty-five cases of cancer in the jejunum and ileum (*i.e.*, in the small intestine exclusive of the duodenal and ileocæcal regions). The sigmoid, splenic and hepatic flexures are especially predisposed, probably because these sites are more fixed and exposed to irritation. It is usually primary, sometimes secondary by contiguity, but very rarely metastatic.

Originating in the intestinal glands, it is usually annular in form, and causes stenosis and ultimate occlusion of the bowel; it is less often nodular and is very rarely diffuse and flat. Histologically it may be an adenocarcinoma (especially when it occurs in the duodenum), scirrhus (in the sigmoid), medullary, cylindrical-celled, (in the cæcum and sigmoid), colloid or flat-celled (in the rectum). Beginning in the mucosa, early involvement of the other coats and lymph vessels usually occurs, followed by later involvement of the peritoneum and metastasis, especially in the liver. (A. O. J. Kelly finds in the literature 49 appendiceal cancers.)

Symptoms.—1. Disturbance of the Intestinal Function.— (*a*) There is usually *constipation*, resulting from narrowing of the intestinal lumen; it is most marked in tumors of the large intestine. Ulceration frequently causes diarrhœa. (*b*) The stools are irregular and sometimes assume peculiar forms, as band-like or *sheep-like stools*, to which, however, undue importance has been attached, as they occur in inanition, starvation, constipation, etc. (*c*) *Blood*, *mucus*, and *pus*, are common in the fæces, though equally frequent in other ulcerations and in enteritis. The evacuations may be stinking. (*d*) Fragments of tissue are uncommon; they also occur in dysentery and are diagnostic only when showing carcinomatous arrangement of the cells. (*e*) *Pain*, due to enterostenosis, is usual; it may be sacral when due to sigmoid cancer. (*f*) Sudden complete *occlusion* of the bowel may follow previous gradual narrowing of the lumen. (*g*) The lower the tumor in the intestinal tract the greater is the *tympany* and *fæcal retention* and the purer the pus and blood (*i.e.*, the less mixed with the fæces). Above the stricture the bowel is dilated (tympany) and its muscle is hypertrophied (*visible peristalsis*). (*h*) Indicanuria has much the same significance as it has in intestinal obstruction.

2. Cachexia and Marasmus.—These, with the above symptoms, are most suggestive.

3. Tumor.—Until a tumor is detected, the symptoms described are not diagnostic. It is best felt if the bowel is empty; it may be confused with a normal contracted colon, the edge of the ileopsoas, or with fæcal masses; it is irregularly oval in clear cases, though in annular forms it escapes detection, for it merely constricts the bowel. It is usually tender, and is movable or immovable accordingly as the bowel involved is naturally free or fixed (incomplete peritoneal investment), or is adherent to other structures by adhesions or extension by contiguity. Whether free or fixed, the tumor is generally below the navel. Gas and fluid are sometimes felt or heard to escape through the stenotic point, and distention of the large bowel by introduction *per rectum* of air or fluid may aid in localization of the tumor. If the tumor grows from a part of the gut which is movable it may disappear and reappear from time to time. When no tumor is detected by abdominal palpation and inspection, a rectal and vaginal examination should always be made.

4. Complications.—Complications may first declare the malignancy of primarily ambiguous symptoms, as (*a*) icterus, especially in duodenal cancer; (*b*) hemorrhage from the bowels; (*c*) rupture into the bladder or vagina, or into the stomach (with fæcal vomiting and diarrhœa from food entering the colon).

Diagnosis.—The diagnosis is often difficult, except in cases of rectal localization, for latency is common and many symptoms merely indicate stenosis without declaring its nature. According to Leube, differentiation is required from other movable and immovable tumors:

1. Movable Tumors.—(*a*) These may be pyloric, requiring differentiation from duodenal cancers; the symptoms may be identical, but icterus, the presence of free HCl, bile, and pancreatic juice, in the stomach are aids in diagnosis. (*b*) The corset liver, movable spleen and kidney will be considered under those topics. (*c*) Omental and mesenteric tumors may be confusing, but they do not produce the same enterostenosis. (*d*) Scybala may co-exist with malignancy; the repeated use of enemata and cathartics determines the diagnosis (see CONSTIPATION).

2. Immovable Tumors.—(*a*) Renal and retroperitoneal tumors lie beyond the colon (on its inflation) and rarely cause the same degree of intestinal obstruction. (*b*) Encapsulated peritoneal exudate may be difficult of diagnosis; the author has seen two chronic appendicitic abscesses in individuals over sixty years of age which were diagnosticated as tumor, were hard on palpation, and which recovered by discharging pus into the bowel. (*c*) Ovarian or uterine tumors are less obstructive and are located more in the pelvis. (*d*) Localized inflammatory infiltration of the intestinal wall is distinguished by its regressive evolution. Bittorf, particularly, has called attention to circumscribed inflammation of the sigmoid flexure and colon.

Rectal cancer should receive special consideration in diagnosis. It is usually annular, and when developed is rigid, frequently resembles an inverted funnel, becoming narrow upward and may even resemble a hard cervix uteri. The colloid form extends upward and downward, is often tubular, and causes much regional invasion. A systematic rectal examination—which is unaccountably more often neglected than any other simple method of physical examination—saves many lamentable errors. The early stricture may yield when ulceration develops, and this usually occurs with discharge of blood, often ichorous pus, and sometimes malignant tissue. Pain is usually but not always present, tenesmus is the rule, and hemorrhoids are very suggestive. Cachexia develops with great rapidity, though it is no necessary symptom; the author has seen death result from obstruction with perfect preservation of color, nutrition, and energy. Intoxication symptoms are frequent, as stupor and other evidences of brain depression.

Syphilitic strictures are slower and subject to greater variations than those due to cancer; tuberculous ulcers are flatter, softer, and more undermined.

Cancer in any part of the bowel cannot be clinically differentiate from sarcoma, of which Lecène (1904) collected 75 cases involving th small bowel; their course is more rapid and they seldom stenose th lumen. (Ward [1904] found 34 lipomata recorded.)

Prognosis.—The prognosis of all forms and locations is bad. Mar asmus, coma carcinomatosum, anasarca, thrombosis, embolism, sec ondary infections, stenosis, or perforation, may be the cause of death.

Treatment.—Treatment is that of enterostenosis, in regard to die Opium must inevitably be used toward the end. Operation, eithe palliative (enterostomy, colostomy) or curative (enterectomy), ma be indicated. In 1896, Wolfler's series of cases of resection resulted i a mortality of 54 per cent., as against a death rate of 39 per cent. i resection for causes other than malignancy. Hochenegg (1902) in 19 radical operations, reports a mortality of but 12 per cent., and estimate his recoveries at 16 per cent. Czerny (228 operated cases, 1902) report 14 per cent. of permanent recoveries.

(J). ENTEROPTOSIS.

Enteroptosis is not a disease *per se*, but only a condition in which th abdominal viscera "fall" or prolapse in the abdomen. The stomac (gastroptosis), intestine (enteroptosis), colon (coloptosis), or soli viscera (splanchnoptosis), as the liver, kidney, or spleen, may be implicated. The condition is known as Glénard's disease, and Glénar referred it to laxness of the hepatocolic ligament. Ewald and other combat this view, and explain the transverse rigidity felt in the abdomen not as the colon, but the pancreas, exposed by sinking o the intestines. Its exact explanation is still disputed, though whateve causes relaxation of the abdominal wall or of the intra-abdomina suspensory structures may induce prolapse, as trauma, coughing, los of weight, pregnancy, tumors of the liver, spleen or kidney, spina deformity, and corsets. Seventy-five per cent. of the cases occur i women. Stiller maintains that splanchnoptosis is a congenital asthen condition whose most frequent stigma is a floating tenth rib (*cost fluctuans decima*); this is found in 80 per cent. of cases. He also speak of a *habitus enteroptoticus*, which somewhat resembles the *habitu phthisicus;* in the normal chest the vertical line drawn from the ensifor to the navel is short, compared with the distance from the median lin horizontally, to the floating ribs; in the *habitus enteroptoticus* th vertical line is much longer and the horizontal line is shorter tha normal and shorter compared with the vertical line; this *habitus* i considered a congenital predisposing cause, and enteroptosis is th resulting acquired disease. The tendency develops the disease, whe the patient's nutrition suffers.

Symptoms.—**1. Stomach Findings.**—Gastroptosis is characterize by falling of the organ to a lower point in the abdomen, whereby bot its upper and lower limits are abnormally low, the epigastrium i flattened, and the hypogastrium is distended. Eichhorst and Einhorn find it in 35 per cent. of women and in 5 per cent. of men. Dilatation of the stomach is excluded by finding the lesser curvature at a lower point than normal, which is determined by inflation with carbon dioxide. In marked gastroptosis the lesser curvature lies near the umbilicus; dilatation and gastroptosis may co-exist. The sharp angle formed at the pylorus or duodenum, too, may cause difficulty in gastric evacuation. Gastric symptoms may be noted, as anorexia, a sense of pressure, eructations, nausea, and gastric or intestinal hyperæsthesia, or as complications, vomiting, hyperacidity, or motor insufficiency. In many cases gastric and other symptoms are entirely lacking.

2. **Intestinal Findings.**—Enteroptosis or coloptosis is frequently found with gastroptosis. The transverse colon sinks in its central segment so as to assume the form of the letter U or V. Constipation, sometimes alternating with diarrhœa, and rumblings in the bowel, are frequent.

3. **Visceral Findings.**—Floating kidney, liver, and spleen, will be described separately; in this connection the only necessary comment is that floating kidney is neither cause nor effect of gastroptosis or enteroptosis, but merely coördinate to a third general causal factor.

4. **Nervous Symptoms.**—These occur in 50 per cent. of cases. Many subjects are nervous, delicate, slight, and irritable women. The most common nervous symptoms are lassitude, depression, a sense of cephalic pressure, backache, and other *neurasthenic* manifestations. The abdominal aorta frequently throbs with violence. The patient complains of a "falling out" sensation. Emaciation, constipation, and anæmia, are quite common.

Treatment.—(*a*) For the *neurasthenic symptoms* the treatment outlined under neurasthenia (*q. v.*) is indicated. The diagnosis should not be disclosed to the patient, for this initiates, by suggestion, a long train of additional symptoms. (*b*) *Increase in weight* should be sought, by ample feeding and rest in bed, which indication is met by the rest cure. (*c*) *Supports* to restore and maintain the viscera in their normal location often give considerable and immediate relief. The direction of the pressure should be upward and inward. Strips of adhesive plaster are useful but are uncomfortable. (*d*) *No drugs* can restore tone to the lax ligaments. Strychnine may be beneficial as a general and digestive tonic. (*e*) *Operation,* indicated only in cases with neurasthenic symptoms; sometimes, however, surgical interference is contra-indicated by these very symptoms. Shortening the mesentery, resecting parts of the recti muscles, suture of the liver, stomach and other organs to fixed tissues, have been performed seemingly with success, but this procedure can as yet receive no general commendation.

(K). DILATATION OF THE COLON.

Colectasia may be classified as follows: (*a*) *Gaseous distention*, which may occur in acute toxæmias. Fenwick recorded a case so marked that death resulted from heart paralysis. The treatment is that of tympanites (*v.* Typhoid and Enteritis). (*b*) Cases which are due to *obstruction* by fæces, foreign bodies, volvulus. (*c*) The so-called *idiopathic colectasia*, which occurs in children (in 80 per cent. of cases) as a congenital lesion, or in male adults over fifty years. Up to 1904 80 cases were published (Kredel). It is also known as Hirschsprung's disease, being first described by Hirschsprung, of Copenhagen. Treves holds that it is always due to obstruction, though cases without obstruction are noted, as Formad's case, in which there was great hypertrophy of the muscularis without organic stricture; the maximum circumference of the colon was 30 inches and its weight with the contained fæces was 47 pounds. The leading symptoms are obstinate constipation and meteorism. Of the reported cases only about 25 per cent. recovered either under medical treatment or after resection of the sigmoid, or colostomy. Death results from marasmus, auto-intoxication, intestinal obstruction, perforation, or peritonitis.

(L). INTESTINAL HEMORRHAGE.

Etiology.—There are five groups of causes. (*a*) *Abnormal intestinal contents,* as hard fæces, foreign bodies, gall-stones, caustic poisons, or parasites (anchylostomum or distomum). (*b*) *Intestinal lesions,* as ulcerations (dysenteric, tuberculous, duodenal, typhoid, carcinomatous, polypoid, hemorrhoidal), inflammation, as enteritis senilis, toxica, or cathartica; invagination, volvulus, and trauma. (*c*) *General affections;* cardiac or portal stasis, amyloid disease, infarction of the superior mesenteric vessels, inanition, aneurysmal rupture, scurvy, uræmia, and cholæmia. (*d*) *Infections,* including typhoid, dysentery, malaria, yellow fever, syphilis, tuberculosis, septicopyæmia, and hemorrhagic exanthemata. In the young (melæna neonatorum) sepsis is a prominent factor (see Hemorrhagic Diseases of the New-Born). (*e*) Blood *from the stomach.* The blood may be of capillary, arterial, or venous origin.

Symptoms.—The symptoms are: (*a*) *Blood in the fæces.* It may be *obvious* or "*occult*"—*i.e.,* found only microscopically, chemically, or spectroscopically. Blood is generally present microscopically a number of hours before it is seen by the naked eye. It is usually mixed with fæces, and dark, tarry and offensive when it comes from the small gut; it may coat the movements and be bright in color when it comes from the lower intestine. (*b*) *Acute anæmia* may follow profuse hemorrhage; this is evidenced by pallor, syncope, rapid heart, and slight febrile movement. (*c*) There are, finally, the symptoms of the *causal disease,* as typhoid or cancer

Diagnosis.—The diagnosis of enterorrhagia concerns (*a*) the detection of *blood*, which may be simulated by bismuth or blueberries, but its presence is easily established by the red disks, the hæmatin crystals, the guaiac reaction, and the spectroscopic lines; (*b*) the question whether the blood comes from the bowel or from the mouth or stomach; (*c*) whether it comes from the large or small gut (*v.s.*); and (*d*) its causes, which are most often hemorrhoids, typhoid, dysentery and cancer of the lower bowel. It goes without saying that no examination of the abdomen is permissible at the time of the hemorrhage.

Prognosis and Treatment.—The prognosis and treatment is involved in the basic lesion (*v.* TYPHOID). Symptomatic treatment includes absolute rest of body and mind; 5 grains of ergotin and one grain of opium should be given every two hours until bleeding ceases.

(M). PILES.

Etiology.—Hemorrhoids result from (*a*) *stasis*; portal stasis operates through the superior hemorrhoidal and inferior mesenteric veins, and is more potent than cardiac stasis, which congests the middle hemorrhoidal and internal iliac veins; (*b*) from *constipation*, pregnancy, and other obstructive lesions, as cancer of the rectum. According to Duret, vigorous contraction of the abdominal muscles is more potent as a cause than is constipation. Piles are most common in the fifth decade of life and in men. Hemorrhoids are usually spoken of as varices but, as Reinbach has shown, they are often cavernous angiomata.

Symptoms.—*External piles* appear as tender, painful, dusky-red or purple swellings outside the sphincter ani; they occur singly or may grow in a circle around the anus; they may be soft or hard. The pain produced is throbbing and may be very severe. Itching is very common. They may bleed, waste, ulcerate, or suppurate. Piles which bleed are called "open," and those which do not are called "blind." *Internal piles* occur above the sphincter, below which they may prolapse and strangulate. Tenesmus, mucus secretion, and dull pain sometimes referred to the back and the sacro-ileac articulation, may be noted.

Diagnosis.—Mere inspection is sufficient to exclude prolapse, condylomata, papillomata, etc. The possibility of carcinoma in their etiology should be borne in mind.

Treatment.—(*a*) *General* treatment should deal with the basic factors. Exercise is indicated for those of sedentary habits, and constipation (*q.v.*) should be relieved. (*b*) *Local* treatment: a small *injection of cold water* before each movement softens the irritating hard passage and constricts the piles, thereby favoring their reduction. *Cleanliness* is of prime importance in the relief of itching, for which also a 1 per cent. carbolic salve is of great value. In acutely painful cases a cocaine salve is beneficial. The following formulæ are astringent and anodyne:

℞
Extracti opii . . . . gr. ij.
Extracti belladonnæ . . . . gr. ij.
Acidi tannici . . . . ʒss.
Olei theobromatis . . . . q.s.
M. et ft. in suppositoria No. X.
S.—One at bedtime.

Or,—

℞
Cocainæ (alkaloid) . . . . gr.iij.
Iodoformi . . . . gr. x.
Orthoformi . . . . gr. v.
Olei theobromatis . . . . q.s.
M. et ft. in suppositoria No. XII.
S.—One at bedtime.

They must be given with reserve, lest the opium or cocaine habit be acquired. *Surgical* treatment is indicated when local measures fail.

(N). DIARRHŒA.

Definition.—Increased frequency of the stools with decreased consistence. Diarrhœa is not synonymous with intestinal catarrh, though a sharp differentiation is often difficult. In some individuals two or three movements a day are physiological.

Etiology.—(*a*) *Irritation* from the intestinal contents may cause it. Diarrhœa cathartica; some cathartics, as the aromatics, increase peristalsis, and others, as salts, cause increased transudation; diarrhœa dyspeptica is caused by intestinal or gastric dyspepsia, and by fruits and vegetables; diarrhœa stercoralis is caused by irritation from the fæcal masses; diarrhœa entozoica is caused by intestinal parasites. In general there are *two mechanisms*: (i) the stools are hurried through the bowel by increased peristalsis (irritation of the ganglia in the gut), in which case bile is found in them; and (ii) there is increased serum transudation into the intestine, when little or no bile is found. (*b*) *Nervous diarrhœa* causes from two to fifteen thin movements, containing no increased mucus; they are attended by rumbling in the bowels, peristaltic unrest, and are chiefly observed after mental excitement, as in taking examinations, or in neurotic individuals (hysteria, neurasthenia, or exophthalmic goitre); some cases are reflex, as from uterine or dental irritation. (*c*) *Toxic substances* in the blood may cause irritation of the ganglia or act upon the brain; for instance, uræmia, typhoid, sepsis, cholera, and possibly cold, produce diarrhœa by one or both of these mechanisms.

Treatment.—Opium is the most valuable remedy in diarrhœa, except in the stercoral and dyspeptic types; it acts less by checking secretion than by suspending peristalsis and mitigating pain and spasm; the crude drug is superior to its alkaloids, as its absorption from the bowel is slower, and its effect is therefore more marked; ¾ of a grain of the extract, or a dram of the camphorated tincture, should be given with bismuth in massive doses as in acute or chronic enteritis (*q. v.*). Important adjuncts are *local heat* (quieting irritation and peristalsis), *rest*, and a *spare diet* (boiled milk, eggs, toast, and later lean meat, as in enteritis.). In nervous diarrhœa two drops of Fowler's solution before meals is often advantageous.

(O). CONSTIPATION.

Constipation is a relative term, for some persons normally have movements every second or third day, and constipation (insufficient amount of fæces) may exist even with daily movements.

Etiology.—(1) "*Physiological*" constipation results from irregularity in going to stool, as in travel, from obesity, from insufficient exercise or water, from loss of fluid by sweating, from a dietary rich in proteids, as milk, and from various kinds of over-medication. (2) Constipation may be *symptomatic of other affections*, as (*a*) *gastric* ulcer, cancer, dilatation, or hyperacidity; (*b*) *intestinal* obstruction, cancer, adhesions, catarrh, passive congestion, or Glénard's enteroptosis; (*c*) *pelvic* conditions, as pregnancy, ovarian or uterine disease, or enlarged prostate; (*d*) *infections* (which stimulate the inhibitory nerves); (*e*) *nervous* disease, meningitis, tumors, myelitis, the neuroses; (*f*) diabetes and old age. (3) *Habitual constipation*, constipation *per se*, may occur in families. It may result from insufficient nervous energy (acquired or congenital) in the large intestine. Atony of the intestinal muscle may result from general debility or marasmus; it is very frequently a sign of neurasthenia, hysteria, or hypochondriasis, and results but seldom from actual disease of the musculature itself—that is, from atrophy or degeneration. Nervous constipation may be due to a spastic condition of the bowels.

Symptoms.—Symptoms other than constipation are often entirely absent, even after weeks, and when present are more often *local* (anorexia, coated tongue, bad taste in the mouth, or abdominal uneasiness) than general.

It should be remembered that two or three movements in one day or a movement every two or three days come within physiological limits. The stools normally accumulate in the sigmoid and pass every twenty-four hours into the lower rectum, causing a desire to defecate, usually at the same hour each day. The constipated stool is dry and accumulates in the haustra of the colon or ampullæ of the rectum. Constipation may occur even with daily movements, a small amount being retained each time. It is difficult to state the quantity of the normal passage, which certainly does not depend wholly on the amount of food ingested; fæces consist largely of mucus and bacteria, and form even during starvation; experimentally, fæces form in a loop of intestine isolated by ligature. The stools may resemble sheep's dung, and this is probably due to intestinal muscular spasm. Retained fæces are usually voided spontaneously in a few days as scybala covered with mucus which results from irritation or with a serous diarrhœa. Considerable obstruction may cause *fæcal colic,* which is due to contracti of the gut above the obstruction; there may be *stercoral fever* a *meteorism.* The retained *fæces may be palpable.* Still more mark obstruction may very rarely simulate organic enterostenosis, bei marked by collapse, vomiting, and great vesical or rectal *tenesmu* which should always excite suspicion. Extreme neglected cases ma occasionally result fatally, especially in the old, debilitated, or insane, in cases in which defecation causes great pain, as from fistula or pile

Fæcal concrements (coproliths) almost always occur in the larg intestine. When large they may simulate tumors of the intestine omentum, kidney, or spleen, from which differentiation may be possibl only after thorough purging and flushing. They may be soft and easil indented with the finger, hard, nodular, and tender from the bowe ulceration they incite, or, far less often, tender from localized peritoniti The history is not always conclusive, because fæcal tumors may develo with daily though inadequate movements. Gersuny found that o pressure the bowel may at first seem to adhere to the fæcal masses an then become free, though the author and many others have failed t note Gersuny's sign.

General symptoms are uncommon. Nervous symptoms are usuall causal rather than sequential, as in the neurasthenic subject, wh believes he would be well if his bowels would move, and who has mo unpleasant sensations when they do. Vertigo, pressure in the hea and mild dyspnœa are, in the author's opinion, always neurotic an never coprostatic. Copropsychiatry is, according to Nothnagel, gross exaggerated. *Intoxication* (ptomainæmia) is rare, but has been co sidered causal of neuralgia, chlorosis, and numerous other merely co comitant conditions. The aromatic sulphates of the urine may increased, which is due to formation in the gut of indol, skatol, an pyrocatechin.

Treatment.—Treatment of the causal factors (*q.v.*) is most i portant, as of irregularity of habit, insufficient exercise, or neurastheni

1. Diet.—(*a*) *Coarse foods,* as whole wheat, graham, rye, co meal, and bran breads, probably act less mechanically than chemical by the production of lactic or oleic acid. (*b*) *Fruits* relax the bowel because of the malic, tartaric and citric acids they contain, as plum apples, peaches, pears, prunes, strawberries, and grapes; grape sometimes and bananas and persimmons usually cause constipation (*c*) *Vegetables* act by the acids and gases they form; melons, sprouts cabbage, cauliflower, cucumbers, turnips, carrots, spinach, tomatoes, asparagus, onions, cress, celery, and squash, should be given freely, but it should be borne in mind that excessive amounts may cause fatigue of the bowel and constipation. (*d*) *Sweets,* in so far as they do not cause indigestion, are laxative; if used with discretion, sugars, syrup, candy, sauces, molasses, jellies, jams, marmalades, and honey, are valuable laxatives. (*e*) *Fats*—Moderate amounts of butter and olive oil are helpful; vegetable are superior to animal fats, which tend to derange digestion. (*f*) *Cold water,* taken on rising and through the day, is valuable in exciting peristalsis. Charged waters operate similarly. Hard water (containing an excess of lime) constipates, which point should be remembered both in regard to drinking and cooking. (*g*) The following are *to be avoided:* red wines (containing tannic acid), excess of meat, eggs or carbohydrates (rice, buckwheat, macaroni, and others containing little water), tea, milk (sometimes laxative when not taken in sips but swallowed in larger quantities) and beef tea. The diet recommended affords material for bacterial action, which follows the idea advanced by Strassburger and Lohrisch, that constipation is due to impaired motility, resulting in turn from too complete utilization of the average diet by the intestinal bacteria.

2. **Punctuality at Stool.**—The necessity of being punctual at stool was insisted upon by Trousseau. The best time for evacuation is immediately after breakfast, except in cases of piles or prolapse which may come down during the day after the morning stool; in these cases the bowels should be moved at bedtime.

3. **Posture at Stool.**—Squatting is the natural posture; this may be approximated by leaning well forward or by placing the feet on a footstool; the thighs thus support and compress the abdomen, a matter of particular importance when it is relaxed. Working the thumb of the left hand downward over the abdomen from the navel to the left iliac fossa is also recommended.

4. **Massage.**—Massage may give relief if continued for months. Its direction is not especially important, though the course of the colon should be followed. It should be given for a quarter of an hour, preferably before breakfast. Sahli's method was to roll over the abdomen a 7-pound cannon-ball covered with chamois. Massage should be gentle, especially in the aged. It is seldom beneficial in the obese. It is indicated in atonic but contra-indicated in spastic constipation.

5. **Mild Faradization.**—This arouses short contractions in the colon and is given with a moist sponge. Strong applications are avoided. Faradization and galvanization are inferior to massage.

6. **Hydrotherapy.**—The cold compress to the abdomen is a good adjunct.

7. **Exercise.**—Exercise is indicated to develop and contract the abdominal muscles, as in tennis, or rowing, or by lifting the legs while lying on the back, or lifting the rigid body from the lying to the erect posture solely by the abdominal muscles. Walking, bicycle-riding, and horse-back riding, do not exercise these muscles equally well. In women with ovarian and uterine diseases, exercise only increases the constipation, which is generally due to reflex spastic inhibition of peristalsis, as shown by the laxative effect of narcotics (Littlejohn and Brunton). It is claimed that constipation is *spastic* (not atonic) in 25 per cent. of female cases, as evidenced by colic, palpable or visible knots in the intestine, and sometimes by ribbon-like passages; in these cases irritating and coarse diet, massage, cold, and electricity, are to be avoided. Hot Sitz baths or full hot baths, hot compresses to the abdomen for spasm or colic, are indicated; an enema of eight ounces of

warm olive oil is given each evening, and an attempt is made to have it retained until morning; one-third of a grain of extract of belladonna is given as a suppository one to three times a day, to relieve spasm and the frequent rectal tenesmus; the diet should not consist of coarse food but of fine carbohydrates, butter, and cooked fruit.

8. **Enemata.**—Enemata excite peristalsis, lessen intestinal hyperæmia, and apparently are beneficial to the hepatic secretion and circulation. They do not derange digestion as do cathartics, but after a time they may lose their effect by dilating the colon, whence the indication for their intermittent use. Water and olive or linseed oil maybe used; oil enemata (five to eight ounces) are especially indicated in spastic constipation and colica mucosa, in which the oil may be left in the bowel over night. Glycerine suppositories are valuable, if used alternately with other methods. In fæcal impaction with marked obstruction enemata are invaluable, and should be combined with cathartics and digital evacuation of the hardened masses.

9. **Cathartics.**—The use of cathartics is governed by the following general rules: (*a*) they should be administered in chronic cases only after failure of the measures enumerated above; (*b*) their most successful employment is intermittent and alternating; (*c*) drastics, hydrogogues, and cathartics producing secondary constipation, should be avoided, save, of course, in extreme cases.

Varieties.—Aloes, found in nearly all proprietary and officinal pills, should be avoided in piles and uterine hemorrhage, because it congests the pelvic vessels. Like other cathartics, it is usually given in combination, *e.g.*—

℞

Extr. aloes aquosi	gr. ss.	Acting in fifteen to twenty hours, on the large bowel chiefly.
Extr. rhei.	gr. ij. to v.	Actively purgative in four to eight hours; stomachic and tonic.
Extr. nucis vomicæ . . .	gr. ¼.	Acting on peristalsis and combating atony.
Resinæ podophylli	gr. ⅒	Acting in ten hours, producing "bilious stools."
Extr. belladonnæ. . . . . . (or extr. hyoscyami)	gr. ¼.	Relieving spasm (griping) and operating on the intestinal nerves.
Extr. teraxici	gr. j.	

M. et ft. pil. I.

Rhubarb has the disadvantage of griping and of producing secondary constipation, which is caused by its tannic acid ("a patient sells his soul to rhubarb"), whence it should never be given alone:

℞

Pulveris rhei compositi	ʒiv.
Sodii sulphatis	ʒiv.
Sodii bicarbonatis	ʒj.

M. et S.—One teaspoonful at bedtime.

Cascara sagrada is an excellent laxative, and produces very little constipation afterward; the fluidextract, fluidextractum rhamni purshianæ, ♏x to xx, is very bitter; the solid extract, extractum rhamni purshianæ, is given in doses of from two to eight grains. *Licorice* is given as pulvis glycyrrhizæ compositus, ʒss to ʒj. *Castor oil* is a soothing laxative, relieving irritation and spasm (in nervous or lead constipation, in which latter it may be combined with opium gr. ¼); its offensive taste may be partly overcome by combining with glycerine and gaultheria:

℞

Olei ricini	℥ij.
Olei gaultheriæ	gtt. xl.
Glycerini	℥ij.

M. et S.—One teaspoonful to one tablespoonful.

In anæmic patients the following formula is recommended:

℞

Ferri sulphatis	gr. x.
Extracti aloes aquosi	gr. v.
Extracti rhamni purshianæ	gr. xx.
Extracti belladonnæ	gr. iij.
Extracti nucis vomicæ	gr. iij.

M. et ft. pilulæ No. X.
S.—One after meals.

Calomel should be given in the evening in fractional doses, and may be advantageously combined with podophyllin, which acts in the same time (eight hours); they should be followed the next morning by half an ounce of magnesium sulphate. *Senna* easily deranges digestion; a small amount may be added to prunes and both cooked together; *confectio sennæ* is given in dram doses. *Tobacco smoking, and coffee* in the morning, are beneficial, but tea promotes flatulency and constipation. Consideration of the salines and drastics is intentionally omitted, as their use except for emergencies is contra-indicated.

(P). FUNCTIONAL (NERVOUS) AFFECTIONS OF THE BOWEL.

1. Neuroses of Motility.—(*a*) *Nervous diarrhœa* has been partly considered. It may occur in the neuroses, in reflex irritation during dentition, or in the crises of tabes; it is usually transient and is marked by absence of inflammatory symptoms, as mucus, pus, or blood. There is constant danger that nervous diarrhœa may be confused with organic disease. In some cases ingestion of food may be immediately followed by diarrhœa, and is treated by giving ♏ij of Fowler's solution before meals. (*b*) *Enterospasm* is caused by simultaneous contraction of the circular and longitudinal muscles, which normally contract alternately; the abdomen is temporarily retracted, and in very exceptional cases obstruction (*ileus spasticus*) and fæcal vomiting occur. (See DYNAMIC ILEUS, SPASTIC CONSTIPATION and COLICA MUCOSA.) (*c*) *Sphincter spasm*, aside from local rectal or anal lesions, results from sensory hyperirritability, or decreased cerebral inhibition; it is most common in hysteria and tabes. (*d*) *Peristaltic unrest* may be associated with diarrhœa if the colon is involved, but more commonly affects the small bowel only, when constipation or normally frequent movements are usual. (*e*) *Nervous constipation* (atony) may result from the neuroses, intoxication from carbon dioxide, and organic cerebrospinal diseases, in which condition (*f*) *sphincter paralysis* may also result.

2. Neuroses of Sensation.—Among the neuroses of sensation, neuralgia mesenterica (*enteralgia*, enterodynia) is most important; it is inflammatory, nervous, or colicky in origin; strictly speaking, it is always nervous, *i. e.*, not due to organic causes; practically, other forms of abdominal pain are frequently included under enteralgia. The chief symptom is the abdominal pain, which is usually umbilical, and sometimes relieved by pressure and sometimes is not; the abdominal wall itself may be hyperæsthetic. Reflex disturbances in other organs may accompany enterodynia and aid in establishing its functional character; they are hiccough, vomiting, dyspnœa, palpitation, rectal or vesical tenesmus, testicular retraction, muscular twitchings, etc. The *diagnosis* necessitates exclusion of innumerable affections, chiefly abdominal but also general: (*a*) rheumatism of the abdominal muscles which is superficial; (*b*) lumbo-abdominal neuralgia, in which Valleix's three tender points are found (see NEURALGIA); (*c*) hysteria, which may simulate many affections; its stigmata should be looked for; (*d*) tabetic crises; (*e*) intestinal affections, as appendicitis, peritonitis, coprostasis, parasites, flatulent colic (from beer, milk, or spoiled food), excessive catharsis, lead colic, or colica mucosa; (*f*) *gout*, arthritis, or cold; (*g*) malaria and typhoid; (*h*) renal and biliary calculi.

Its *treatment* and that of the motor disturbances is of the fundamental nervous state. Symptomatically, hypodermics of narcotics produce the quickest but most dangerous cure.

A hypodermic injection of atropine is often helpful.

℞

Spiritus ammonii aromatici	ʒiij.
Tincturæ cardamomi compositæ	ʒiij.
Spiritus chloroformi	ʒij.
Tincturæ opii camphoratæ	ʒiv.
Tincturæ asafœtidæ	ʒiv.

M. et S.—One tablespoonful in hot water every fifteen minutes for three or four doses.

3. Neuroses of Secretion.—The secretion neuroses are important. The chief types are serous nervous diarrhœa and colica mucosa, which have already been considered.

(Q). AFFECTIONS OF THE MESENTERY.

I. Inflammation.—Mesenteritis is chiefly important in its relation to ascites, tuberculous peritonitis, and other peritonitides (*q. v.*).

II. Hemorrhage.—Hemorrhage is most rare as a primary manifestation. It is generally secondary to acute hemorrhagic pancreatitis, retroperitoneal hæmatoma, aneurysmal extravasation, or the hemorrhagic specific fevers.

III. Diseases of the Mesenteric Vessels.—(*a*) *Infarction* by embolism or thrombosis was first described by Virchow, and involves chiefly the arteria mesenterica superior, which is essentially a terminal vessel. Most cases develop in men past middle life. Jackson, Porter, and Quimby, have collected 225 cases. (See SYMPTOMS OF VALVULAR HEART DISEASE.) Its onset is sudden, with intense abdominal pain, collapse, nausea and vomiting (usually bloody and perhaps fæcal, due to intestinal obstruction), diarrhœa (40 per cent.), and bloody stools (40 per cent.). Unless there is an apparent cause for embolism, as (mitral) valvular disease, the diagnosis of infarction is rarely made, for peritonitis or obstruction is usually suspected. The usual outcome is early death from collapse, but sometimes, if the patient lives long enough, acute peritonitis sets in, with distended abdomen and diffuse tenderness. In very rare cases collateral circulation allows of recovery. In the Porter, Jackson, and Quimby series, 94 per cent. were fatal. The treatment is purely symptomatic and supportive, but successful operation is recorded by J. W. Elliott, Leclerc, and Buetter. (*b*) *Peri-arteritis nodosa* (*v.* DISEASES OF THE VESSELS). (*c*) *Dilatation* of the mesenteric veins and phlebosclerosis occur, especially in cases of liver cirrhosis. (*d*) *Suppuration* (thrombophlebitis suppurativa) may mark umbilical pyæmia of the new-born or may carry infection from the appendix or rectum. (See HÆMATEMESIS.) (*e*) Simple thrombosis of mesenteric veins. Kraft in 1897 collected 16 cases; in over half of the cases syphilis or liver cirrhosis was the cause.

IV. Affections of the Chyle Vessels.—These offer more pathological than clinical interest. These vessels may become varicose or even hyperplastic (chylangioma). Cysts containing chyle may cause tumors, and, by rupture, may result in chylous extravasation in the mesentery, or chylous ascites (*q.v.*). Twenty-four chylous cysts are described by Broca.

V. Mesenteric Tumors.—These may be dermoid, hydatid, serous, sanguineous, chylous, or malignant. In some cases they may attract only accidental pathological attention, or they may in other instances fill the entire abdomen. Their symptoms are those of an abdominal tumor, which is centrally located, is movable, and covered with a zone of resonant intestine. Confusion is possible with hydronephrosis or ovarian cysts.

DISEASES OF THE LIVER.

(A). ACUTE YELLOW ATROPHY.

Definition.—An acute and usually fatal degeneration of the liver cells, accompanied by shrinking of the liver, icterus, and nervous toxæmia. Ballonius (1600) reported the first case. It is a rare affection; Best collected 450 cases.

Etiology.—(*a*) Sixty-six per cent. occurs in *women* and 50 per cent. between the ages of *twenty and thirty years*. It is rarely observed in infancy or old age. (*b*) Thirty-three to 50 per cent of women with the disease (or 27 per cent. of all reported cases) are in the *last half of pregnancy;* the first three months of gestation almost render the individual immune (Eichhorst). It occurs less frequently in the puerperium. (*c*) It rarely occurs as an *epidemic;* 10 soldiers were affected in Arnould's series. (*d*) *Infections,* as osteomyelitis, diphtheria, sepsis, erysipelas, typhoid, recurrent fever, and secondary syphilis (10 per cent. Lebert); or (*e*) *poisoning,* as from phosphorus, alcohol, ptomaine poisoning, chloroform narcosis, and possibly bacterial poisons, may antedate the disease.

It may occur primarily in a sound or secondarily in a diseased liver (fatty or cirrhotic liver, or in bile stasis). Worry is thought to be a predisposing factor, caused, *e. g.,* by pregnancy or syphilis.

Pathology.—The liver is small, the minimum weight recorded being 400 grams, or about thirteen ounces; in marked cases it is so lax that it can be rolled up; the capsule is wrinkled, the color is usually yellow (*atrophia hepatis fusca*), and the lobular markings are obscure. The liver cells are granular, fatty, indistinct in contour, and some are tinged with bile; some are necrotic and others show evidences of regeneration. Foci of red tissue are observed; this is usually considered as an advanced stage in which the fatty necrotic cells have been absorbed; this tissue shows few or no cells, is homogeneous, and is composed of detritus; the left lobe is often red, showing the most advanced process, and the right lobe is yellow, showing the earliest changes. The cells of the bile channels are degenerated and sometimes appear to multiply. Crystals of tyrosin are found. The apparent increase of the connective tissue is relative only. Sometimes infiltration with red or white cells occurs. In other organs icterus staining, small hemorrhages, and fatty degeneration, are found, as in the heart, kidneys, muscles, epithelial cells, and glands of the bronchi, lungs, and digestive tract.

In *nature* the disease is sometimes considered inflammatory (acute parenchymatous hepatitis), but is usually classified as a degenerative or necrotic process. It is not yet determined whether the disease is an *infection,* to which it has a certain resemblance, or an *intoxication.* The changes in other organs are held to result from the liver change or from a common unknown cause. Bacteria are found in some cases, but their role has not yet been determined. Various microörganisms have been found at autopsy, as the pneumococcus, pyogenic organisms, colon bacillus, and other organisms, but not in all cases; Prochaska cultivated the streptococcus, from a case of Eichhorst, before death.

General Clinical Picture.—Acute yellow atrophy begins with a *prodromal stage* in which stomach disturbance predominates; icterus develops in a few days or a week, and the *second stage* then begins suddenly with cerebral symptoms of unrest, delirium, oppression, vomiting and convulsions; with shrinking of the liver, hepatic tenderness, enlarged spleen, cutaneous and mucous membrane hemorrhages, abortion and hemorrhage if pregnancy exist; with urinary symptoms, as bile, albumin, leucin, tyrosin, and other unusual products of metabolism in the urine; and with subnormal or normal temperature. The issue is almost invariably fatal.

Individual Symptoms.—The *prodromal stage* may last from a few days to three and a half weeks or more, and is not characteristic. In the latter half of gestation, icterus and hepatic tenderness are suspicious symptoms. The stomach symptoms and icterus become marked. Icterus, which is rarely absent except in extremely rapid cases, is obstructive from intrahepatic changes; the stools are light (acholic) and the jaundice increases with the second stage. Occasionally there is some prodromal swelling of the liver.

In the second stage, (*a*) the *hepatic dulness lessens* to one-half or one-quarter of its former dimensions, which first involves the left lobe. The liver is small, flabby, and falls back toward the spine so that in some cases there is no liver dulness. The liver dulness may remain normal, first, if death is rapid, as from hæmatemesis (Leube); second, if the liver is cirrhotic; or, third, if it is adherent to the abdominal wall. The liver is *tender and painful,* and pain is occasionally referred to other parts of the abdomen. The region of the liver may appear sunken and the liver may feel flabby or may pit to the finger (Leube). (*b*) The *spleen is enlarged* in 66 per cent.; it is not enlarged when perisplenitis, profuse hemorrhage, or diarrhœa, are present. (*c*) *Vomiting* is *almost invariable,* the vomitus consisting of mucus, bile, and finally blood; it is accompanied by hiccough, a dry brown or white tongue, sordes, and constipated, usually uncolored, stools. (*d*) The *nervous symptoms* may come on gradually with headache and dulness, or very abruptly with *delirium,* anxiety, *meningeal symptoms,* grinding of the teeth, trismus, wide pupils, amaurosis, cerebral vomiting, and convulsions (in 33 per cent. of adults and almost constantly in children). The nervous symptoms are due to hepatic insufficiency (hepatargia), the degenerated liver being unable to protect the nervous system, as it does in health, against various ptomaines and leukomaines. (*e*) The *urine is decreased* or even suppressed. *Bile pigment, albumin,* casts, and epithelial cells, are found. Unusual products are found in the urine, due to the breaking down (*autolysis*) *of the liver cells; leucin and tyrosin* occur most frequently, though they are neither constant nor pathognomonic; sarcolactic acid, oxyamygdalic acid, peptone, and albumose, are also found. The urea is decreased a may be entirely absent. The ammonium compounds, which norma constitute 2 to 5 per cent. of the nitrogen excretion, are increas even to 17 or 20 per cent., because they are needed to neutralize increased amount of acid products in the blood and because the li cells cannot convert the ammonium compounds into urea. The u acid and xanthin are increased. Indicanuria may be noted. Strang glycosuria is rare, though the glycogenic function of the liver c hardly be normal. Bouchard, Hervouet, Brouardel, and Chauffa speak of urinary crises with increase in the amount of urine voi and in its nitrogenous constituents. (*f*) *Fever* may occur early in disease, but the temperature is usually normal or subnormal at height of the process. The *pulse* may be at first slow, but later beco *very rapid*. *Dyspnœa* is almost invariably present. (*g*) *Hemorrha* occur in the majority of cases, largely from the stomach and into skin. Bowel hemorrhage is less frequent, as are those from the no mouth, and genital mucosæ. Uterine hemorrhages occur if pregna exist. Hemorrhages in the retina are frequent, together with wh flecks due to tyrosin deposit and fatty change. Punctate hemorrha into the serous membranes are common autopsy findings. Erythe atous eruptions, muscular pain, and articular swellings, may occ In a case seen by the writer herpes was the first symptom. Leuk cytosis is infrequent.

Course.—The clinical course is rapid; 50 per cent. of cases d between the fifth and fourteenth days, and 30 per cent. between t third and fifth week. The second stage usually lasts one and a h to three days, rarely more than a week, but may be longer in the ra cases which recover. The more abrupt and severe the cereb symptoms, the more rapid is the course. Pregnancy hastens the iss Remissions may be observed; temperature may develop during death agony. The pulse becomes very rapid, the breathing diffic and irregular, and the symptoms of nervous excitation are follow by paralysis of the brain centres.

Diagnosis.—The diagnosis is based upon (*a*) the *icterus*, (*b*) *nerv* *symptoms*, and (*c*) *shrinking of the liver*. Diminution of the liver m be simulated by tympanites or by the colon lying over the liver; may not shrink when previously cirrhotic or when bound to the abdo inal wall by adhesions, even though the liver cells atrophy. The uri shows distinct disturbances of metabolism, which are valuable dia nostically with the above mentioned symptoms. Confusion may occ in *icterus gravis*, or in the so-called bilious typhoid, pneumonia, rec rent fever, puerperal fever, yellow fever, phosphorous poisoning Weil's disease; most of these affections are febrile, while acute yell atrophy is almost always without fever; in addition to this, the intens of the icterus and shrinking of the liver, severe cerebral symptoms which periods of depression and exaltation alternate, and the urin findings, are diagnostic. The greatest difficulty of diagnosis is *acute phosphorous poisoning*, for which Quincke gives the follow

differentiation, though Leube maintains that differentiation is impossible, except etiologically.

Acute Yellow Atrophy —vs.—	Acute Phosphorous Poisoning.
One to two weeks of moderate symptoms from icterus.	Acute gastro-intestinal symptoms; then icterus on the third day.
Icterus intense.	Icterus less.
Liver sensitive, smaller. If colon is distended, entirely obliterating the liver dulness, the urinary symptoms are suggestive.	Liver sensitive and swollen.
Liver cells greatly degenerated, and but little increase in the fat (5 per cent.).	Very extensive fatty degeneration (30 per cent.).
Sudden cerebral symptoms with alternating irritation and depression one to two days before death.	Cerebral symptoms more of depressive order.
Urine; oxyamygdalic acid, leucin, tyrosin.	Sarcolactic acid (though peptone, sarcolactic and tyrosin are found in both diseases).
Hemorrhages: smaller and fewer.	Hemorrhages: more numerous.
Duration of one to two weeks.	Duration shorter.

Though admitting that subjects of acute yellow atrophy may recover, such recoveries usually indicate errors in diagnosis. From *simple atrophy* of the liver differentiation is easily made by the general marasmus and the absence of icterus and of nervous and urinary symptoms. *Cirrhosis* is differentiated by absence of ascites and the presence of a larger spleen, firmer liver, and collateral circulation (*v.* page 724).

Treatment.—Treatment is wholly expectant and entirely negative in its results. The heart depression, vomiting, and nervous excitement, are treated as in typhoid (*q.v.*).

(B). PORTAL CIRRHOSIS.

Synonyms.—Laennec's cirrhosis, alcoholic or atrophic cirrhosis.

Classification.—The classification of the varieties of cirrhosis is difficult; it may be made upon an etiological, pathological, or clinical basis. From a practical standpoint the chief points are included in the following classification:

(1). Capsular or Glissonian.	Portal vein syphilis. Chronic perihepatitis.
(2). Vascular.	*Portal vein* cirrhosis, first in clinical importance; its leading type is the alcoholic, atrophic type of Laennec. (A sub-type is a hypertrophic cirrhosis, like the atrophic in every respect save that the liver remains large.) *Hepatic vein* cirrhosis, occurring in stasis (cyanotic induration), and, according to French authors, with portal vein cirrhosis.
(3). Biliary Cirrhosis.	Hanot's disease.
(4). Mixed Cirrhosis.	(Portal vein and biliary cirrhoses combined.)

Of these forms, given in the hope of elucidating the nume classifications, two main forms are conspicuous—the portal and bili

Definition.—Portal cirrhosis is a fibrosis of the liver, character (*a*) etiologically by alcoholism; (*b*) anatomically, by induration aro the portal vein radicles and enclosing numbers of liver lobules, wh cells degenerate; and (*c*) clinically, by portal obstruction (dyspep hæmatemesis, enlarged spleen, and ascites), and often by signs of hep insufficiency.

Etiology.—(*a*) Sixty to 75 per cent. of cases are found in between *forty and fifty years* of age. Musser (1889) collected cases in children. (*b*) *Alcoholism* is the chief though not the s cause; cirrhosis may be due to whisky, wine, beer, or absinthe, especia in sedentary individuals; alcohol is absorbed by the gastric and intesti veins, and is conveyed to the portal vein radicles, where its to effects are expended on the liver cells or interstitial tissue, but in wh sequence it is not known. Of late the role of alcohol has been d puted, but it remains a striking though perhaps an indirect fact (*c*) *Syphilis* is a cause, especially in the young, in whom the tox are directly conveyed, by the umbilical vein, to the liver. (*d*) *Ot infections,* as malaria (especially where it is endemic), cholera, typho tuberculosis, scarlet fever, measles, and diphtheria, are possible caus experimentally cirrhosis has been caused by the Bacillus pyocyane Bacillus coli, and staphylococcus; focal mycotic necrosis is thoug to be followed by fibrosis; in this group the toxins probably enter the hepatic artery. (*e*) *Spices,* curry, drastics, lead, phosphor arsenic, hæmochromatosis, anthracosis (Welch), silicosis (Adami), c tain shell-fish, and possibly auto-intoxication from dyspepsia, are exc tional etiological factors. It may be associated with gout, diabet rickets, or other constitutional affections. Experimental cirrhosis h been produced by lactic, acetic, and butyric acid. There may *multiple causes, e.g.,* syphilis plus malaria or alcoholism. In so cases no cause is discoverable.

Pathology.—The essential change is fibrosis around the branc of the portal vein. Some consider that the connective tissue growth primary, causing, as it contracts, atrophic or fatty alteration of t liver cells, sometimes with pigmentation; others maintain that t cells are first injured and are replaced by fibrous tissue. Whatev the pathogenesis, the connective tissue forms and surrounds sever lobules at a time (*multilobular cirrhosis*). Histologically the cirrhos is *atrophic,* and this point is emphasized first, because the size a weight of the liver may vary. Some clinicians maintain that the liv is first large (the so-called hypertrophic stage) and later is shrunken Undue importance is attributed to size alone, for a normal-sized o even enlarged cirrhotic liver is histologically atrophic. An actuall atrophic liver may be large from (*a*) fat deposit, (*b*) passive congestio (*c*) compensatory hyperplasia, or (*d*) active congestion. The *weigh* is *usually decreased* from 1,500 or 2,000 gm. (50 or 65 ounces) to 1,00 gm. or less. Its *edge is rough; i.e.,* granulations can be felt whic represent the remaining relatively normal liver tissue. The *surface* is sometimes smooth, and microscopic examination may be necessary for confirmation. Granulations also occur in fatty liver, pylephlebitis, and passive congestion. Its *consistence is increased.* The liver is *gritty* to the knife; its color is yellow (cirrhosis), perhaps green or brown. The *connective tissue is increased* and prominent, encroaching on the parenchyma. Injections of the organ through the portal vein are not successful; *i.e.*, there is portal obstruction, which causes many of the clinical symptoms. No connection between the portal vein and the hepatic artery can be demonstrated, though injections are successful through the hepatic artery and the biliary system.

General Clinical Picture.—The disease often exists for several years without symptoms; this latency is usually marked by enlargement of the liver, which is due to compensatory hyperplasia. In the minority of cases, swelling, pain, or active hyperæmia of the liver, dyspepsia, fever, or icterus, are observed; they last a short time only and are by no means characteristic. With marked shrinking there are symptoms (*a*) of general *disturbance of nutrition,* as constipation, emaciation (which is often not noticed because the abdomen remains obese), and an earthy, sallow look to the skin, in which dilatation of the small arterioles (toxæmia or alcoholism), dryness, and hemorrhages, are also observed; (*b*) of *portal congestion,* as early hæmatemesis, epigastric pressure, ascites, small liver, large spleen, and disturbed breathing, circulation, and digestion; (*c*) of *intoxication,* resulting from functional failure of the heart, liver, and kidneys. (*d*) There may be *complications,* as hemorrhage, diarrhœa or the so-called cholæmia.

Symptoms.—1. The Liver.—The liver may be enlarged at first; it is enlarged about *as often as it is decreased in size;* the left lobe suffers most and earliest contraction. The liver may be found to be high, as a result of paresis of the diaphragm, due to perihepatitis. Its *edge is hard,* but often cannot be felt, even after tapping, on account of its shrinkage or because the bowels cover it. The *edge is uneven,* but its granulations may be confused with the small islets of the fatty tissue in the abdominal wall, which are due to unequal disappearance of the adipose tissue as a result of emaciation. *Palpation* of the liver is *the only certain way* of determining its lower limit, the results of percussion being decidedly unreliable. In infantile forms of portal cirrhosis the liver and spleen are larger than in adult types.

2. Portal Stasis.—This results from obstruction to the return portal circulation. (*a*) The peritoneum is thickened, œdematous, and the seat of transudation, *i.e., ascites,* which occurs in 85 per cent. when the patient dies of the cirrhosis itself and in 50 per cent. when he dies from other causes. It is more common in small than in large livers. Some writers hold that toxæmia and peritonitis are more potent factors than portal obstruction. *Ascites* usually *appears first without general œdema,* though later the legs may become swollen as the ascites compresses the inferior vena cava. Pre-ascitic œdema of the legs or abdominal wall may develop from cardiac or renal complications, involvement of the cava by perihepatitis, or from thrombosis of the iliac veins. Under ascites, its physical signs will be more closely considered. The amount of fluid may be enormous; Liebermeister reports a case in which he withdrew nine gallons.

The fluid is usually amber-colored, seldom red from admixture of blood, or very rarely turbid from fat (adipose ascites) or lecithin (chyliform ascites); the two latter forms also contain nucleo-albumin. The *specific gravity* is 1,008 to 1,015, with 0.6 to 1 per cent. albumin. Though ascites is rather rarely absent in advanced cases of atrophic cirrhosis, the patient may die before ascites develops—for instance, from hemorrhage, which is dangerous when there is no ascites (Leyden). An extensive though slowly developing *collateral circulation* may permanently relieve an earlier ascites. It is not an infallible sequence of atrophic cirrhosis (Lecorché, Hanot), and may disappear after hemorrhage (Fauvel), diarrhœa (Linas), or carcinoma of œsophagus (Lecorché, Telamon). (*b*) *The spleen* is from two to six times its normal size, which is due to the early toxæmia and the later stasis. The *splenic tumor is present in* 75 per cent. of cases and is a measure of the degree of cirrhosis. Its capsule is hard and thickened (sometimes preventing splenic enlargement), the trabeculæ are thicker, and a systolic murmur is sometimes heard (Bouchard and Leudet). (*c*) *Gastro-intestinal catarrh* is due to stasis and to alcoholism. Pain after eating, especially in obese persons, a coated tongue, vomiting, singultus, constipation, piles, diarrhœa (which is rare unless toward the end of the course), and meteorism, also occur. (*d*) *A collateral circulation* may be evidenced by enlargement of the veins about the navel (*cirsomphalos* or *caput Medusæ*). Prominence of the epigastric veins occurs regularly, but results from compression of the inferior cava. *Anastomoses* occur (i) between the median and inferior hemorrhoidal veins (passing by the hypogastric to the cava) on the one hand, and the superior hemorrhoidal (passing by the superior mesenteric to the portal vein) on the other hand; (ii) between the coronary veins of the stomach on the one hand, and the œsophageal and phrenic veins on the other, which empty into the vena azygos; (iii) between the veins in the ligaments of the liver and new adhesions and the phrenic veins; between the veins of the *ligamentum teres* and the veins of the abdominal wall and epigastric veins, which may sometimes be felt and may produce murmurs (Bamberger and von Jaksch); (iv) the mesenteric, peritoneal and parumbilical veins also communicate with those of the abdominal walls. A well-developed collateral circulation is both a favorable and an unfavorable symptom; on the one hand it *relieves* the *portal stagnation* and probably gives the over-taxed liver a better opportunity to perform its functions; on the other hand, the dilated collateral vessels are likely to *rupture* and they convey directly into the general circulation *toxic substances* which the liver normally destroys or alters; this produces, according to Stockton, the frequent arteriosclerosis, granular kidneys, and more acute toxæmia, which resembles uræmia. (*e*) *The kidneys are congested*, degenerated, and often show hypertrophy. The urine is decreased in amount, darker, and of higher specific gravity, because the arterial tension is low in cirrhosis. More urine is often secreted during fasting than during digestion (opsiuria), which is the converse of normal conditions. The *urobilin may be increased* two-fold, giving a fluorescent filtrate when equal parts of urine and a 10 per cent. solution of zinc acetate in absolute alcohol are mixed and filtered. The chlorides are decreased, as they accumulate in considerable amounts in the ascitic transudate. The urea is often decreased, but the *ammonium compounds* (as in acute yellow atrophy) are often increased, less from interference with the liver functions than from increase of acid products in the blood (acidosis), which the ammonium salts seek to neutralize. *Albuminuria* may result from stasis or nephritis (*v. i.*). Blood may be found in the urine, as a result of stasis in the bladder. Glycosuria is uncommon save in the pigmentary cirrhosis (see DIAGNOSIS). Alimentary glycosuria may be produced by administration of 100 gm. (3 ounces) of grape sugar with a meal.

3. **General Symptoms.**—There is considerable *loss in weight*, some *anæmia*, and a sallow, *earth-like color*. Great malnutrition is readily explained (*a*) by the inability of the liver to properly handle the carbohydrates, proteids, etc., and (*b*) by the diversion through the collateral circulation of toxins into the general circulation, because the liver is the normal barrier against toxins which are injurious to the nervous system. In a recent case with well-developed collateral circulation, scurvy developed which the writer interpreted as toxæmic. It was once thought that bile salts or pigment entered the blood and produced "*cholæmia*," or that substances were retained which the bile normally excreted, producing "*acholia*"; now the term *liver insufficiency* (*hepatargia*) is more often employed. Toxæmic nervous symptoms include adynamia, headache, itching, delirium, stupor, convulsions, paralysis, contractures, or coma. Much depends upon the activity and integrity of the kidneys, which may for a time maintain adequate excretion.

Complications of Cirrhosis.—1. **Hemorrhage.**—(*a*) *Cachectic* capillary hemorrhage from the nose, mouth, lungs, or stomach, is less frequent than it is in biliary cirrhosis. Epistaxis occurs from dilatation of the vessels on the anterior part of the septum. (*b*) Of *mechanical* congestive hemorrhages from the œsophagus, stomach, bowels, kidneys, bladder, or uterus, those from the *œsophagus* are most important. Zenker found œsophageal varices in 20 per cent. of cirrhosis autopsies. Their rupture causes *hæmatemesis* (which occurs in 23 per cent. of cirrhosis cases), chiefly in adults and rarely in children; if they are very profuse, blood may come up without vomiting. It is usually an early symptom, antedating ascites; the amount is generally profuse, and in one of Osler's cases amounted to 10 pounds; it often causes blood in the stools. It may pour out slowly and form dark clots; it is usually attended by less collapse than is hemorrhage from ulcer, but acute anæmia or slight fever may follow it. Preble in a careful study of the subject concludes that: (i) Hemorrhage is not a rare com-

plication of cirrhosis. (ii) It occurs chiefly in atrophic forms. (
In 33 per cent. the first hemorrhage is fatal; in 66 per cent. hem
rhages occur over a period of several months to years, eleven ye
being the maximum. (iv) In 33 per cent. a diagnosis of cirrhosis w
possible at the time of the first hemorrhage. (v) Œsophageal varic
were present in 80 per cent., and in over half of these macroscop
rupture was detected; yet (vi) fatal hemorrhage may occur witho
macroscopic rupture, and is due to a simultaneous rupture of ma
capillaries. (vii) Hemorrhage may be the first sign of cirrhosis, b
other symptoms are usually found first. (viii) The symptoms
cirrhosis were typical in but 6 per cent. of cases of œsophageal vari

2. **Peritonitis.**—(*a*) *Chronic retracting* peritonitis (in 10 to 19 p
cent., Gratia), may occur with shortening of the mesentery and inte
tines; according to Botazzi it prevents absorption of the ascites. (
Tuberculous peritonitis (10 to 25 per cent.) may occur with fever, pai
and often a hemorrhagic exudate which produces tuberculosis o
inoculation. It usually follows the ascites of cirrhosis (the conver
being very rare) and hastens the fatal outcome. One would thin
that stasis would tend to prevent the development of tuberculosi

3. **Nephritis.**—Nephritis occurs in 33 per cent. of cases.

4. **Heart.**—The heart is arteriosclerotic in 60 per cent.; chron
myocarditis, endocarditis, fatty degeneration, and dilatation of th
right ventricle, often occur. The arterial tension is low in cirrhosis.

5. **Lungs.**—In the lungs, œdema, catarrh, emphysema, various i
flammations, and very frequently pulmonary tuberculosis occur; th
latter is rare in infantile cirrhosis. Right hydrothorax occurs fro
stasis in the vena azygos and in the right intercostal veins; righ
sided pleurisy, due to extension of perihepatitis through the diaphrag
occurs in 10 per cent. of cases (Caro).

6. **Brain.**—Pachymeningitis, meningitis, or brain hemorrhage, ma
occasionally occur.

7. **Fever.**—"*Intermittent hepatic fever*" may result from hepatargi
(hepatic intoxication), perihepatitis, or from complications. The temperature is usually subnormal in advanced cases, though the author has often observed slight terminal temperature and leukocytosis, which is referable to terminal infections.

8. **Icterus.**—In atrophic cirrhosis jaundice is a genuine complication; it is due to duodenal catarrh, enlarged portal glands, or a diffuse cirrhotic process (Andral), though few patients pass through the disease without having a muddy yellow areola under the eyes. If present it is incomplete, and is caused by compression of bile ducts by contracting connective tissue. Its frequency is 15 to 20 per cent.

9. **Anasarca.**—Anasarca results from pressure on the cava by an extensive ascites, from perihepatitis with cicatricial involvement of the cava, and from complicating cardiac or renal disease and marasmus.

10. **Other Complications.**—These are rarer; for instance, pylethrombosis, clubbed fingers, alcoholic multiple neuritis, or delirium tremens; acute yellow atrophy and venous thrombosis seldom occur.

Course and Prognosis.—The clinical course is essentially chronic; it usually lasts not more than two or three years, but its duration, after the diagnosis is once made, is more often months than years, though a ten to fifteen year course has been observed. Following Stricker, Hanot, and Lenhartz, the author excludes all acute cases from the category of cirrhosis. *Compensatory factors* include (*a*) the collateral circulation, which may throw toxins directly on the nervous centres and produce symptoms like those of uræmia; (*b*) hyperplasia of the liver cells, which may produce veritable adenomata. As Hanot puts it, the diagnosis is based on the condition of the connective tissue, but the prognosis depends on that of the liver cells. (*c*) The kidneys may compensate to a small degree. Hale White holds that ascites is ominous and that the patient rarely survives the second tapping; this does not conform to the author's experience, though ascites is a late symptom; the author has seen numerous cases which survived frequent tappings, and he has felt justified in excluding perihepatitis, which White considers explanatory of these cases. The prognosis is relatively favorable in subjects under forty years with large livers and small spleens. Recovery after a positive diagnosis has been made, is possible, especially if the diagnosis is made early. In advanced stages death is the usual outcome, although the author has seen relative recoveries lasting for five or six years follow adhesions from multiple paracentesis.

Death results from hepatargia (cholæmia, acholia), marasmus, hemorrhage, ascites, intercurrent pneumonia, or other terminal infections; exhausting diarrhœa or vomiting; cardiac, renal, or other complications (*v. s.*).

Differential Diagnosis.—Two classes of conditions require differentiation: on the one hand, those in which the liver is large; on the other, those in which the liver is small.

1. When the Liver is Large.—(*a*) The *fatty liver* is soft and presents no evidences of portal stasis. (To avoid repetition the diseases are differentiated later, in tabular form [see page 724].) (*b*) The *amyloid liver* is hard; the spleen, kidneys, and alimentary mucosa, are almost always involved, icterus is very rare, and occurs only when amyloid glands at the *porta hepatis* produce mechanical icterus; anæmia is common. (*c*) *Leukæmic* deposits in the liver cause hepatic intumescence, but the blood examination is decisive. (*d*) Some swelling of the liver may result from *simple icterus*, but the history, absence of portal stasis, and shorter course, are distinctive. (*e*) *Passive hyperæmia* from heart or lung disease produces an evenly enlarged tender liver, subicterus, and cyanosis. (*f*) Cancer of the liver is usually clearly marked by hard and nodose enlargement, by detection of the primary growth (*e.g.*, in the stomach), by icterus, and cachexia; in some cases early cirrhosis may simulate cancer, as in a patient whom Kahler saw and diagnosticated cancer, though the later course proved that cirrhosis was present. (*g*) In *syphilis* of the liver the history, knobs, and depressions, the more rapid course, greater perihepatitic pain, the icterus (in 33 per cent.), the findings in other parts, and the results of treatment, usually make the diagnosis clear (see SYPHILIS). (*h*) *Pericarditic pseudocirrhosis*, described by Pick (1896), occurs in young, rheumatic, and cardiac subjects, and is characterized by symptoms which on the one hand resemble extreme passive congestion of the liver, and on the other a capsular cirrhosis; the liver is large and firm, there is recurrent ascites without œdema of the extremities, and little or no icterus. Its mechanism is disputed; contracting pericarditic adhesions may perhaps cause dilatation of the right auricle and vena cava inferior, thereby distending the hepatic vein system; or possibly the inflammation extends down the cava inferior to the liver; the liver, however, is permanently and disproportionately congested. (*i*) The *enlarged liver in Banti's disease* is cirrhotic, but the spleen is enlarged first and there is early splenic anæmia while the cirrhosis and hæmatemesis occur later.

2. When the Liver is Small.—The following possibilities should be considered: (*a*) *Syphilis*, characterized by stellate scars, nodes, and pain. (*b*) *Passive congestion;* the nutmeg liver may atrophy, but anasarca is observed first and usually there are primary and sufficiently characteristic heart findings. (*c*) Simple *marantic atrophy* is of little clinical interest; the liver is small, there is no portal stasis, and arteriosclerosis is found in other organs. (*d*) *Pylethrombosis* is characterized by a rapid onset with swelling of the spleen, severe or repeated hæmatemesis, dilated veins, and rapid recurrence of the ascites after tapping; it is due to some special cause, as pressure of a tumor, nearby ulcers, suppuration, or gall-stones. (*e*) Perihepatitis has a very much slower course than has cirrhosis; it lasts for years with intermissions, and very often results from pericarditis. (*f*) In *chronic peritonitis* the fluid is turbid, often hemorrhagic, with high specific gravity and many leukocytes; it is characterized by absence of symptoms of portal vein stasis; there may be pain, fever, and, in the tuberculous form, positive results from inoculations; the differentiation of chronic peritonitis is considered in detail under ASCITES. (*g*) The peculiar *pigmentary cirrhosis* of hæmochromatosis (diabète bronzé) was first described by Troisier (1871), was named by Recklinghausen, and was particularly elaborated by the French school (Hanot, Chauffard). Its symptoms are three-fold: (i) cirrhosis (which causes the hemorrhages, indurated liver, and enlarged spleen), (ii) diabetes, and (iii) pigmentation of the skin, which somewhat resembles Addison's disease. It is thought that some toxic substance destroys the red blood cells (hæmolysis), and so operates on the cells of the liver, pancreas, and other organs, that they transform the soluble blood pigment into insoluble hæmosiderin. This pigment and an iron-free hæmofuscin are found in the muscles of the intestine, heart, liver, and pancreas, whose cells become pigmented, degenerated, and necrotic; the pigment passes into the connective tissue of the part. The essential lesion is pigmentation of the liver, skin, and pancreas, with the fibrosis of the liver and of the islands of Langerhans in the pancreas, which latter causes the diabetes. Diabetes has occurred in all but five of the French cases, but seems less common in those reported in this country. In Letulle's series of 30, 29 were males between forty and fifty years of age.

Treatment.—1. Causal Therapy and Prophylaxis.—These have but limited applications, save in preventing further injury to the affected liver by syphilis and by use of alcohol. All forms of liquors or medicinal tinctures are absolutely interdicted, but too frequently with no effect. Syphilitic forms are greatly benefited by appropriate treatment, which some clinicians think may help all forms of cirrhosis.

2. Medicinal Treatment.—This is of little value, for cirrhosis cases come under observation only after contracting connective tissue has already formed. Naunyn has used ten grains of ammonium chloride three times daily, but it does not arrest cicatrization. Rolleston believes that potassium iodide and ammonium chloride are beneficial in acid intoxication (acidosis); he never gives arsenic and salicylates.

3. Diet.—The diet should exclude rich proteids, for meat is less well tolerated than milk. Skimmed milk is best for it contains but 1 per cent. of fat. Rich or highly seasoned foods are to be avoided.

4. Symptomatic Treatment.—The same treatment which is instituted in chronic gastritis or alcoholism (*q. v.*) may be indicated. Flatulence should be met by calomel in fractional doses, then by salol 10 grains after each meal, or possibly by beta-naphthol 10 grains, though it may cause renal irritation. Diarrhœa should not be checked, at least not until the bowels have been thoroughly cleansed, for it is eliminative. Unusual splenic intumescence may be treated by purging, for it is often a forerunner of hæmatemesis. Hæmatemesis should be treated by absolute rest and by rectal feeding, as in gastric ulcer (*q. v.*); calcium chloride ʒss to j may be given by rectum for two or three doses. Pain is controlled by application of heat locally, by calomel, ammonium chloride, and by salines. Nervous symptoms necessitate the same treatment as uræmia; namely, laxatives, sweats, and transfusions to which sodium bicarbonate may be added as it is in diabetes, for the acidosis.

5. Ascites.—Ascites is seldom benefited by the use of cardiants, sweats, diuretics, or purges. Early repeated punctures (see ASCITES) are beneficial, and may induce adhesions and consequent collateral circulation.

6. Operation.—Talma and Van der Meulen (1889) recommended operative measures, which were later amplified in various ways and induce a collateral circulation; these consist of sewing the omentum to the abdominal wall, rubbing the surface of the liver until it is abraded to promote adhesions, and other similar measures. In the series collected by Harris, Greenough, and Allyn, amounting (in 1903) to 125 cases, about 40 per cent. were improved, and about 90 per cent. were alive and in improved health two years after the operation. Montprofit found in 224 operations, 30 per cent. cures. Bunge collected (1905) 274 operations of the Talma-Drummond type; the immediate mortality of the operation

3.6 per cent.; of the surviving cases, 14 per cent. were some- improved, 30 per cent. recovered, and 56 per cent. were not oved. Perhaps better results will be recorded if operation is rted to early in the disease, but it must be recalled that the ascites l collateral circulation involve vital as well as mechanical considera- ns. How the operation is beneficial is still a matter of doubt; its echanical collateral side is known; perhaps lessening of venous nsion in the portal circuit helps the liver cells to a better functional ctivity.

(C). BILIARY CIRRHOSIS.

Definition.—A cirrhosis occurring chiefly in adolescence, not due to alcoholism, and characterized by fibrosis of a mono- and intralobular type, by enlargement of the liver without contraction, by splenic enlargement, by chronic icterus without gross obstruction of the bile vessels, by paroxysmal fever, and by absence of portal stasis. It is also known as *Hanot's disease*, for it was first clearly described by him, in 1876. The term hypertrophic cirrhosis is unfortunate and ambiguous.

Etiology.—Its etiology is obscure. Alcoholism is not a factor. Sometimes it apparently results from (*a*) *infection*, either an ascending *cholangitis* due to the Bacillus coli or pneumococcus, or an infection through the blood stream. (*b*) *Age*. Most cases develop between the twentieth and thirtieth years of age; it is rare in persons over forty; a considerable percentage occurs in the young, especially in residents of India (Gibbons, Ghoses). (*c*) *Sex*. Over 80 per cent. of adult cases are *males*; in the young the sexes are equally involved.

Symptoms.—The onset is usually characterized by the early development of icterus and less often by malaise, muscular asthenia, fever, splenic pain, dyspepsia, diarrhœa, or abdominal pain.

Cardinal Symptoms.—1. The *liver* is *uniformly enlarged*, and may weigh five to ten pounds (2,000 to 4,000 gm.). Its surface is smooth and its edge is fairly even, though somewhat blunt. Its pathological characters will be considered under diagnosis. Its dulness extends from the fourth rib in the nipple line to the umbilicus or even to the iliac crest; it visibly bows outward the right costal arch; and possibly by its weight drags down the right shoulder. The gall-bladder is not enlarged for there is no obstruction of the large bile-ducts. The liver progressively increases in size, often paroxysmally with fever, pain over the liver, and increase of the icterus. It never shrinks, except when combined with portal cirrhosis.

2. *Icterus develops early*, and is *constant*, *permanent*, *and characteristic*. It varies in degree though it is usually quite intense. It is due to obstruction of the smaller bile vessels; it is not certain that it is due to over-secretion of bile (polycholia) or of bile pigments (polychromia). In extreme cases the skin becomes green or brown. It is frequently accompanied by itching, xanthelasma, eczema, and lichen

3. The *spleen is hypertrophied,* is usually increased relatively more than the liver, averaging one to two pounds (400 to 1,000 gm.) in weight but sometimes actually outweighs it (2,600 gm.), especially in infantile forms. It is hard, smooth, and evenly increased in all its elements and dimensions. It is often clearly visible and is distinctly palpable.

Other Symptoms.—*Digestive.*—The condition of the tongue and of the appetite is variable. Nausea, vomiting, dyspepsia, and tympanites, are uncommon. Diarrhœa is frequent. The normal or diarrhœal movements contain bile (in over 90 per cent. of cases).

Nutritive.—Nutrition is often maintained for years, despite the icterus, but wasting subsequently appears. Development is retarded in young subjects, and infantilism may be as marked as in cretinism or congenital syphilis. Clubbed fingers, from hyperplasia of the soft tissues, is not an uncommon development; Gilbert and Lereboullet collected 40 cases in 1901.

Urinary.—The urine is highly colored, bile-stained, and is usually increased in amount; its freezing point is high and it may contain urobilin, indican, or albumin.

Circulatory.—The pulse is not slow; the heart is often dilated and may reveal a systolic murmur; dyspnœa is frequent, and results from pressure on the diaphragm by the swollen liver and spleen; the blood shows secondary anæmia, decreased coagulability, and sometimes moderate leukocytosis. Arthritis and multiple neuritis are rare complications.

Diagnosis.—The following table shows the essential pathological and clinical points of contrast with the portal type:

PORTAL (ATROPHIC) CIRRHOSIS.——*vs.*	——BILIARY (HYPERTROPHIC) CIRRHOSIS.
1. *Etiology:* alcoholism; over forty years of age.	Infection; under forty years.
2. *Liver,* large or small (possibly both in succession); shrinks often.	Always large and to greater degree. Never shrinks in pure forms.
Granulations: larger.	Small; very fine.
Yellow.	Dark, icteric, green.
3. *Connective tissue* and inflammation ensnaring several lobules (multilobular or annular), and around portal vein radicles.	Begins in lobule (monolobular or insular) around smaller bile radicles (cholangitis) in early stages at least, later less clear.
Distinction between connective tissue and lobules sharp.	Not sharp.
Connective tissue, firmer, more fibrous; more elastic tissue.	More delicate, reticular, richer in cells, embryonal.
Scar-like and contracts.	Does not contract (elephantiasis hepatis).
4. *Liver-cells,* early fatty, degenerate, disappear.	Seldom fatty, remain normal for long time, may regenerate, more pigmented.

(Concluded on page 706.)

PORTAL (ATROPHIC) CIRRHOSIS.——vs.	——BILIARY (HYPERTROPHIC) CIRRHOSIS.
5. *Portal system* cannot be injected by portal vein.	Easily injected.
6. *Bile vessels*. (Proliferating bile vessels in biliary form are not characteristic, and are probably merely compressed rows of liver cells.)	
7. *Icterus* infrequent, a complication; when present, usually catarrhal.	Constant throughout course, and still the stools contain bile.
8. *Ascites* rarely absent in advanced cases.	Absent in all (except mixed forms).
9. *Collateral* circulation—usual.	Never.
10. *Hæmatemesis*, frequent, early, mechanical.	Very rare, late, cachectic, or toxemic.
11. Constipation usual.	Diarrhœa quite frequent.
12. Duration of two to three years.	Five to ten years.
Death results from hæmatemesis, nephritis, tuberculosis, peritonitis.	Rare; death from hepatargia (so-called cholæmia) with fever, nervous symptoms, delirium, typhoid state, coma.

It is a rare affection, and a diagnosis of biliary cirrhosis is too often made. If the cardinal features under its definition are borne in mind, it can usually be separated with ease from the following affections: (*a*) In *hepatic cancer,* the subject is older and more cachectic, the course is shorter, there is no splenic tumor, the primary growth *e.g.*, in the stomach, may be recognized, the liver often presents irregularities, the neoplastic obstruction of the bile duct produces acholic stools, and obstruction of the portal vein causes ascites. (*b*) Multilocular *echinococcus* (*q.v.*) also produces icterus, and enlarged liver and spleen, but is exceedingly uncommon. (*c*) *Fatty and amyloid* liver are unattended by jaundice, and in amyloidosis the enlargement of liver and spleen is usually associated with albuminuria, diarrhœa, œdema, and cachexia. (*d*) *Obstruction* of *the bile ducts* may produce an enlarged gall-bladder (*v.* ICTERUS); the stools are usually but not necessarily acholic (*e. g.*, in stone of the common duct). The spleen is not large. In this connection it may be stated that some recognize a (so-called) biliary cirrhosis due to chronic obstruction of the duct, as described by Charcot and Gombault in 1876; Osler and Hale White properly hold that simple mechanical obstruction never causes fibrosis, though it may follow ascending cholangitis. The liver is far less large in this obstructive type and is seldom hard. (*e*) In hæmochromatosis and pigmentary cirrhosis, icterus is absent and glycosuria is present in about 85 per cent. of cases. (*f*) For *Banti's disease*, see *diagnosis of portal cirrhosis*. (See page 724.)

Treatment.—The treatment is that of portal cirrhosis; no means are known which stop the formation of connective tissue. A generous diet may be given. Cold and dampness are especially to be avoided. Calomel in small doses (grain $\frac{1}{80}$ to $\frac{1}{40}$ *t. i. d.*) may be given for weeks a time, and salol, the salicylates, and simple salines, are some- es beneficial. Operative relief is suggested by drainage of the gall- dder, but in the reported cases the true biliary cirrhosis is often fused with obstructive icterus: operation might be helpful in ary cirrhosis due to ascending infections.

(D). ABSCESS OF THE LIVER.

uppurative hepatitis may be considered as a local affection, though sometimes a part of septicopyæmia (*q. v.*). Virchow found it in 1.5 cent. of autopsies. Ninety-seven per cent. occurs in males.

arieties.—1. In the *septic type,* 53 to 55 per cent. of cases (Bären- ing and Luda) the hepatic artery is the usual carrier of infection; primary focus is usually in the external parts, especially the skull ppocrates). It may result from osteomyelitis, external inflam- tions, pulmonary abscess or gangrene, putrid bronchitis, and, less n, from ulcerative endocarditis. Liver abscess occurs in 15 per cent. ases of surgical septicopyæmia (Bärensprung). Infected thrombi the peripheral veins may become detached, infarct the lungs, and nce reach the arterial circulation. In rare cases infective material y fall back from the cava into the hepatic vein; this is known as rograde embolism. The bacteria are those enumerated under sis—the pyogenic organisms, pneumococcus, colon, and typhoid cillus.

The *symptoms* of septic abscess are not distinctive because, first, they e obscured by the general toxæmia, and, second, the multiple abscesses en scattered through the liver usually produce few decisive local mptoms. The liver enlargement is uniform and is generally indis- guishable from the cloudy swelling of sepsis; fluctuation is rare; the rtal circulation is rarely obstructed; pain and tenderness are usually esent; jaundice is inconstant; the spleen is swollen from sepsis; the urse is rapid. The septic type may occasionally, and the traumatic ten, consist of a single large abscess.

2. The *second variety* results from infection through the tributaries the *portal vein* (31 per cent.) (Kelsch contends that 85 per cent. of 500 er abscesses were due to dysentery); it has been known since Mor- gni's time and is clinically the most important type. Any ulcerative suppurative affection in parts containing these branches may be usal, for instance, amœbic dysentery (the most frequent cause), pendicitis (in 6 per cent. of its fatal cases), neglected hemorrhoids, stric ulcer or cancer, and disease of the bile vessels, the vessels of hich are portal tributaries. Pyogenic organisms, the Amœba dysen- riæ, foreign bodies (as fish-bones, pins, liver flukes, and parasites), may ter by the portal circulation. Pus organisms may enter and infect berculous, echinococcic, or actinomycotic foci, of which latter 40 cases e recorded. The form of lesion varies; (*a*) there may be the *solitary* or pical *liver abscess,* which will be considered separately, or (*b*) there

may be suppurative pylephlebitis, which is less a suppuration of the liver substance than an extension of inflammation in the portal vein itself, and follows in a dendritic fashion through all of its intrahepatic ramifications; its branches are distended and present small accumulations of pus, which may appear as yellowish foci through the capsule, but show more clearly on section as portal vein suppuration; the whole liver may be involved, or certain areas may be exempted by total thrombotic occlusion of the larger branches. In some cases the purulent thrombophlebitis may be traced back to the original lesion in the stomach or intestine; the liver is enlarged but the diagnosis is rarely more than a probable one.

3. The *third variety* is infection by the *bile vessels* (10 per cent.), in which an ascending cholangitis (*q.v.*), often calculous in origin, produces multiple small abscesses through the liver substance; this is rather similar in appearance to the pylephlebitic form. These three varieties account for 94 to 96 per cent. of cases of liver abscess.

4. The *fourth variety* is infection in infants through the *navel*, and is an infrequent occurrence.

5. The *fifth variety* is direct *involvement* of the liver *by contiguity*, which may occur in various diseases, as in suppuration of the gall-bladder or ulcer of the stomach.

Solitary or Tropical Abscess.

The abscess is solitary in 75 per cent. of cases; in 11 per cent. there are two, and in 14 per cent. more than two, foci. Twenty to 30 per cent. of cases of amœbic dysentery, by far the most common cause, is complicated by liver abscess. Amœbic abscess may even occur without intestinal ulceration, though amœbæ are found in the stools; in 5 out of 27 of Futcher's cases there were no intestinal symptoms. Over 50 per cent. of tropical abscesses operated on in England by Manson contained amœbæ. Shiga's bacillary dysentery is rarely a cause. Single abscess may occasionally occur from trauma, pyæmia, typhoid (probably pyæmic), or in hydatid cysts. It occurs chiefly in the tropics, though it is seen in the Southern states and sometimes in the North. In the tropics it is thirty-five times as frequent in Europeans as in the natives; alcoholism and a rich diet are particularly predisposing factors; 97 per cent. occurs in males and 50 per cent. in those between twenty and thirty years of age.

Pathology.—Amœbæ reach the liver by the portal vein, and cause necrosis by their toxins; this is followed by suppuration, which may cause single or multiple foci. The smaller and sometimes the miliary foci contain glairy, gelatinous, translucent fluid; the larger foci contain white, yellow, green, reddish (like anchovy sauce, Budd), or chocolate-colored fluid, which may be serous, bloody, or viscid. Lafleur and Councilman consider that the absence of leukocytes in the wall and contents is pathognomonic of amœbic abscess. Red disks and necrotic liver cells are frequent. As Kartulis pointed out, the pus is usually sterile. The amœbæ are more abundant in the abscess wall than in its contents, and may be found in the vessels outside of the abscess. In contrast to other abscesses, the walls of the amœbic type are soft, ragged, and produce but little granulation tissue or fibrous encapsulation.

Symptoms.—Absence of all symptoms (latency) occurs in 13 per cent. of cases. Symptoms may be classified as (1) septic, (2) hepatic, and (3) other symptoms and complications.

1. **Septic Symptoms.**—(*a*) *Fever* is the most important; it is remittent or intermittent, is less often continuous, and in chronic cases there may be a normal or subnormal register. (*b*) *Rigors* and sweats are frequent. (*c*) *Leukocytosis* occurs in about 50 per cent. only. In 43 uncomplicated dysentery cases, the leukocyte count was above the average count of the abscess cases in 23.4 per cent. (Futcher). (*d*) The *pulse* is small and frequent. (*e*) Progressive *emaciation* is usual, and the facies is a combination of hectic, depression, subicterus, and cachexia, which is difficult to describe. (*f*) The urine is febrile and may contain albumin or albumose.

2. **Hepatic Symptoms.**—The *liver* is enlarged from the abscess and from sepsis. The *abscess* in 70 per cent. is located in the *right lobe;* Sérégé by injections has shown that venous blood from the pancreas and intestine is conveyed to the right lobe (whence the right lobe is chiefly involved in intestinal ulceration or cancer), while the blood from the stomach and spleen finds its way to the left lobe. The *enlargement* is more often *upward* than downward, wherein abscess differs from cancer and resembles echinococcus. The swelling is often localized, occasioning a *dome-like dulness on the upper border* of the liver (Frerichs); in other cases there is bulging of the lower interspaces or of the hypochondrium. In very large abscess the entire organ is greatly swollen, as in one reported case of an abscess which contained 2½ gallons of fluid. When it occurs below the ribs, fluctuation in the liver, and, less often, œdema of the abdominal wall, may be noted. The liver consistence may be increased at first. (*b*) *Pain is usually* present, and is due to increased weight or to capsular tension and inflammation; perihepatitis or sometimes pleurisy may cause a friction rub which results from fresh inflammation; or may cause adhesions in more chronic cases. The patient usually lies on the back or right side to avoid the pain caused by sagging of the liver which occurs when he is in the left lateral decubitus. Pain is present in the right shoulder in 17 per cent., being reflected from the phrenic filaments on the liver convexity along the phrenic nerve to the acromial branch of the fourth cervical nerve, with which the phrenic nerve is connected; in rare cases the deltoid may waste; the left shoulder is seldom the seat of referred pain. (*c*) *Tenderness* over the liver is usually circumscribed, but sometimes general. The right rectus muscle is frequently tense.

3. **Other Symptoms; Complications.**—(*a*) *Icterus* occurs in 16 per cent. of cases; it may be slight from sepsis or from compression of some

bile ducts within the liver, or it may be complete from catarrh of the common duct. (*b*) The *spleen* is *seldom enlarged*, except from a causal septicopyæmia. (*c*) A dry spasmodic *reflex cough* may be noted (*tussis hepatica*). (*d*) *Rupture* may be the first sign of abscess of the liver. In Waring's 300 cases, 56 per cent. remained intact, 16 per cent. were operated on, and 28 per cent. ruptured. (i) It occurs *into the lungs* in about 10 per cent. (average of the figures of Waring and Cyr abscess of the lung results more often from direct rupture than from indirect metastasis by the hepatic vein. The patient presents signs of lung abscess (*q. v.*) and expectorates chocolate-, anchovy- or cream-colored pus containing amœbæ, often liver and lung tissue, hæmatoidin crystals, and perhaps bile. Death may be caused by profuse hæmoptysis. (ii) Rupture into the *right pleura* (6 per cent.) causes empyema. Pleurisy, both fibrinous and serous, may also occur without rupture, resulting from attenuated infection passing through the lymphatics of the diaphragm; a right-sided friction rub, serothorax or empyema, should always suggest the possibility of an hepatic etiology. (iii) Rupture into the *peritoneum* (6 per cent.) causes localized or diffuse peritonitis. A subacute serofibrinous peritonitis rarely results, but the author has seen this form, as well as an encapsulated serous effusion, cover the convex and anterior surface of the liver. (iv) Rupture into the *bowels* (colon 2 per cent. and duodenum 1 per cent.) causes pus to be voided, sometimes in large amounts, by the stools. (v) Less frequent are ruptures into the stomach, pericardium, pelvis of the kidney, portal or hepatic vein, inferior cava, bile vessels, or abdominal wall. (*e*) Less frequent complications are pyloric obstruction (from abscess of the left lobe), pseudorheumatism or clubbed fingers (from toxæmia), cerebral abscess, and intestinal obstruction.

Diagnosis.—Diagnosis is based on (*a*) an *etiological factor;* (*b*) symptoms of *sepsis;* (*c*) *hepatic signs*, localized enlargements, pain, and tenderness; the *x*-rays may be useful when there is bulging of the convexity covered by the lung; (*d*) exploration with the *aspirating needle*, which is usually without danger. The author has seen, as a result of puncture, two deaths from peritonitis and one from an enormous hemorrhage into the peritoneal cavity in a case of abscess with intense icterus; puncture should be performed only when an operation can be done immediately after pus is found; pus is far less often found in multiple small abscesses than in the large solitary form.

Differentiation.—*Five types* of liver abscess are often described; (*a*) the latent, (*b*) typhoid (see TYPHOID TABLE, including SEPSIS and MALARIA), (*c*) malarial, intermittent, (*d*) phthisic, and (*e*) a type declared by rupture, either pulmonary, peritoneal, or pleural.

Subphrenic abscess simulates pneumothorax (*q.v.*) rather than liver abscess, because of the presence of tympany; in its etiology and treatment (see LOCALIZED PERITONITIS) it resembles liver abscess. In *gall-stones*, with or without suppuration, the fever usually occurs in paroxysms separated by fairly long intervals, jaundice (if present) is apt to become more intense with the fever, the history is different, and marasmus is rare. Abscess of the abdominal wall shows no respiratory excursion in the exploratory needle introduced into the abscess. Differentiation from *pleurisy*, *echinococcus*, and *cancer* of the liver, is considered under these topics and also in the differential table (page 724).

Prognosis and Treatment.—Ninety-three per cent. of unoperated cases die from unfavorable ruptures (peritoneal, pleural, pulmonary), sepsis, marasmus, and amyloid disease, which may occur after weeks or months. It is possible for an abscess to desiccate or to heal after breaking into the lung or bowel but this cannot be depended upon. The treatment is solely operative; Solonoff (1903), quoted by Rolleston, in a collection of 1,094 cases estimates the mortality at 30 per cent.

(E.) TUMORS OF THE LIVER.

Tumors of the liver are chiefly *carcinoma*. *Benign tumors* possess little clinical interest, as fibroma, lipoma, teratoma, adenoma, or angioma, which latter is more frequent in the liver than in any other organ. Single adenoma may develop from the liver cells or bile ducts; multiple adenomata occur chiefly in cirrhosis. (Three cases are reported of adrenal "rests.") *Sarcoma* is rarely primary, there being hardly twenty clear cases on record; it may develop from the connective tissue, endothelium of the lymph or bloodvessels or Kupfer's cells, as a single primary tumor, multiple primary tumors, diffuse infiltration, or possibly as a primary melanotic growth, which, however, is almost always secondary to melanotic sarcoma of the eye or skin.

Cancer of the liver occurred in 2.7 per cent. of Virchow's autopsies, and ranks third among internal cancers: the frequency of cancer localization varies somewhat; the usual order of frequency is cancer of the uterus, stomach, mamma, and liver; Eichhorst's order is uterus, stomach, mamma, œsophagus, and liver. (*a*) *Primary cancer* is 20 to 48 times as infrequent as secondary cancer; 66 per cent. occurs in males. It originates from the liver cells (88 per cent.) or bile vessels (12 per cent.). The main forms are observed (i) as sharply outlined massive local swelling (in 23 per cent. of Eggel's 163 cases reported in 1901); (ii) as sharply demarked multiple nodes of moderate size (in 65 per cent.) of which one of the tumors is primary and the rest secondary; and (iii) as diffuse infiltration, fusing with the parenchyma (in 12 per cent.); (iv) there is a fourth form, which develops in hepatic cirrhosis. The writer has seen primary cancer develop from an hepatic cyst. Metastases occur in 66 per cent (Eggel). (*b*) *Secondary cancer* results from primary cancer in the stomach (33 per cent.), in the colon (12 per cent.), œsophagus, pancreas, gall-bladder, uterus, ovaries, and mamma, by metastasis or extension by contiguity; cancer cells may enter by the portal vein and hepatic artery, or much less frequently by retrograde metastasis through the hepatic vein; the lymph vessels do not carry cancer infection, as their current flows from within to without the liver. Their structure is that of the primary type.

Symptoms.—1. Hepatic Symptoms.—(*a*) The *liver enlarges rapidly;* it may increase to six or eight times its normal size and may weigh 30 to 40 pounds. Its *edge is uneven* though fairly sharp; on its *surface are lumps* which may present a central umbilication. *Great unevenness* is more characteristic of secondary than of primary tumor in which the liver is more uniformly swollen. The *enlargement* is chiefly *downward* though the liver is sometimes high because of ascites, tympanites, or adhesions. Nodules in the falciform ligament may be felt near the navel or linea alba. Sudden increase in size occasionally results from hemorrhage which is due to icterus. There is no enlargement when the nodules are few and small, and the liver is smaller than normal in 10 per cent. of cases (liver cirrhosis plus malignant adenomata). (*b*) *Hepatic pain* is *more conspicuous than in any other liver lesion.* It may be caused by capsular tension, increased weight, perihepatitis, pleuritis, or, less frequently, from rupture of tumor particles into the bile passages, which may simulate the pain of gall-stones. (*c*) The *liver is often tender.* (*d*) Exceptionally pulsation may be transmitted from the aorta, or there may be a venous hum over the liver.

2. Cachectic Symptoms.—These are adynamia, marasmus, anorexia, secondary anæmia, leukocytosis (in any carcinoma, but especially in hepatic), insomnia, stupor, and illusions. Cachexia probably results from the katabolic toxins elaborated by the tumor, and possibly also from hepatic insufficiency (*v.* CIRRHOSIS). Fever may result from the tumor alone, from cholangitis, and from secondary or terminal infection. The rare peripheral neuritis and some of the urinary findings may be referred to cachexia. In some rare cases there may be no cachexia.

3. Compression Symptoms.—(*a*) *Jaundice* occurs in 50 to 90 per cent. and more frequently in secondary than in primary cancer. It usually results from mechanical pressure on the larger bile ducts by the tumor or by carcinomatous glands. Pressure on the ducts within the liver often produces moderate jaundice, and some bile then appears in the stools; complete icterus may result from catarrhal or calculous obstruction (especially significant when there is no ascites) or from pressure by enlarged lymph glands. Exceptionally the tumor grows into and along the bile ducts, thereby totally obstructing them. (*b*) *Ascites* is present in 50 per cent. of cases. It is usually moderate in degree and results from pressure on the portal vein or its larger branches, peritonitis, or portal vein thrombosis (pylethrombosis); occasionally it is due to the cirrhotic form of cancer, growing of the tumor into the hepatic or portal vein, rupture—perhaps with syncope and great hemorrhage—of a cancer nodule into the peritoneum, or rarely to plugging of the liver capillaries by the pigment of a melanosarcoma (Hektoen and Herrick). A collateral circulation may develop. Friction from perihepatitis may be noted. The fluid is usually serous or hemorrhagic, less often adipose, chylous, or opaque (*v.* CIRRHOSIS), and very rarely contains melanin (sarcoma). (*c*) *Other compression symptoms* include splenic tumor from marantic pylephlebitis or pressure on the splenic vein (11 to

17 per cent.), hydronephrosis, vomiting from pyloric obstruction, dyspnœa from pressure on the heart and lungs, and œdema from pressure on the cava inferior.

4. **Other Symptoms.**—The cervical glands are seldom swollen unless there is coincident gastric cancer. Carcinomatous nodes may appear at the navel or even in the skin over the liver, which is due to regional extension. In liver cancer combined with cirrhosis any cirrhotic symptom may occur. The urine shows increased destruction of the tissue nitrogen; the nitrogenous extractives are increased from the normal 0.6 or 0.8 per cent. to 13 or 23 per cent. Acetone, and diacetic, lactic, beta-oxybutyric acids, and indican, are not infrequently present. The urine is decreased, high-colored, and is often icteric. Albuminuria is due to stasis and toxæmia (Teissier speaks of hepatogenous albuminuria). The urea may be decreased; leucin and tyrosin are sometimes present. Melanuria (sarcoma) is sometimes detected by adding to the urine a solution of ferric chloride; the pigment exists in the blood as melanin or melanogen, the latter of which is precipitated as the urine stands, or by the addition of some oxydizing agent as nitric acid; its presence nearly always indicates involvement of the liver by a melanotic tumor; melanuria has often been confounded with indicanuria and alkaptonuria.

Diagnosis.—The chief features are rapid enlargement of the liver, irregular tumors, pain, icterus, ascites, cachexia, and, in 50 per cent. of cases, the recognition of the primary tumor. Other causes of liver enlargement must be considered as in cirrhosis. (See also DIFFERENTIAL TABLE, page 724.) Icterus with ascites is always suggestive. In some cases liver involvement is not suspected. The differentiation is as follows: (*a*) *Cirrhosis* (see both forms). (*b*) *Syphilis* (*q.v.*), as a rule, produces smaller, harder tumors, of less rapid growth, which are more often associated with splenic tumor and albuminuria, much less often complicated by ascites or icterus, and respond to mercury and iodide. (*c*) The *amyloid liver* is equally large and but seldom causes confusion except when complicated by gummata; amyloidosis of the spleen, kidney, and intestines, usually co-exists with amyloid liver. (*d*) *Hydatid cysts* develop more slowly and *earlier in life*, and cachexia appears late if at all. Multilocular echinococcus produces enlarged liver, ascites, and icterus, but the spleen is also enlarged; puncture may give characteristic findings; the affection is very rare. (*e*) *Liver abscess* is seldom accompanied by icterus and ascites; puncture often excludes those doubtful cases of cancer in which there is fever, leukocytosis, and semifluctuation of the necrotic neoplastic nodes. (*f*) In *congested liver* the causal heart or lung disease, the symmetrical liver swelling, other signs of stasis, diffuse tenderness, and subicterus together with cyanosis, are distinctive, though the writer knows of four cases of nutmeg liver in which the stasis unaccountably caused more local than general hepatic enlargement. (*g*) The *corset liver* can be easily differentiated. The general symptoms, such as cachexia, and the compression symptoms of cancer, are duly considered. (*h*) *Cancer of* *the stomach* (*q. v.*) is readily differentiated by its peculiar chemism and its signs of stagnation; gastric cancer may, of course, be the cause of hepatic involvement. (*i*) *Renal or adrenal tumors* are covered by the inflated colon, encroach less upon the thorax, evert the ribs less, have (less or) no respiratory excursion, and often are separable from the liver by an intervening strip of tympany.

(*j*) *Differentiation between primary and secondary tumors* is difficult, yet is somewhat irrelevant according to Leube, as the causal growth leading to hepatic metastasis is latent in 50 per cent. of secondary tumors, and great caution should be exercised in diagnosticating primary liver tumor. The following characteristics of primary tumor are of relative importance, though subject to many exceptions: the solitary tumor, its rapid growth, infrequent jaundice and ascites, lesser emaciation, and more rapid course, which results in death, according to Hale White, in less than four months. Secondary tumors cause death within seven months, from cachexia, ascites, and intercurrent pneumonia.

Treatment.—Treatment is entirely palliative for the pain and ascites except when, in very exceptional cases, early operation (for primary growths) can be performed. Luecke reported the first recovery, which afterward proved to be a gumma; his operation was followed by a few others, the cases of Nothnagel, Keen, Mayo Robson, Bardeleben, D'Urso, Israel, Hochenegg, Heidenhain, von Bergmann, and others, which included recovery from cancer, sarcoma, and malignant adenoma.

(F). ECHINOCOCCUS CYSTS OF THE LIVER.

The parasite will be considered under ANIMAL PARASITES and we will here consider the symptoms of its hepatic localization, which constitutes about 60 per cent. of echinococcus disease; though the adult parasite occurs in the intestine of the dog, wolf, fox, and jackal, its cystic form occurs in man alone.

Symptoms.—*Fully half the cases* of liver echinococcus are clinically *latent.* Cases with symptoms show the following *liver findings:* (i) A *cyst on the lower border* or anterior surface may produce a visible, palpable, round and *smooth bulge* which everts the costal arch or much less often affects the left lobe. In some cases cysts in the right lobe lead to compensatory hypertrophy of the left lobe, which may equal the size of a normal liver. The tumor is as large or larger than an orange. It is tense, elastic, semi-solid, and on percussion is dull; it may fluctuate and give the "hydatid thrill" of Briancon and Piorry, a vibration which is elicited by laying three fingers over the cyst and tapping with the middle one; the thrill is not a frequent finding, and may also occur in ascites and sarcoma. The cysts rarely contain gas, which is due to the Bacillus coli. In large cysts (36 to 70 pints are reported to have been found) the liver may tip so that the right lobe is lower and the left higher than normal. (ii) *Cysts on the convexity* crowd the diaphragm upward, may cause symptoms resembling those of pleurisy (*q.v.*), produce a dome-like dulness as in cancer, and may compress the lung or even the heart. (iii) *Liver pain* and *tenderness are rare* without suppuration, which may induce perihepatitis; pain, as in cancer, may be referred to the right shoulder. Pleuritic pain follows rupture into the pleura. (iv) *Exploratory puncture* (Récamier and Cruveilhier) is dangerous, because severe nervous toxæmia and cardiac collapse may follow, as described under echinococcus of the lung and pleura, where also the finding of pus, the characteristic membrane, chemical contents, and hooklets, are enumerated.

Rupture may occur into the lungs (in 11 per cent. of liver echinococcus); it may simulate phthisis or gangrene of the lungs; cysts may rupture into the pleura, peritoneum, kidney, stomach, bowel, bile passages, pericardium, portal or hepatic vein, and cava inferior; heart embolism or external rupture may occur.

Pressure symptoms are rare, as icterus (5 per cent.), pyloric obstruction, ascites, albuminuria, *caput Medusæ*, or œdema of the legs. Dyspnœa may result from pressure on the lungs, or vomiting from pyloric stenosis. In some cases there is a distaste for fat or meat. Hemorrhage from the alimentary tract is rare. The blood is usually normal, though the eosinophiles are usually increased in nonsuppurating cysts. Urticaria may occur after puncture or rupture, or without either incident.

Diagnosis.—Diagnosis depends on (*a*) the history of the case, including close contact with dogs; (*b*) the smooth, round tumor; (*c*) the thrill; (*d*) slow development; (*e*) the infrequency of constitutional disturbance, pain and fever; in other words, the predominance of the signs over the symptoms.

Differentiation.—(*a*) From *other liver cysts;* the small cysts which occur in cirrhosis and in obstructive icterus are of no clinical importance. Simple serous cysts may, in exceptional cases, hold several (even 13) pints; they rarely cause symptoms, rupture or hemorrhage. Diffuse, congenital, cystic disease may cause dystokia; in adults also the cysts are probably congenital or developmental in origin, and are associated with cystic kidneys, in 4 to 27 per cent. of cases (Lejars, Luzatta, and Johnson). Moschcowitz (1906) collected 85 cases of non-parasitic cysts of the liver; they are usually multiple and subcapsular and are often associated with other congenital anomalies such as hare-lip, spina bifida, hypospadias, or supernumerary fingers. He concludes that (i) they are always associated with congenitally aberrant bile ducts; (ii) they are embryonal "rests"; (iii) the cysts are due to inflammatory hyperplasia of these ducts or to congenital obstruction in them. Hoffmann reported 18 operated cases. (*b*) *Malignant disease* causes cachexia more pain (than does echinococcus), multiple tumors, ascites, and icterus. (*c*) *Syphilis of the liver* (*q.v.*) more often causes albuminuria or splenic tumor, and responds to antisyphilitic remedies. (*d*) From *pleurisy;* the *x*-rays may show the localized tumor on the liver convexity; but the upper line of pleuritic dulness is much less often convex upward; there may be thora symptoms, as hæmoptysis, when there is rupture into the lung; ca ful examination of the sputum or aspirated fluid shows the diagnos hooklets. (*e*) In rare cases a pendulous, pedunculated, movable c (v. Frerichs) may necessarily resemble pancreatic cysts, enlarg kidney, gall-bladder or ovarian cyst. Rupture into the bile passa may simulate the pain of gall-stones. (*f*) Liver abscess is usua distinguished with ease, though one may remain in doubt in ca of suppurating cysts; the eosinophiles are not increased in li abscess; both conditions are strictly surgical. (See table page 72

Prognosis.—The prognosis depends largely on whether surgery invoked. Cysts may, however, calcify or indurate spontaneous According to Cyr, the mortality of rupture into the peritoneum 90 per cent., into the pleura 80 per cent., bile ducts 70 per ce bronchi 57 per cent., stomach 40 per cent., intestines 15 per ce and externally 10 per cent.

Treatment.—When drainage by the trochar is performed (Hipp rates), it is said that 45 to 77 per cent. of cases recover, and so are benefited by injection of iodine or electrolysis. A few die su denly from a toxalbumin, which is formed when the parasite di it causes urticaria, convulsions, cardiac failure and other sympto similar to those produced by myrtilotoxin in mussel-poisoning. *Di surgical intervention* is attended with the least risk. *Prophylaxis* necessary, such as the burning of slaughter-house offal and avoidi close contact with dogs.

Multilocular or Alveolar Echinococcus.

This form is due (see ANIMAL PARASITES) to another parasite; is extremely infrequent and only about 100 cases are recorded.

Symptoms.—The right lobe of the liver alone is involved in per cent. of cases, and the left alone in 10 per cent. The liver hard (from fibrosis), nodular and seldom fluctuates. This rare aff tion is attended by splenic tumor (90 per cent.), icterus (80 per cent dyspepsia, ascites and, less commonly, by fever, polyuria, collateral c culation, gastro-intestinal hemorrhage, late emaciation (cachexia), a metastases in the heart and lungs by way of the lymph or blo vessels; or death may occur after a number of years from cholæmia hemorrhages. The diagnosis is very difficult, though it is sometin aided by exploratory puncture, and the most frequent confusion with cancer of the liver. The only hope of recovery is in extirpati which has been successfully done by Terillon, Bruns and others.

(G). FATTY LIVER.

The term "fatty liver" (*hepar adiposum*) includes fatty infiltration and fatty degeneration; as a distinction between them is of more speculative than clinical interest, they may be considered together.

Etiology.—(*a*) *Obesity* and (*b*) *decreased oxygenation* from carcinosis or any other cachexia, rickets, profound or pernicious anæmia, phthisis, cardiac stasis and kindred causes may produce fatty liver. (*c*) *Toxæmias*, as tuberculosis, the acute specific infections, infantile diarrhœa, acute yellow atrophy, and suppuration, (*d*) *poisons*, as phosphorus, alcohol, arsenic, or chloroform, and (*e*) *local diseases* of the liver, as cirrhosis or nutmeg liver are etiological factors.

Pathology.—The fat, which is normally found in the liver up to 2 to 5 per cent. of its weight, may amount to over 40 per cent. The liver is *enlarged*, and may weigh as much as 12 pounds or 4,500 gm. Its *surface is smooth*, its edges are rounded, its *consistence is decreased* and its color is yellow. On section it is anæmic, yellow and smooth, though the lobules may be slightly prominent. The color is yellow when oleic acid predominates, and pale when there is more palmitin and stearin. Slight quantities may be detected only on microscopic examination. When cut with a warm knife the fat may be seen on the blade. Microscopically, fat is seen which, *in fatty infiltration*, is located principally in the periphery of the lobule and obscures the liver cells; when the fat is dissolved by ether, the normal liver cells are clearly seen. *In fatty degeneration*, fat forms at the expense of the liver cells, *i.e.*, the percentage of albumin is decreased. The statement that fatty infiltration occurs in large globules and fatty degeneration in smaller ones is not wholly correct. Pathologists have lately been inclined to consider infiltration of more importance than degeneration.

Symptoms.—(*a*) *The positive findings* are as follows: the liver is *symmetrically enlarged* and *smooth* and rarely extends below the navel. Its *edge is rounded*, soft and can usually be palpated, even in obese persons. (*b*) The *negative findings* are absence of pain, tenderness, icterus, splenic tumor, ascites or gastro-intestinal hemorrhage. The urobilin may be decreased.

The *diagnosis* is easily made, and liver stasis, amyloid degeneration, and cirrhosis can be readily excluded (see DIFFERENTIAL TABLE, page 724). The treatment is merely that of the causal obesity or of other factors involved.

(H). AMYLOID LIVER.

Amyloid degeneration was first clearly described by Rokitansky.

Etiology.—(*a*) *Ulcerative tuberculosis* of the lungs or bones causes over 50 per cent. of amyloidosis. (*b*) *Inveterate syphilis*, particularly the ulcerative and osseous types, produces about 25 per cent. of amyloid disease. (*c*) *Chronic suppuration,* in connection with, or independently of, the above causes, has become uncommon since the introduction of asepsis and antisepsis. Krawkow produced amyloid degeneration experimentally by inoculations of the staphylococcus. (*d*) As exceptional causes, intestinal ulcerations, exhausting diarrhœas, rickets, leukæmia, pseudoleukæmia, and ulcerating neoplasms may be mentioned. Seventy-eight per cent. occurs in males (Frerichs).

Pathology.—Amyloid degeneration is much less frequent in this country than in Europe. Its *localization,* in order of frequency, is in the spleen, kidneys, liver and intestines. The liver is uniformly enlarged and may weigh 13 to 16 pounds (5 to 6,000 gm.), even in a child. It is hard, smooth and very pale. Its edges are more blunt than normal, though somewhat sharp. On section the general appearance is waxy, speckled, somewhat resembling raw ham, the edges are slightly translucent and the lobules are indistinct. If a thin slice is placed in diluted Lugol's solution, the amyloid areas assume a brown color (Virchow), the name (amyloid) referring to this starch-like reaction; the waxy substance, however, is a proteid, and reaches the liver by the hepatic artery, whence it is first deposited in the middle zone of the lobule, and later infiltrates the central and peripheral zones. It is essentially an infiltration, affecting primarily the capillaries of the hepatic artery. Slight lesions may necessitate the use of the microscope for detection. Amyloid liver may co-exist with gumma, cancer, or cirrhosis.

Symptoms.—The large, tense, symmetrical, painless, smooth liver, with rounded edges, is characteristic. Its measurements may attain those caused by cancer. The onset and development are usually slow, but acute amyloid has developed within a month. *Splenic* amyloid *enlargement* is usually also noted, as well as *albuminuria* from renal amyloidosis (*q.v.*). In only 0.8 per cent. of amyloid degeneration is the liver alone involved (Fehr). *Gastro-intestinal symptoms,* as nausea, vomiting and diarrhœa may result from amyloidosis of the alimentary capillaries. In advanced cases *cachexia, œdema* and ascites may develop. Hepatargia is rare.

Diagnosis.—Diagnosis depends on (*a*) the recognition of a *cause;* (*b*) the characteristic, smooth, *symmetrical enlargement,* which is less hard than that of cirrhosis; (*c*) the association with *amyloid disease* of the *spleen, kidney,* and alimentary tract; (*d*) the *absence of icterus* (which very rarely results from amyloid portal glands) and of *portal stasis* (ascites is infrequent, is a late symptom and results from renal implication); and (*e*) the small amount of urobilin in the stools. *Differentiation* from cirrhosis (*q.v.*) and cancer (*q.v.*) presents no difficulties. Gummata in the liver plus amyloid disease may cause confusion. No diagnosis is possible in the exceptional cases in which the liver is slightly enlarged, because it is only slightly diseased, or in those cases in which splenic tumor and albuminuria are absent. Other diffuse and focal hepatic diseases are differentiated in the table on page 724.

The *prognosis* of pronounced cases is unfavorable, though light grades may exceptionally regress (Budd, Bamberger, Frerichs, Graves).

Treatment is unavailing; it is solely that of the causal condition and of the symptoms as they arise.

(I). ANOMALIES OF FORM AND LOCATION OF THE LIVER.

In *situs viscerum inversus* the liver lies on the left side, which in pathological conditions may lead to serious diagnostic error. Fenger and Billings mention a case of icterus and gall-stones in situs inversus which was diagnosticated as acute yellow atrophy!

Tilting of the liver (ante- and retroversion), *dislocation downward* by pleuritis, pneumothorax or tumors, or *upward luxation* require but passing mention.

The liver may be found in the thoracic cavity as a result of congenital malformation or severe trauma.

"Wandering liver" (*hepar migrans s. mobile*) occurs in women, (in 88 per cent.) and usually after several pregnancies. Its *causes* are those of enteroptosis, with which it usually occurs, *viz.*, flaccid abdominal walls, possibly congenital laxness of the suspensory ligaments, tight lacing, or very rarely from traction, as by a tumor. J. Dutton Steele collected 100 cases, which Clark and Dolley (1905) increased to 118; they report one case with a mesohepar attached to the diaphragm and with absence of the coronary and right lateral ligaments.

Symptoms.—Symptoms may be entirely lacking. In 5 per cent. the onset is acute (Graham), though usually gradual. *Subjectively,* vague pain or oppression over the liver, gastro-intestinal disorders or nervous symptoms may be present. *Objectively,* the liver is lower and is more mobile than normal; there is sometimes cardioptosis (Einhorn), usually gastroptosis, and in 20 per cent. nephroptosis (J. D. Steele). Its notch can be distinctly felt, often also its convex surface and sometimes the tense, stretched ligaments. It can be replaced. The entire liver, in extreme instances, may be below the costal arch, and tympany, due to the bowel, may exist between it and the lung. In a recent case seen with Dr. DeLee there was tympany between the left costal arch and the prolapsed spleen, and tympany between the right arch and the liver, even though the patient was five months pregnant. In very rare instances there may be pain similar to that of gall-stones; ascites, or splenic tumor, result from torsion of the portal vein.

Treatment.—The *treatment* is (*a*) *mechanical,* which consists of replacing and supporting the liver with appropriate binders or pads; (*b*) *symptomatic,* with due consideration of the neurasthenic manifestations; (*c*) *dietetic,* in order to increase the body-weight and (*d*) *operative,* in which the liver is sewed to the diaphragm or abdominal wall. In 39 cases, hepatopexy cured about 75 per cent.

The corset liver is found in 25 per cent. of female cadavers (Leube), though it is much less frequent in the United States than in foreign countries. Corsets cause pressure-atrophy of the liver substance, which is followed by induration, peritoneal thickening and deformity; these changes are most conspicuous and usual in the right lobe. The liver may be merely constricted, or dislocated upward; or the part below the constricting zone may hang loosely as upon a hinge; this may cause diagnostic confusion with floating kidney, enlarged gall-bladder or other tumors. The gall-bladder is said to be dilated in 60 per cent. of cases, though icterus is quite infrequent. Subjective manifestations are often absent, though there may be a sense of pressure over the part. Sometimes paroxysmal syncope and vomiting may occur; they are due to congestion of the snared-off portion of the liver. Physical examination often determines the condition, especially when, by careful palpation, the hinged and hard part can be proven directly continuous with the liver's known margins. Respiratory excursion is naturally imparted to it in most cases. Confusion necessarily arises when a loop of gut causes a small zone of tympany between the hinged segment and the liver proper, but even then careful palpation frequently demonstrates its continuity with the liver. Congenital tongue-like lobes may be confused clinically with corset liver; they are often cirrhotic and relatively often the seat of malignancy, suppuration or syphilis. Corset liver is regarded as a disposing factor in gall-stone formation. Treatment is seldom possible nor is it important, though prophylaxis and perhaps resection should be mentioned.

(J). AFFECTIONS OF THE BLOODVESSELS OF THE LIVER.

I. Active Hyperæmia of the Liver.

A degree of arterial hyperæmia is physiological during digestion, when an excess of blood is found in the portal circulation. An accentuation of this condition is common in heavy eaters and drinkers, in trauma and acute infections, in the latter of which it may be combined with venous hyperæmia. Heat is probably not a cause, though some physicians in the tropics speak of tropical hyperæmia. The alleged vicarious hyperæmia from suppressed menstruation, the menopause, or diseases of the internal female genitalia, is based wholly on theory. The symptoms resemble those of passive hyperæmia (*v. i.*), *viz.*, some hepatic enlargement, moderate discomfort in the right hypochondrium, and also, it is alleged, slight icterus. Treatment is seldom indicated, save in cases of dietetic excesses, where gastro-intestinal symptoms also obtain. Then a simple, moderate, or milk diet, together with calomel and saline purgation, is indicated.

II. Passive Hyperæmia of the Liver.

Etiology.—Passive congestion of the liver results from various kinds of stasis, the stress in all of which is felt in the efferent hepatic (sublobular) veins within the liver. It results (*a*) from *cardiac* insufficiency, which is caused by valvular diseases, particularly mitral, myocardial maladies or pericardial lesions (effusion or synechia;) (*b*) from *pulmonary* stasis, due to emphysema, chronic bronchitis, asthma, induration or obliterative pleurisy; (*c*) from stasis caused by *spinal deformity*, aneurysm, mediastinal tumor, *perihepatitis*, or pressure on the cava inferior.

Pathology.—The essence of passive congestion is over-filling of the sublobular or the *hepatic venous circulation*, malnutrition of the liver cells (*fatty degeneration*) and in some advanced cases, *increased connective tissue*. The liver is usually *symmetrically enlarged*, *firmer*, and a darker red than normal, though hepatic hyperæmia lessens after death. The capsule is often thickened or œdematous. On section the so-called *nutmeg appearance* is noted, but this seldom occurs over the entire liver. The *central veins* with their radiating offsets are *dark*, cyanotic, and sometimes sclerotic from stasis, while the periphery of the lobule is yellow-white from cloudy or fatty change (malnutrition or impaired oxygenation). The nutmeg appearance is not pathognomonic of passive congestion. Long-standing congestion results in atrophy and pigmentation of the cells in the periphery of the lobule(Virchow's *red atrophy*); there is usually increased connective tissue with fine granulations; this is the *atrophic cyanotic liver* or the cardiac cirrhosis of the French; fibrosis is probably for the most part relative or apparent, for the liver parenchyma atrophies; true cirrhosis may co-exist with cardiac disease.

Symptoms and Diagnosis.—(*a*) The *causal heart* (or lung) lesion has its appropriate symptoms. (*b*) The *liver* is *evenly enlarged*, both on percussion and palpation, sometimes also on inspection; if ascites is present, thrusting palpation with the finger-tips usually outlines the organ; the liver may reach to the navel or even lower and may evert the ribs. Its *edges are firmer* than normal, somewhat rounded, *sensitive* (from capsular tension) and *descend with inspiration;* the incisure is clearly felt. If there is relative tricuspid insufficiency, a systolic expansile pulsation is detected by palpating with both hands, which are separated from each other with each systole. This is readily differentiated from the non-expansile pulsation which is transmitted to the liver from the aorta. (*c*) The *hepatic intumescence varies greatly*, as the heart responds to rest or to digitalis, or fails in compensation. (*d*) The *skin* shows *cyanosis* and is also *tinged with slight icterus;* to the experienced eye, it has a characteristic appearance, which is probably due to a static catarrh of the finer interhepatic bile radicles. (*e*) For other symptoms, see SIGNS OF STASIS under VALVULAR DISEASE. The spleen is seldom enlarged save from cardiac infarcts or complicating liver cirrhosis. Hæmatemesis is infrequent. Albuminuria and gastro-intestinal symptoms are referable to the heart. *Ascites* is usually *late* in appearing and moderate in degree; the earlier statement, that an ascites, which is disproportionately marked in comparison with the general œdema, indicates the atrophic (indurated) nutmeg liver, is incorrect, because a cardiac ascites sometimes occurs without general œdema. Toward the end of the course there may be signs of renal and hepatic insufficiency.

Differentiation is seldom complicated, for the fundamental heart lesion, the evenly enlarged, tender liver, which varies in size from time to time, and the slight icterus with the cyanosis, are usually unmistakable. Certain features may sometimes mislead, as the occasional unequal distribution of the liver changes, which may suggest tumor; the tenderness in two cases which the author saw was thought to be suppurative. (See table page 724.) The treatment is that of uncompensated valvular disease (*q. v.*).

III. Pylethrombosis—Pylephlebitis.

Thrombosis or phlebitis of the portal vein is practically always *secondary* to *compression or obstruction,* as liver cirrhosis with which 33 per cent. of pylephlebitis cases occur; to cancer, ulceration, abscess or *inflammation* of contiguous structures, gall-stones, cholangitis and chronic proliferative peritonitis; and to *diseases of the intima,* as phlebosclerosis and syphilitic endophlebitis; all cases are promoted by slowing of the portal current. Parasites (distoma and bilharzia hæmatobium) and embolism are rare causes (embolism rarely causes complete liver necrosis unless the hepatic arteries are simultaneously closed). Pylephlebitis and thrombosis occur chiefly in males over forty years of age.

A fatal obliterating endophlebitis exists with symptoms very closely resembling those of atrophic cirrhosis; Hess in 1905 collected 23 cases from the literature.

Symptoms.—These may be entirely absent because of collateral compensation through the hepatic artery or Sappey's accessory portal vein. The *onset* is usually sudden, with symptoms of *acute portal stasis.* The *spleen is enlarged* in 75 per cent., unless fibrous changes in its pulp or capsule prevent its swelling. *Ascites* develops in 66 per cent. and rapidly recurs after tapping. *Gastro-intestinal* symptoms are usual, as *hemorrhage* (44 per cent.), intense *sudden epigastric pain,* vomiting, diarrhœa or sometimes acute intestinal obstruction. The *portal vein* is sometimes *varicose* below, and is generally *obliterated* (pylephlebitis adhæsiva) at and above the point of obstruction; this leads to *shrinking of the liver,* particularly if the hepatic artery is sclerotic, in which case the entire liver may rarely be hemorrhagically infarcted. The liver is said to become lobulated, but this probably results from antecedent syphilis. Symptoms of hepatargia (hepatic insufficiency or toxæmia) may develop. Alimentary glycosuria is fairly frequent.

Œdema of the abdominal wall, development of a collateral circulation, and icterus are uncommon.

Diagnosis.—Diagnosis is difficult, unless the onset is typically sudden, in which case pylethrombosis may be considered. Save for their acuity, the findings are those of atrophic cirrhosis; a caput Medusæ rather indicates occlusion of the portal radicles than of the portal trunk. The ascites may suggest carcinoma or peritonitis, or the hemorrhage may simulate gastric ulcer or hepatic cirrhosis.

Course and Treatment.—Death may occur in a few days or the patient may live for months or years (six to twenty years). Treatment is ineffectual, save in early syphilitic cases. Citric acid (ʒj) may be given every few hours, in an attempt to lessen thrombosis. Talma's operation may be considered.

Pylephlebitis suppurativa has been considered under liver abscess. It is a pyæmia of the portal vein; metastasis or extension occurs in the main portal trunk and its branches from infection of its radicles of origin; ulcerations and inflammations in the gastro-intestinal tract are its cause in 75 per cent. of cases; appendicitis causes about 40 per cent. (Dieulafoy's "appendicular liver") and gall-stones, infected piles, and pancreatic or umbilical suppuration cause most of the other cases (*v.s.* LIVER ABSCESS, SECOND VARIETY).

The *symptoms* are those of the *original disease*, those of *sepsis* and those of *portal stasis*. In very rare cases the thickened vein may be palpated. The spleen is enlarged, chiefly from sepsis, though sometimes from stasis. The liver is tender and somewhat enlarged, icterus occurs in over 50 per cent., and the usual signs of sepsis prevail. The diagnosis is always difficult, but is based on *symptoms* suggesting liver abscess (though *without the findings* of solitary abscess) on the one hand and those of sepsis on the other. The treatment is practically futile, being supportive and rarely, if ever, operative.

IV. Affections of the Hepatic Artery and Vein.

These are of more pathological than clinical interest. *Infarction* of the hepatic artery with complete necrosis of the liver is very rare.

Aneurysm (*q.v.*) can seldom be diagnosticated. Pulsation is unknown; pain is nearly always present, icterus and a tumor occur in the majority of cases, ascites is uncommon and death usually results from gastro-intestinal hemorrhage.

Thrombosis of the hepatic veins is very infrequent, only 12 cases being reported. It may follow perihepatitis, or, less often, compression by a neoplasm, or fibrous obliteration of the inferior cava. Ascites and splenic tumor sometimes result. Suppurative thrombosis is always secondary to liver suppuration. Retrograde embolism (Heller) is mentioned under liver abscess.

Syphilis of the Liver (see SYPHILIS).

DIFFERENTIAL TABLE OF LIVER DISEASES.

	SIZE.	EDGE.	SURFACE.	CONSISTENCE.	ICTERUS.	ASCITES.	SPLEEN.	PAIN.
ACUTE YELLOW ATROPHY.	Very small.	Even.	Smooth.	Soft.	Intense and invariable.	None.	Enlarged in 66 per cent.	Usual.
PORTAL CIRRHOSIS.	Somewhat decreased. (Sometimes normal or enlarged.)	Sharp, (possibly finely nodular).	Smooth (possibly finely granular).	Hard.	Seldom (15 per cent).	*Usual* in 50 to 85 per cent.	Usual (75 per cent.).	Inconstant.
BILIARY CIRRHOSIS.	Very large.	Sharp.	Smooth.	Hard.	Constant.	None.	Constantly; very large.	None.
ABSCESS.	Enlarged (mostly right lobe or on convexity).	Uneven, if abscess is on lower part.	Nodular.	Soft even fluctuating.	Seldom (16 per cent.).	None.	None (save from sepsis).	Common.
TUMORS.	Greatly enlarged.	Nodular.	Nodular.	Hard.	Frequent (50 per cent.).	Frequent (50 per cent.).	Rare (in 11 per cent.).	More than in any other liver lesion.
HYDATID.	Enlarged (right lobe).	Usually even.	Nodular.	Semi- fluctuating.	Seldom (except in multilocular 80 per cent.).	None (save in multilocular).	None (save in multilocular 90 per cent.).	Seldom (except in multilocular).
FATTY LIVER.	Moderately enlarged.	Even.	Smooth.	Soft.	None.	None.	None.	None.
AMYLOID.	Greatly enlarged.	Sharp yet more rounded.	Smooth.	Hard.	None.	None (except late from cachexia or nephritis).	Usual.	None.
PASSIVE CONGESTION.	Moderately enlarged; may shrink late.	Rounded.	Smooth.	Firmer than normal.	Subicterus plus cyanosis.	Some from cardiac stasis.	None.	Tenderness usual.
SYPHILIS.	Enlarged, (save in rarer atrophic type).	Nodular or lobulated.	Nodular.	Hard.	Seldom (33 per cent.).	Seldom (save from atrophic type).	Frequent.	Frequent.

SES OF THE GALL-BLADDER AND BILE VESSELS.

(A). ICTERUS.

or jaundice is a symptom of various affections of the liver
vessels; it usually results from obstruction to the bile flow,
haracterized by staining of the skin, mucosæ, and urine by
bile pigments.

y.—Most cases of icterus are due to *obstruction* either within
the liver and biliary system; but slight obstruction is neces-
ause the bile is secreted under very low pressure. (*a*) It
lt from *hepatic diseases.* It is (i) frequent in carcinoma,
irrhosis, atrophic cirrhosis, multilocular echinococcus and
ongestion; (ii) infrequent in unilocular echinococcus, abscess,
ilis; (iii) absent in fatty and amyloid liver and in simple
itis. Pick, Liebermeister, and Minkowsky believe that
lly functional liver disturbance may produce icterus (para-
f bile, "diffusion-icterus" or paracholia) which is analogous
nt albuminuria (in its relation to Bright's disease). (*b*) Dis-
the *biliary tract* may be etiological factors, as (i) catarrhal,
, or purulent inflammation; (ii) calculous obstruction; (iii)
l and (iv) cicatricial occlusion; (v) malignant growths; (vi)
obstruction by ascaris, distoma, or hydatid vesicles. (*c*) It
ult from disease in *contiguous structures,* as (i) duodenal
atrix, catarrh, or malignancy; (ii) tumors of the colon or
ands; (iii) tumors or inflammation of the pancreas, stomach,
mentum, mesentery, and peritoneum, and (iv) rarely from
osis, or aneurysm. (*d*) The *so-called hæmatogenous* or *uro-*
us is a possible factor in the etiology. It was formerly main-
at toxic and toxæmic conditions break down the blood cells
is), and thus cause icterus (the cytohæmolytic icterus of
It is now thought that most of these cases are essentially
e (thickening of the bile or cholangitis of the smallest rad-
ough the obstruction of the small bile ducts within the liver
mplete. From a recent work by Eppinger, it appears that
ler bile radicles dilate and sometimes rupture, letting the
he lymph vessels which absorb it; he thinks the mechanical
n lies in the capillaries which were blocked by fibrin or bile
The jaundice in these cases is slight, bile pigment (bilirubin)
nd in the urine, and the stools are bile-stained. This type
) in various infections, relapsing fever, septicopyæmia, etc.,
rnicious anæmia, and (iii) in toxic conditions, poisoning by
us, chloroform, felix mas, and santonin.

neonatorum may be considered here. (*a*) The *physiological*
rs in 30 per cent. (to 80 per cent.) of children; it appears

on the second day, lasts two weeks and is never fatal; its cause is not clear, though breaking down of the blood, increased blood pressure within the liver and persisting patency of the ductus venosus are suggested. (*b*) The much rarer *pathological* type may be caused by sepsis, syphilis, Winkel's disease or still more exceptionally by congenital obstruction, gall-stones (10 cases collected by Skormin), acute yellow atrophy (7 cases) or catarrhal icterus (but 3 cases recorded). Glaister reported a family in which 6 cases of jaundice developed after birth. Krannhals recently published 26 cases of congenital "family icterus" most of which had an enlarged spleen.

Symptoms of Icterus.—Bilirubin, found normally in the bile, enters the blood in biliary obstruction, by the lymph vessels and possibly also by the bloodvessels, and produces the following symptoms:

1. *Staining of the skin and mucosæ* occurs in two or three days. The scleræ, skin and accessible mucosæ are yellow, and, in cases of long standing, dark brown. The skin itches because of its dryness, or because of the action of the bile on the nerves. Eruptions of urticaria or xanthoma may occur.

2. The *urine contains bilirubin* before icterus is seen in the skin. Adding nitroso-nitric acid to the urine in a porcelain dish or on filter paper gives the characteristic play of colors, of green, yellow, violet and red (Gmelin test). If the urine is agitated, the yellow color appears in its foam. Confusion is possible with melanuria, hæmaturia, urobilinuria or staining of the urine with rhubarb, picric acid, santonin, senna and chryosophanic acid. The subject of *urobilinuria* and urobilin-icterus has caused much discussion; urobilin is formed in the intestine by the reduction of bilirubin, whence it is absorbed and excreted by the urine; it never stains the skin, and icterus results from bilirubin alone. To reconcile the apparent inconsistencies in the urinary findings and in various statements, it may be said that there are three classes of cases; (*a*) in complete biliary obstruction with icterus, only bilirubin is found in the urine, for none of it reaches the intestine to be converted into urobilin; (*b*) in partial obstruction, bilirubin is found in the blood and urine and also some urobilin, which is produced in the intestine from the bilirubin, for there is much intestinal fermentation; (*c*) in slight obstruction, there is no bilirubin but much urobilin in the urine. *Albuminuria* may occur or *cylindruria* alone, and the hyaline casts or desquamated epithelium are then bile-stained. The *sulphates*, which normally constitute 14 to 25 per cent. of all the sulphur output, may be increased to over 40 per cent. *Indicanuria* is often marked and is due, like the sulphate increase, to greater intestinal putrefaction.

3. *The digestive tract* suffers from the exclusion of bile. The tongue is coated, the breath has a disagreeable odor and the mouth may have a bitter taste, which is due to toxæmia. There may be an especial aversion to fats and sometimes to proteids, and there may be nausea. The *abdomen is distended*, which is thought to be due to the absence of the antiseptic action of the bile, though the importance of this

action has been overestimated. Starchy food is not wholly absorbed, albumin digestion is somewhat impaired and fat is found in large amounts in the stools (*steatorrhœa*). The function of the pancreas in this respect is far more important than that of the bile. Vegetables are absorbed better than animal fats. The normal amount of fat found in the fæces is 7 to 10 per cent., and this is increased in icterus to 55 or 78 per cent. The *stools* are *clay-colored*, *constipated* and *stinking;* they may shimmer when the fatty soap crystals are abundant; they are also *dry* (the bile in the fæces amounts normally to nearly a quart a day). If the obstruction is incomplete—as in many intrahepatic diseases—the stools are merely somewhat lighter than normal; a nitrogenous diet may impart a brownish color to the fæces. The fæces may appear deceptively dark if voided with the urine which lends a yellow color to their surface.

4. *Cholæmia* (hepatargia or hepatic insufficiency) may finally result. It was once thought to be due to the cholestearin, or bile salts, but more probably results from injury to the liver cells which then fail to destroy the usual intestinal toxins and those formed in excess by the increased putrefaction. Its symptoms rather resemble those of acute yellow atrophy, *i. e.* nervous excitation followed by sopor, irregular breathing, or cutaneous and other hemorrhages. Most subjects of icterus are depressed, irritable, sleepless and somewhat emaciated. The hemorrhages are thought by Mayo Robson to result from coincident pancreatic involvement (diminished lime salts or the glycerine which is formed by fat necrosis of the pancreas).

5. There may be *other symptoms*. (*a*) The *heart* rate is *slow*, particularly in catarrhal forms or recent cases and in the absence of pain, fever, and intoxication; it may go as low as 21 (Frerichs); it has been said that this is due to degeneration of the heart muscle or action of the bile-salts on the intracardiac ganglia, but it is probably caused by vagus stimulation, for atropine will raise the rate (Weintraud). The arterial pressure is low and the capillaries wide. A functional heart murmur may develop; this is usually caused by myocardial weakness. Potain holds that the lung capillaries contract reflexly, thereby increasing the work of the right heart and causing its dilatation. The *respiration* rate is *decreased*, even to 7 or 9 a minute; the temperature is usually normal. (*b*) The *blood* shows the bile stain, and in severe cases delayed coagulation, anæmia, and leukocytosis. The undiluted or diluted blood may give a partial Widal reaction (see TYPHOID). (*c*) The *liver cells* may suffer mechanically and chemically from bile stasis, and may be smaller, altered in form, pigmented, necrotic and bile-stained. The gross and minute anatomy varies with the cause. The bile vessels are often dilated, sometimes thickened, and occasionally ruptured. The condition of the gall-bladder will be discussed under gall-stones and pancreatic cancer. As stated under biliary cirrhosis, it is questionable whether simple biliary stasis ever causes induration of the liver. (*d*) The spleen is occasionally enlarged, especially in children, from infection or portal stasis, but splenic tumor

is no part *per se* of bile stasis. (*e*) The *tissues* most *stained* are t skin, mucosæ, fasciæ, intima and connective tissues; the fluids mo stained are the urine, serous transudates and purulent exudation The milk, sweat (the fœtus, in pregnant women), and pneumon sputum are sometimes stained, and the tears, saliva, mucus, glandul and epithelial structures (except the renal cells and lowest layers the rete Malpighii), muscles and nervous tissues (except in the mo severe type of icterus neonatorum) are seldom icteric. (*f*) Xanthops yellow coloring of objects seen, is fairly common, and is due to t action of bile pigment on the nervous tissues. The explanation nyctalopia and hemeralopia is uncertain. (*g*) Xanthelasma multipl develops in some cases. Twenty-three cases were collected by t London Pathological Society in 1882. Futcher in 1905 reported thr cases in women about forty years of age; 80 per cent. of report cases of xanthelasma occur in chronic icterus (in women more fr quently than in men), due to gall-stones, stricture of the bile-duct cirrhosis, hydatids, and cancer. They are often symmetrical, oft begin on the eyelids or the folds and creases of the skin, in one ca were found in the bile ducts, rarely disappear and are probably d to some toxic substance, though Williams believes they are epitheli in origin. Some few cases begin before puberty and are probab hereditary.

Diagnosis.—A diagnosis is easily made. Icterus may not be notic in artificial light, for daylight is absolutely essential for its certai detection. It is seldom confused with anæmia, cachexia, Addison disease or the pigmentation of vagabondism. Pingueculæ a often mistaken for scleral staining, but only on superficial exami ation.

The *prognosis and treatment* cannot be considered, as icterus is b a symptom. The details depend on the basic affection. (See CATA RHAL JAUNDICE for treatment in general.) The patient may recov after four years (Budd); cases of icterus lasting six years (Murchiso Barth, Bosnier), ten to twenty-five years (Legendre, Galliard) or eve fifty-two years (Masing) have been reported.

(B). CHOLELITHIASIS.

Gall-stones are found in 6 to 10 per cent. of autopsies. Among t many literary contributions to this subject are those of Nauny Langenbuch, Kehr, Riedel, Lange, Mayo Robson, Keen, Feng Murphy and Mayo.

Etiology.—1. **Immediate Cause.**—The actual cause is catarrh inflammation of the gall-bladder or bile vessels, which promotes t formation of (or actually secretes) cholestearin and lime. Stagnati of bile is merely a predisposing cause. Bacteria play an importa role, especially the typhoid (found in 33 per cent.) and colon bacil they have been found in calculi, and cholelithiasis has been expe mentally produced by their inoculation; the Bacillus typhosus may live seven years in the gall-bladder.

2. **Predisposing Factors.**—(*a*) *Age;* 50 per cent. of cases occur in persons *over forty years* of age; the affection is rare in those under twenty years but has been seen in the young and even in the new-born. (*b*) *Sex;* 70 to 80 per cent. occur *in women,* in whom pregnancy, menopause, lax abdominal walls, enteroptosis, and constipation, sedentary life, and lacing, are regarded as promoting factors; Bollinger and Bother found that 40 per cent. of women, who have gall-stones, also have the corset liver. According to Naunyn, gall-stones are found in 25 per cent. of autopsies on women over sixty years; 90 per cent. of women affected have borne children. (*c*) *Other possible factors* are *sedentary habits, obesity,* splanchnoptosis, luxurious living, insanity, cancer of the stomach, biliary stasis, and foreign bodies in the biliary tract; Kehr holds that stone-formation is induced by long intervals between meals. Cholelithiasis is more common in cool than in warm climates.

Properties of Gall-stones.—(*a*) *Site;* they are almost invariably formed in the gall-bladder; very rarely do calculi develop in the intrahepatic ducts as reported in McArthur's recent case. (*b*) *Size;* they range from the size of sand or gravel to that of an egg; a calculus 12 inches in length is recorded; the heaviest weight reported is over four ounces (135 gm. Ritter). (*c*) *Form;* they are usually round and rarely spinous; when multiple they are polygonal and facetted. Intrahepatic calculi may be cylindrical or branching like coral (Cruveilhier). (*d*) Their *number* probably averages a dozen; according to Riedel 44 per cent. are single; Naunyn reports 5,000 in one case, and Otto 7,802 stones in another case. Chopart instances a case in which the liver could scarcely be cut because of the numberless stones in the intrahepatic ducts. (*e*) *Consistence;* gall-stones can usually be indented by the finger. (*f*) The *time required* for their growth is about six months. (*g*) *Chemistry;* the most common form consists of *cholestearin* and *bile-pigment* (bilirubin and biliverdin) with some lime as a cementing substance; these calculi are small, numerous, usually yellow, laminated and contain about 75 per cent. of cholestearin. *Cholestearin* stones are not common; they are translucent, become opaque when dry, are light in weight and color, and are combustible; they present a crystalline fracture like that of camphor, they dissolve in ether and alcohol, from which the crystals are precipitated and produce a red tinge on addition of chloroform and sulphuric acid; cholestearin comes not from the bile but from the inflamed mucosa of the gall-bladder. *Pigmentary stones* are also uncommon; bilirubin imparts a brownish and biliverdin a greenish tint to them. *Calcium carbonate* stones are rare, are grayish in color and heavy. Stones in the liver are constituted of calcium and bilirubin as in Lewis L. McArthur's recent case. Traces of magnesium, bile acids, fatty acids, iron, and copper have been found. The nucleus may consist of mucin, bacteria, epithelium, blood-clot, cholestearin, calcium, bile pigment or rarely of foreign bodies.

Symptoms.—Kehr, Riedel and Paulsen assert that 95 per cent of cases of gall-stones have no symptoms, especially in elderly subjects with atrophic gall-bladders. Naunyn states that gall-stones are found at autopsy in 25 per cent. of women over sixty years of age. Stones may be felt or may crepitate in the gall-bladder, or be found in the fæces, especially in old people with lax tissues, without any subjective symptoms.

An *attack of biliary colic* embraces several important elements. (*a*) Colic is usually *sudden* in onset and very frequently begins at night. The pain is great, and many women state that it is more severe than labor pains. It begins in the right upper abdominal quadrant and may radiate to the back, navel, hypogastrium, left side, and even to the arms, legs, testes, or chest. Irregular or large stones cause the greatest agony which is controlled only by large or repeated hypodermics of morphine. The pain usually endures for minutes to a few hours, and may soon recur. According to Riedel colic is absent in 20 per cent. of cases, which implies that the stones are small or the tissues are lax. The cause of biliary colic is mechanical,—the attempt of the stone to migrate produces the spasm of the cystic and common ducts during its passage but some consider that inflammation is the chief factor; Kehr maintains that cholecystitis starts the stones moving by the exudation it induces. The author has seen violent colic result from this mechanism as well as from cholecystitis without stones. It is thought that quiescent gall-stones may be started onward by palpation of the gall-bladder, trauma, muscular exertion, pressure by corsets, particularly after reduction of flesh, menstruation, or delivery. (*b*) *Reflex nausea* and *vomiting* are frequent; Kehr thinks that they indicate cholecystitis (Mierzkowski found the bile sterile in only 22 per cent.). Vomiting relieves the pain, and this frequently causes an erroneous diagnosis of gastralgia. Torsion of the cystic duct may cause vomiting but not colic. (*c*) *Chills and fever* occur in 60 per cent. and probably indicate bacterial inflammation (though they are possibly reflex). (*d*) The *gall-bladder* is *always tender*, there being hyperæsthesia over the ninth costal cartilage or over a point two-thirds of the distance between it and the navel. In 33 per cent. of cases the gall-bladder is palpable. The right rectus muscle is rigid. Boas and Gruh describe a right-sided hyperæsthetic zone which lies between the spine and posterior axillary line and at the level of the lower dorsal or upper lumbar vertebra. (*e*) *Icterus*, which is so often emphasized as a diagnostic point, is present in but 10 to 20 per cent. (Riedel and Kehr), and in but 14 per cent. of Murphy's series. It may be mechanical or inflammatory; Deaver is inclined to refer it to inflammation in the pancreas (*v. i.*). (*f*) *Other symptoms* of the paroxysm are rapid pulse, sweating, prostration, concentrated urine, constipation, abdominal retraction and, after the seizure, marked prostration. In some cases convulsions or other hysterical manifestations may be precipitated.

Attacks of migraine are not infrequent (Kehr). There may be reflex coughing or reflex contraction of the pulmonary capillaries, which puts extra work on the right heart and causes its dilatation. The patient may suffer actual collapse, and in nine cases death has occurred in the attack (Naunyn). Glycosuria, albuminuria, leukocytosis, and splenic swelling are probably inflammatory in origin. The stone may fall back into the gall-bladder or pass into the bowel if it measures not more than 1 cm. It is detected by washing of the stools through a cloth; confusion with lumps of olive oil, which may have been given to relieve the colic, is unnecessary. The passage of a round calculus suggests, but does not prove, that it is the only one. Stones passing into the bowel may be dissolved, unless coated with cholestearin; this may explain why stones are not more often found in the fæces. The great importance of gall-stones lies in their complications.

Complications.—Complications may be grouped as *mechanical* or *inflammatory* but as this involves descriptive repetition, both forms may be combined topographically. Such complications as liver abscess or pyæmia may develop from stones which were without any clinical symptoms.

1. Gall-bladder.—Mechanical complications include pressure on the duodenum or pylorus and rupture of the gall-bladder, which, however, are generally inflammatory; pressure on the common duct; hyperchlorhydria and hypersecretion; and cancer of the gall-bladder (*q.v.*). Inflammatory complications are more serious; all forms and sequences of cholecystitis (*q.v.*) may develop, as hydrops, empyema, gangrene, ulceration, deformity, hemorrhage, atrophy, calcification or liver abscess; also pylephlebitis, adhesions producing pyloric obstruction with hyperchlorhydria, dyspepsia, and sometimes hemorrhage into the stomach; adhesions causing duodenal obstruction; and peritonitis, either local (near the gall-bladder, or subphrenic abscess) or diffuse; Courvoisier reported 49 cases of the localized and 70 of the diffuse type. A friction rub may be heard over the gall-bladder a day or two after the colic. The distended gall-bladder rarely exceeds the size of the fist, is pyriform, can be moved laterally and moves with the liver, from which it may seem to be separated by a groove; it may be sometimes confused with appendicitis, the kidney, ovarian cyst or ascites, and has even been found in a femoral hernia.

2. Cystic Duct.—Mechanical impaction may cause icterus by inducing spasm or catarrh of the common duct (in 10 to 15 per cent., Riedel) or by directly compressing it. The duct may become twisted, stenosed, or obliterated.

3. Common Bile Duct.—Its mechanical occlusion is usually but not invariably preceded by colic, and impaction occurs oftenest at its lower end (50 per cent. Courvoisier, or 67 per cent. Robson), is caused most frequently by one stone, though as many as 88 have been found (Mayo Robson). Permanent occlusion by stone in the common duct, or in the cystic duct pressing upon it, causes marked icterus, usually without sepsis. The occlusion may be partial or intermittent, by a ball-valve action of the stone, described by Osler and Fenger, which allows some bile to enter the gut and, unfortunately, some bacteria to enter the duct, thus adding bacterial to mechanical injury. In isolated cases there is no icterus but usually the icterus is sudden in onset and slow in subsidence. In partial or intermittent obstruction there is often the intermittent hepatic fever, which was first described by Charcot and later clearly defined by Osler. Budd well compared to urethral fever. During the paroxysms the fever rises to 103 or 10 often with chills, sweats, leukocytosis, vomiting, and increased tenderness, pain, and icterus. The spleen may swell as well as the liver from cylindrical or saccular dilatation of the bile channels and from cholangitis. Osler has correctly insisted that this group of symptoms occurs without suppurative cholangitis, though of course with infection. The gall-bladder is not enlarged, but is usually shrunken, following Courvoisier's law; Courvoisier (1890) found the gall-bladder enlarged in 92 per cent. of 100 cases of non-calculous obstruction of the common duct, and shrunken in 80 per cent. of calculous obstruction (87 cases); this is caused by infection (Hanot); Eichlin in 139 cases of non-calculous obstruction, found the gall-bladder dilated in 87 per cent., and contracted or normal in 6.5 per cent. each; in 172 cases of calculous obstruction, the gall-bladder was contracted in 66 per cent., normal in 19 per cent. and dilated in 15 per cent. In some cases suppurative cholangitis may complicate stones of the common duct with remittent fever (rather than the above described intermittent fever), enlarged liver, septic symptoms and more intense and more rapidly fatal course. Mechanical dilatation may distend all the bile-vessels and these may be seen under the capsule; ascending cholangitis may cause subcapsular abscess. Cicatricial stenosis of the common duct is rare; pylephlebitis may develop. Malaria is excluded by examination of the blood; malignancy will be considered under tumors of the pancreas, though in obstruction of the common duct, the nutrition is generally but little impaired. Practically, special stress may be placed on (*a*) the periodic fever, (*b*) the icterus sudden in onset an subsiding slowly, and (*c*) tenderness (without pain over the liver).

4. Ampulla of Vater.—Calculous retention in the ampulla m obstruct the flow of the pancreatic juice, though the accessory du may carry it into the intestine; retention may allow bile to enter t pancreatic duct, and result (*a*) in acute pancreatitis, caused by b in the pancreatic ducts (Opie); (*b*) in chronic induration of the he of the pancreas, resembling malignancy (Riedel, Robson, Barlin (*c*) in pancreatic fibrosis with pancreatic calculi due to stasis and inf tion; or (*d*) rarely in glycosuria.

5. Intestinal Obstruction (*q.v.*).—Two hundred and fifty c have been collected. The stone usually ulcerates its way into bowel from the biliary channels or gall-bladder. The obstructi often incomplete and may not occur for some time, sometimes re ing new layers of magnesia or phosphates. According to Courvo the obstruction is in the ileum in 66 per cent., duodenum in 21 per ileocæcal valve in 10 per cent. and sigmoid in 3 per cent. About per cent. of cases die.

6. Fistulæ.—Naunyn assembled 384 cases. The external form constitutes about 50 per cent. of cases (Naunyn and Courvoisier); they are most often located near the navel, and about 40 per cent. of them heal. In the duodenal form (28 per cent.) the fistula is between the duodenum and gall-bladder and less often between the common duct and the gall-bladder. In 13 per cent. there is a fistulous opening into the colon. Courvoisier and Graham collected 34 cases of bronchial fistula. In rare cases, stones enter the stomach (five cases recorded, Snively, 1903), portal vein, hepatic artery, kidney, urinary bladder (200 gall-stones were evacuated in the urine in the case of Barraud-Palletan), pericardium, retroperitoneal tissue, vagina or even the pregnant uterus.

7. Renal Calculus, Glycosuria, Etc.—In 700 cases of gall-stones, Kraus found the following complications: renal calculus in 6 per cent., glycosuria in 7 per cent., diabetes in 4 per cent., and gout in 2 per cent.

Diagnosis.—The diagnosis is easily made when colic, vomiting, tenderness of the gall-bladder and icterus are present, but icterus is absent in at least 80 per cent. of all cases. The *x*-rays may produce no shadow in cholestearin calculi.

1. The Colic.—This is most apt to be confused with (*a*) *gastralgia*, for both conditions are relieved by vomiting; due regard for other symptoms of gall-stones, and the habit of doubting every diagnosis of gastralgia, will save many mistakes. (*b*) A diagnosis of *hepatic neuralgia* should always be made with reserve, though pain near the liver does occur in neurotics. (*c*) *Gastric or duodenal ulcer* (*q.v.*), and (*d*) hyperchlorhydria and hypersecretion (*q.v.*) are easily distinguished. (*e*) *Lead colic* has other characteristic symptoms, such as the gingival lead line, obstinate constipation, and often neuritic manifestations. (*f*) *Tabetic crisis* is attended by the Argyll-Robertson pupil, absent knee-jerk, and ataxia. (*g*) Renal colic (*q.v.*) is distinguished by pain reflected down the ureter, hæmaturia, and pyuria, and Dietl's crisis in floating kidney, by bimanual examination of the abdomen. (*h*) *Mucous colic* (colitis) is differentiated by the characteristic stools and neurasthenic symptoms. (*i*) *Appendicitis* is characterized by its four cardinal symptoms (*q.v.*); Ochsner finds 10 per cent. of his appendicitis cases complicated by gall-stones and 33 per cent. of his gall-stone cases by appendicitis. (*j*) *Acute pancreatitis* (*q.v.*) which may complicate gall-stones, causes greater collapse and more frequent intestinal obstruction. (*k*) *Febrile syphilis* may simulate gall-stones (Riedel) as may also (*l*) the abdominal crises occasionally observed in angioneurotic œdema.

2. Other Affections of the Gall-Bladder and Bile Ducts (see BREWER'S TABLE).

Pathological condition	Pain.	Fever.	Vomiting	Jaund	
I. CALCULOUS DISEASE.					
(a) Stone in healthy gall-bladder, ducts free.	No.	No.	No.	No.	May be present from large number or size of stones.
(b) Stone in healthy gall-bladder, cystic duct temporarily obstructed.	May be absent; generally present during obstruction; paroxysmal.	No.	May be present when colic occurs.	No.	May be present from distention.
(c) Stone impacted in cystic duct.	No.	No.	No.	No.	Present; may attain large size.
(d) Stone in hepatic duct.	Frequently present; irregular type.	Occasionally present.	May be present during pain.	Frequent.	No.
(e) Stone in common duct; acute obstruction.	Present; acute paroxysmal radiating to back.	Generally present.	Present.	Present.	No.
(f) Stone in common duct; movable; chronic.	Periodic attacks of acute radiating pain.	Present with chills and sweats.	Present.	Present; intermittent.	Rarely present.
(g) Stone in common duct; impacted; chronic.	May be absent; frequently present early; may be intermittent; variable.	May be present; variable.	Often present.	Present; progressive; may vary in intensity.	Rarely present.
II. INFLAMMATORY DISEASE.					
(a) Cholecystitis subacute.	Present; paroxysmal during periods of cystic duct closure from stone or swollen mucous membrane.	Present during attacks of colic.	May be present.	No.	Present during attacks of cystic duct obstruction.
(b) Cholecystitis acute.	Acute paroxysmal radiating pain; extending to back and shoulder; may be very severe.	Present, with chills and sweats.	Present; often severe.	No.	Present; tenderness; often muscular rigidity.
(c) Cholecystitis chronic (empyema of gall-bladder).	Severe radiating pain at first; may disappear later; tendency to recur.	Present; severe at first, may diminish later.	Present at first.	No.	Present; with tenderness; may attain large size.
(d) Cholecystitis in previously diseased and contracted gall-bladder.	Present; often severe; paroxysmal.	Present; often with chills and sweats.	Present.	No.	No (occasionally present due to pericystic exudate).
(e) Cholangitis of hepatic and common ducts.	May be absent; generally present when obstruction exists, or severe infection; tenderness and pain over liver in intrahepatic cholangitis.	Present; chills; sweats; severe prostration; general sepsis.	Present.	Present; variable.	No.
III. NEW GROWTHS.					
(a) Carcinoma of gall-bladder.	No; may occur late in disease.	No.	No.	Present late (portal glands).	Hard, irregular, movable tumor at first, later diffuse infiltration.
(b) Tumor of cystic duct.	No; may occur late.	No.	No.	No.	Present when obstruction exists.
Tumor of hepatic [illegible] duct.	No; may occur late.	No.	No.	Present; progressive.	May be present from distention with bile.
	may occur late.	No.	No.	Present; progressive; may become extreme.	Present; generally from distention with bile.

OF THE GALL-BLADDER AND DUCTS.

Urine.	Stools.	Liver.	Spleen.	Ascites.	Remarks.
Negative.	Normal.	Not enlarged.	Not enlarged.	No.	Generally discovered by a dent; often unrecognized.
Negative.	Normal.	Not enlarged.	Not enlarged.	No.	All symptoms promptly reliev as soon as obstruction remove
Negative.	Normal.	Not enlarged.	Not enlarged.	No.	Hydrops of gall-bladder often unrecognized.
Contains bile pigment at times.	May be clay colored if obstruction occurs.	Frequently enlarged.	Not enlarged.	No.	Diagnosis extremely difficult; symptoms generally due to coexisting cholangitis.
Contains bile pigment.	Clay colored.	Not enlarged.	Not enlarged.	No.	Symptoms rapidly disappear when stone passes papilla.
Contains bile pigment.	Clay colored.	May be enlarged during attack.	May be enlarged from pressure of stone on vein.	No.	"Fievre intermittente hépatique" of Charcot resembles malaria; all symptoms disappear during interval.
Contains bile pigment.	Clay colored.	Enlarged.	May be enlarged.	No.	Condition may remain for many years; may only be jaundice with digestive disturbances and loss of weight; history of previous attacks (?). Ascites may be present from pressure of large stone or possibly from hydræmia.
Negative.	Normal.	Not enlarged.	Not enlarged.	No.	Tenderness over gall-bladder; tendency to recurrence; generally associated with stones in gall-bladder.
May contain albumin and casts.	Normal.	Not enlarged.	May be enlarged (sepsis).	No.	May follow typhoid or other septic diseases; onset often sudden; rapid development of severe symptoms resembling appendicitis; may be necrosis of walls of gall-bladder with perforation; local or general peritonitis.
Negative.	Normal.	Not enlarged.	Not enlarged.	No.	Frequently follows acute cholecystitis; occasionally becomes quiescent, presenting practically no symptoms.
Negative.	Normal.	Not enlarged.	May be enlarged (sepsis).	No.	Generally tenderness over gall-bladder area, but no tumor; local peritonitis; diagnosis often extremely difficult.
May contain bile pigment, albumin, and casts.	May be clay colored.	Enlarged.	Enlarged (sepsis).	No.	Often follows severe infections of gall-bladder; generally associated with stones in common or hepatic ducts; severe sepsis; generally fatal in virulent infections (streptococcus).
Negative (at first).	May be clay colored late.	Enlarged late in disease.	May be enlarged late (pressure on vein).	Present late.	Digestive disturbances, progressive loss of weight and asthenia; cachexia; rapidly fatal.
Negative.	Normal.	Not enlarged.	Not enlarged.	No. (?)	Very rare; both benign and malignant growths have been reported; diagnosis difficult.
Contains bile pigment.	Clay colored.	May be enlarged.	Not enlarged.	May be present late.	Very rare; diagnosis difficult.
Contains bile pigment.	Clay colored.	Enlarged.	May be enlarged late.	Present late.	Malignant tumors most common; chronic interstitial pancreatitis from previous infection of biliary passages may remain after cause has disappeared; enlarged portal glands; Hodgkin's disease.

Prognosis.—The prognosis is difficult to formulate. Stones ma pass without recurrence of colic and the passage of smooth calculi ma justify some hope that they are the only ones. The first attack usually the worst. Fever, infection, cancer, and obstruction of th common duct and intestine must be covered in forecasts to patien refusing operation. Riedel held that 10 per cent. recover spontaneous under expectant treatment, while in 90 per cent. of cases surgic intervention is indicated.

Treatment.—**1. Prevention.**—Exercise and deep breathing aid th flow of bile. Lacing is a factor to be regarded. Plenty of wat increases the secretion of bile. Digestive derangements should b corrected. Alcohol is contra-indicated. Kehr believes that food shou be taken in small amounts at frequent intervals and should be chief nitrogenous, in order to favor the secretion of bile. The predisposi gall-bladder inflammation seldom comes under the physician's care, b in suspicious cases, sodium salicylate, gr. x to xv t. i. d. given alterna weeks, acts as a cholagogue—increasing the bile two or threefold—a antiseptic. Mercurials seem to stimulate bile formation less than aid in its evacuation. Potassium iodide and mild salines (sodiu phosphate, ʒss to j) are beneficial.

2. Colic.—Hypodermics of morphine must be given, gr. $\frac{1}{4}$ bei combined with nitroglycerine gr. $\frac{1}{100}$ and (for the first injection in t attack) with atrophine gr. $\frac{1}{150}$; repeated doses of morphine a nitroglycerine are often necessary. Inhalation of chloroform is in cated in severe cases until the morphine acts, but none of these dru should be left in the possession of any patient. A full hot bath a hot fomentations over the liver are valuable accessories. Drink freely of warm water makes the vomiting easier, which in turn relie the pain.

3. Medical Treatment.—This in no way influences the *solution* *gall-stones.* Durande's solvent (oil of turpentine 1 part to ether parts) is useless, and, like chloroform, is only antispasmodic. *Oli* oil at best only facilitates their final passage. Salicylates, calome salines (*v.s.*) and various "cures," as at Carlsbad, Vichy, Marienbad Kissingen, Las Vegas or Bedford Springs, may be useful in quietin concomitant or causative inflammation. Local massage is obviousl injurious.

4. Surgical Treatment.—The author believes that gall-stones shoul be operated on when recognized, thus saving time, suffering, and dange The following *indications* are given by Kehr: "(*a*) I recognize that t latent state ensues in many cases of cholelithiasis after rest cures alkalies and in certain cases is permanent. Particularly in the so-ca cases of chronic recurrent cholecystitis a regular rest cure carried o Carlsbad, Neuenahr, or even at home, will often so mitigate the c that all reason for operation will be absent. But I doubt wheth absolute cure will frequently occur, *i.e.*, the passage of all the st by medical means. It is my opinion that we should never endea force a passage of the stones; it is much better to seek to rende quiescent in the gall-bladder and secure a restitution of the inflammatory processes. The continuous use of hot applications for weeks, in conjunction with rest in bed and the use of Carlsbad waters, will give the best results. "(*b*) Riedel's theoretical justification for early operation—the removal of the calculi while they remain in the gall-bladder—holds good now as ever, as in many cases an early operation is the only salvation for the patient against such grave dangers as perforation, cholæmia and carcinoma. Universal application of early operation, however, in general practice, is absolutely impossible, and Riedel's indications for operation are, for this reason, without practical value. "(*c*) I decline operative interference if the attacks run a mild course, and in the intervals when complete latency exists, *i.e.*, entire absence of sensitiveness in the region of the gall-bladder. "(*d*) Acute obstruction of the choledochus is, with rare exceptions, to be treated medically. Should cholangitic symptoms appear, or should the jaundice be prolonged sufficiently to induce loss of strength and absolute loss of appetite, operation should be performed. "(*e*) Operation is necessary when frequent colics occur, even without jaundice or the passage of stones, which damage the general health, deteriorate the earning capacity, and destroy the pleasure of living. "(*f*) Cases of jaundice which are always accompanied by the passage of stones, belong to the physician; should the attacks multiply, the patient become vitally depressed, and no hope be present for the passage of all the stones, the propriety of the operation is established. "(*g*) Hydrops, empyema of the gall-bladder, and pericholecystitic abscesses belong to the province of surgery. In those rare exceptions in which a sterile hydrops gives rise to a few symptoms, the patient may be ignorant of his swollen gall-bladder until distress begins and multiplies. "(*h*) Surgical treatment should not be postponed too long after the failure of a Carlsbad cure. "(*i*) Gall-stone patients who have become addicted to the use of morphine must, under all circumstances, be operated upon. During the after-treatment the best opportunities are afforded for curing the opium habit. "(*j*) Treatment of carcinoma of the gall-bladder can only be of lasting benefit if the case is operated upon early. Every one, however, dreads an early operation, and late operations are not of much use; therefore the complete cure of the evil will but rarely be effected. "(*k*) Those suffering with chronic jaundice, which is not dependent upon stone in the common duct or incurable disease of the liver, must be operated upon, at the latest, three months after the appearance of the icterus, for it not infrequently occurs that in place of a suspected carcinoma of the head of the pancreas, a curable chronic interstitial pancreatitis will be found. "(*l*) The decision for operation will be rendered easy for both physician and patient upon demonstration of tumor of the gall-bladder, a swollen liver, and the appearance of jaundice and fever. We may also operate on cases without demonstrable local lesions of the gall-bladder and liver, which exhibit severe and continuous symptoms which do not prove amenable to internal medication. We find in such cases, particularly often in men, adhesions of traumatic origin without stones. "(*m*) The sequelæ of cholelithias purulent cholangitis, abscess of the liver, peritonitis due to perforatio subphrenic abscess, severe pyloric and duodenal stenosis, and ga stone ileus, must be treated surgically. "(*n*) Concluding: To prese general indications for a gall-stone operation is not readily possib One must decide each case on its own merits. Men, particula corpulent ones, do not bear the operation well. Women who have borne children lend themselves particularly well to this surgical procedure. If possible one should refrain from operating in cases of diabetes, arteriosclerosis, chronic nephritis and diseases of the lungs and heart."

In Mayo's 1,000 operated cases (1905), the mortality was but 1 per cent. in 416 uncomplicated cases, while those complicated by suppuration or cancer brought the average mortality to 5 per cent.; the death rate in operations on the common duct was 11 per cent. These figures appear to the writer as the best possible argument for early operation. Cholecystotomy was attended by a 2.5 per cent. mortality and it is therefore the better operation, as cholecystectomy entailed 4.3 per cent. of fatalities. In Kehr's last 500 cases (1904), the mortality was 3 per cent. in uncomplicated cases and 13 per cent. with cancer and cholangitis; stones were left in 2.5 per cent., hernia developed in 3 per cent. and adhesions and inflammatory colic occurred in 5 per cent. (*versus* his earlier figures of 4, 7 and 17 per cent. respectively).

(C). CHOLECYSTITIS.

Etiology.—Inflammation of the gall-bladder may occur before, after, with or without inflammation of the bile passages. (*a*) *Predisposing factors are gall-stones* (especially large ones which cannot pass, which cause 65 per cent. of cholecystitis and cholangitis (Dominici) or 90 per cent. in other statistics), bile stasis, trauma, foreign bodies, or previous attacks. (*b*) The *exciting cause* is *bacterial;* the colon, typhoid, pyogenic, pneumococcus, cholera or influenzal organisms may be responsible. They may ascend the bile channels or enter with the blood current. Inflammation from typhoid infection (Louis and Andral) may occur without actual typhoid, or may develop even twenty years later (Osler).

Symptoms.—The symptoms vary with the intensity of infection, which is almost synonymous with the type (simple, membranous, suppurative, phlegmonous, ulcerative, or gangrenous). In light forms the symptoms may not be recognized; this corresponds to post mortem adhesions in cases with no history of cholecystitis. In other cases, perhaps more severe ones, symptoms are obscured by the causative typhoid, or by gall-stones. In the average case, there is (*a*) pain over the part, which may be severe or paroxysmal and may radiate towar the appendix or back. (*b*) *Tenderness* as in gall-stones; it is ofte first general and then local, and the right rectus is tense; it is clear a jab over the part during inspiration. (c) *Fever, increased vomiting* or nausea; fever is absent in mild infections: in olecystitis, may be confused with typhoid relapse (Musser). may be a *palpable gall-bladder.* Its contents may be puru-ema cystidis felleæ) or serous (hydrops cystidis felleæ). ay enlarge the gall-bladder to the size of the fist, or in ses so that it weighs 50 to 60 pounds, contains thirty to s, and fills the entire abdomen; experienced observers have ch cases for ascites; the fluid is alkaline or neutral, serous If very tense the gall-bladder may not fluctuate or if very t be palpable (see FLOATING KIDNEY). There may be pus -bladder without enlargement. In chronic cases it may) *Severe sepsis* may intervene when there is diffuse sup-olangitis or pylephlebitis; this is evidenced by septic fever, cytosis, colon bacilli, pneumococci or other organisms in the len liver and spleen, nephritis, and ulcerative endocarditis. nconstant.

s.—Many cases with cholangitis die in spite of operation. may subside, but recurrence and formation of gall-stones are Under expectant therapy, the outlook is serious in severe perforation, localized or general peritonitis, adhesions (in .), and duodenal or pyloric obstruction may result.

nt.—Rest, milk diet, local heat and morphine may suffice ses, but in the more severe infections drainage is indicated.

HOLANGITIS, CATARRHAL ICTERUS, CONGENITAL OCCLUSION.

tive cholangitis usually is diffuse, co-exists with suppurative s and results from gall-stones with infection. It may occur or foreign bodies in the tracts and in pylephlebitic liver he infection is usually ascending but may follow infection of vein into which their bloodvessels empty. The symptoms itic sepsis have been described under cholecystitis. Drain-cated.

atarrhal jaundice (cholangitis catarrhalis, icterus simplex duodenalis) results from gastroduodenal catarrh which lling with obstruction of the papilla, and probably catarrhal on of the lower portion of the common duct; as few cases topsy, the latter point is not entirely proven, though a small ucus usually occludes the opening. Its usual causes are those of acute gastritis or enteritis. Favoring factors ssive congestion (cardiac or portal), acute infections, toxic phosphorus), nephritis, other diseases of the liver or bile rhosis, cancer, stones, parasites), and very rarely psychic e or menstrual derangement. Epidemic forms occur or h seem to be a separate type of infection (*v.* WEIL'S DISEASE).

Symptoms.—*Gastro-intestinal catarrh* occurs first in many cases and after a few days to a week or so *icterus* appears, with its usual symptoms (*v.* ICTERUS), sudden decolorization of the stools, and bright yellow staining of the skin and urine. However the physician must not expect to always find an antecedent gastroduodenitis, because a primary infectious catarrh of the bile ducts is equally frequent. Other findings are inconstant; the liver is not enlarged at first, but may become slightly swollen later; there is no pain over the liver or ducts, emaciation if present is slight, the spleen and gall-bladder are seldom enlarged, and the *diagnosis* depends entirely on the mode of onset in young, healthy subjects, sequence of symptoms and exclusion of other causes of obstruction. In advanced life, catarrhal jaundice is uncommon and gall-stones and cancer are always to be thought of first.

Prognosis.—The prognosis is almost always good, the jaundice lasting four to eight weeks. The author has seen cases clear in a week and others last five months, though a course of over four to eight weeks renders the diagnosis uncertain. Cases with fever may run a prolonged course. Death rarely occurs. Cases of fatal hemorrhage (Toelg and Neusser), and in the aged, of exhaustion (Leichtenstern), and of sudden death with symptoms of sudden heart-failure, are reported.

Treatment.—(*a*) *Causative* factors, as gastritis or cardiac failure, should be appropriately treated; dyspeptic symptoms yield most readily to gastric lavage; a diet of skimmed milk, which contains least fat, and fractional doses of calomel at night followed by a mild saline in the morning, should be given; but it should be remembered that active catharsis augments or indeed initiates catarrh.

℞

Acidi hydrochlorici diluti	℥j.
Tincturæ nucis vomicæ	ʒiv.
Tincturæ gentianæ co.	q.s. ad ℥iv.

M. et S.—One teaspoonful after meals in half a glass of water.

Calomel was formerly given because it produced green "bilious stools"; this color is not due to bile but to mercuric sulphide. (*b*) In the *diet*, fats are particularly to be avoided, as neither the stomach nor intestine can digest them; tea, coffee, alcohol and coarse foods should be interdicted; as above mentioned, skimmed milk is given and well-cooked carbohydrates are gradually added to the diet; toast, eggs, mealy soups and possibly some lean meat may be given, though meat is often poorly tolerated, seemingly because of exclusion of the pancreatic secretion. (*c*) As long as acute dyspepsia exists, *rest in bed* is indicated, in order to maintain nutrition on the lowest possible diet. (*d*) The bowels should be regulated by *copious injections* of water which are thought to incite peristalsis and therefore dislodge the obstructing mucus plug. (*e*) The *itching* is allayed by 1 per cent. carbolic solution, the use of which requires care, as the urine sometimes becomes cloudy; acetanilide, gr. v q.i.d.; potassium bromide, ʒss, well diluted, by rectum once or twice daily; an occasional hypodermic of pilocarpin hydrochlorate gr. ⅛, warm baths to relieve the ryness of the skin and calcium chloride gr. x to xx t.i.d. for a few ays are indicated.

Chronic catarrhal cholangitis rarely follows the acute form but more ften results from occlusion of the common duct by calculus, cancer, tricture or pressure from without (*v. gall-stones, pancreatic cancer and icterus*).

Congenital Occlusion of the Bile Ducts.—Sixty-four cases are recorded, according to Rolleston, who thinks that some poison possibly initiates cholangitis, which results in a mixed cirrhosis and obliteration of the ducts. Icterus develops within a month after birth and death results in a few weeks or months—nearly always within a year—from cholæmia or hemorrhages.

(E). TUMORS OF THE GALL-BLADDER AND BILE VESSELS.

Cancer of the Gall-Bladder.

Tumors other than cancer are rare and of purely anatomical interest as papilloma, fibroma, lipoma, or adenoma. Rolleston states there are but 9 recorded cases of sarcoma. Fütterer (1901) collected 268 cases of cancer, though it is certainly much more frequent than these figures indicate.

Etiology.—The most striking etiological factor is *gall-stones*, present in 70 per cent. (Musser, Fütterer) to 85 per cent. (Zenker, Courvoisier); it is estimated that 4 to 14 per cent. of cases of gall-stones result in cancer of the gall-bladder, but this seems a very high percentage considering the great number of cases of gall-stones and the small number of cases of cancer of the gall-bladder. Eighty per cent. occurs in women, caused by lacing (Fütterer); the average age of subjects is fifty-eight years.

Pathology.—The tumor may be scirrhus or medullary, but histologically is usually of the columnar type (adenocarcinoma); the spheroidal and squamous types are rare.

Symptoms.—(*a*) *Gall-stones* which irritate the mucosa and cause most cases, usually produce no symptoms though previous colic may be noted in the history. (*b*) A *tumor of the gall-bladder* is palpable in over 66 per cent., first as a smooth oval swelling, and later as a larger and more nodular one; it is usually due to the growth, for the gall-bladder itself is generally shrunken. The fundus is the usual point of origin, being most irritated by calculi; much more rarely the neck of the gall-bladder, or least frequently, the cystic duct, is the point of origin. The tumor appears below the edge of the liver with which it moves and with which it is consecutive on palpation, percussion, and on inflation of the colon. There may be local discomfort and, in rare cases, severe pain like that of gall-stones. (*c*) *Cachexia* appears with all its attendant symptoms.

Complications.—*Secondary growths* in the liver occur in 58 per cent. by lymphogenous routes or by invasion from contiguity and sometimes cause hepatic enlargement; from the liver, extension by the hepatic veins may occasion systemic metastases. *Icterus* occurs in 69 per cent. (Musser) from malignant or inflammatory involvement of the bile ducts; cholæmia is a common termination. Using Musser's figures, growths in the peritoneum occur in 12 per cent., in the lungs or pleura in 10 per cent., in the abdominal lymph glands in 16 per cent. and ulcerative fistula into the colon in 10 per cent. Warthin records adrenal metastases with pigmentation like that of Addison's disease and with vitiligo. Pressure on the pylorus may cause its stenosis, vomiting, or dyspepsia. *Ascites* occurs in 25 per cent., and is due to mechanical compression of the portal vein or to malignant peritonitis. Suppurative cholecystitis and cholangitis may develop, and the case then terminates by sepsis, ulcerative endocarditis, etc. The differential *diagnosis* is considered under gall-stones. The clinical course averages six months. *Treatment* is merely palliative, unless the affection is diagnosticated early enough for operation or is accidentally found in operating for other conditions. Removal of the gall-bladder cancer has resulted in a few permanent cures (Hochenegg, Robson, Warren). Part of the liver also must usually be removed.

Tumors of the Extra-Hepatic Bile Ducts.

Rolleston states that about 80 cases are reported. These tumors are usually small, show little tendency to infiltrate and are cancers of the columnar type. The common duct is most often affected, the hepatic duct much less frequently, and the cystic duct least frequently; these tumors usually occur between the years of sixty and seventy and two-thirds occur in males (Devic and Gallaverdin). The salient symptom is *icterus*, which develops early, increases steadily, is intense, occurs in nearly all cases, causes icteric necrosis of the liver cells in the central zone (Fütterer) and precipitates early cholæmia before metastases have time to develop. The gall-bladder is almost always dilated anatomically in tumors of the common duct and is felt clinically in over 50 per cent. Pain is not common, though that of gall-stones may be simulated. Ascites, dyspepsia and gastro-intestinal hemorrhages (from cholæmia) may develop. Death results most often from cholæmia, and less often from cholangitic sepsis. Differentiation from duodenal or pancreatic tumors (*q.v.*) is always difficult, indeed usually impossible. Confusion with gall-stones (*q.v.*) is possible. *Palliative treatment* consists of draining the gall-bladder to relieve the icterus; radical treatment is extirpation of the deeply located tumor (Kehr, Halsted).

DISEASES OF THE PANCREAS.

(A). ACUTE PANCREATITIS; FAT NECROSIS.

Etiology.—*Infection* ascending from the intestines is the leading ause. (*a*) *Calculus obstructing the common duct,* though allowing bile o regurgitate into Wirsung's duct, may cause acute pancreatitis (Opie), specially when the gall-bladder, the natural reservoir for obstructed ile, is shrunken. Japha found gall-stones in 38 per cent. of 76 cases of cute fat necrosis of the pancreas. According to Quénu and Duval 1906) 118 cases are recorded in which pancreatitis occurred in the ourse of cholelithiasis. Flexner concludes that gastric juice, and Opie hat bile alone may initiate inflammation and fat necrosis, even when here is no infection. Though the accessory duct of Santorini may arry off the pancreatic juice when there is obstruction of Wirsung's luct, it is too small for much compensation in 66 per cent. of cases (Opie); (*b*) *metastatic infection* is an infrequent cause; in one case the uthor observed acute pancreatitis in which acutely inflamed ulcers of the leg may have been the atrium of infection; it rarely follows mumps; (*c*) in some cases suppuration or *ulceration in contiguous tissues* is causative.

Predisposing Factors.—(*a*) *Age and sex;* in Koerte's collection of 41 cases, 90 per cent. occurred in *males beyond middle life;* McPhedran observed a case in a child nine months old. (*b*) *Obesity;* (*c*) *alcoholism;* (*d*) trauma; and (*e*) gastro-intestinal catarrh are factors. (*f*) Parturition appeared to be a factor in 6.6 per cent. of Peiser's collection of 128 cases.

Symptoms.—The symptoms were clearly described and the clinical picture defined by Fitz (1889), though cases were reported earlier (Oppolzer). The *onset is sudden* in adult stout males who have enjoyed previous good health, or who have a history of alcoholism or of gall-stones.

1. *Epigastric pain* is the *initial symptom;* it is very sudden and severe, is often agonizing, may be constant or paroxysmal, and may be confused (or coincident) with gall-stone colic, though it is even more intense; it may be central, diffusely epigastric, to the right or left of the median line, or rarely directly over the head of the pancreas. The author has seen cases in which it radiated into both lower axillæ. The intensity of the pain is referable to pressure on the cœliac plexus (neuralgia or possibly neuritis cœliaca). Much less often the pain is gradual in onset.

2. *Epigastric tenderness* is usual, often over the head (Elliot) or sometimes more to the left and over the tail of the pancreas, whence the inflammation may extend to the left pleura with tenderness and friction, as in one of the author's reported cases. The recti are tense and the epigastrium is swollen.

3. *Vomiting* is early and severe, and brings up food, mucus and sometimes blood.

4. *Collapse* soon follows; it is often very severe, threatens immediate dissolution and too often prevents surgical interference. It is due to pressure, on or actual inflammation of, the closely contiguous cœliac plexus and semilunar ganglia. There may be marked *cyanosis*. In one of the author's cases there was relapsing shock with cyanosis and a pulse of 140, which was so weak that no surgeon would operate during the two weeks before recovery. Collapse usually causes death in 2 to 4 days in unoperated cases.

5. *Fever* is not constant; there may be no elevation or an irregular rise to 103 or 104 degrees. Chills are more common in late suppuration than at the onset.

6. The *pancreas can rarely be felt* because of its deep position behind the colon and stomach, and because the epigastrium is tense, swollen, and tympanitic. If palpable it is small and immobile.

7. The *bowels* are constipated, simulating obstruction. Operation or autopsy at this stage shows an acute pancreatitis, with swelling, a variegated yellow-red or black color, exudation of fibrin, pus cells, blood (hemorrhagic pancreatitis) and areas of acute *fat necrosis* (Balser, 1882). Fat necrosis is caused by the fat-splitting steapsin (Langerhans, Hildebrand, Dettmer and Flexner), which produces glycerine and insoluble fatty acids from the pancreas and contiguous adipose tissues, *i.e.*, omentum, mesentery, and peritoneum; the glycerine is absorbed, the fatty acids are precipitated with lime and become opaque white areas of necrosis as large as a pinhead or saucer, and some of the ferments occasionally escape into the blood, producing toxæmic symptoms or necrosing fatty tissue elsewhere, as in the pericardium or skin.

8. If the patient lives, the *symptoms of localized peritonitis* develop in the upper half of the abdomen and sometimes more to the left side; a pus sac may fill the lesser peritoneal cavity; the localized pain may become general from diffused fat necrosis. In one of the author's cases great dyspnœa attended diaphragmatic pleurisy.

9. *Septicopyæmic symptoms* may appear, as rigor, hectic fever, delirium, pleurisy, hiccough, icterus, diarrhœa, and loss of weight and strength; in one case the author saw acute splenic tumor, acute nephritis and universal swelling of the lymph nodes.

9. Acute *glycosuria* is not common, though present in one of the author's cases.

In the third week *rupture* into the colon may occur; this is attended by lancinating pain, intestinal hemorrhage, even passage of the pancreas by the bowel (Chiari) and relief from all symptoms.

Suppurative types, of which Koerte collected 46 cases, may begin gradually, without acute gangrene, fat necrosis, or hemorrhage. The symptoms are less acute and severe than those of the acute type. The suppuration is localized or there is a diffuse purulent infiltration; it may cause pylephlebitis, liver abscess, subphrenic abscess, burrowing of pus into the peritoneum, loin (which Koerte considers rather characteristic), peripheral tissues, or alimentary tract. Protracted suppuration sometimes is said to cause glycosuria, skin pigmentation, and anasarca. Koerte found 40 cases of *acute gangrenous pancreatitis.*

Diagnosis.—The cardinal features are (*a*) the sudden onset, (*b*) violent epigastric pain and tenderness, (*c*) vomiting, (*d*) collapse, (*e*) epigastric distention. The absence of indican under circumstances in which it usually is increased is suggestive of pancreatic lesions, because trypsin converts albumin into peptone, which in turn becomes partly converted into indol, the source of indican in the urine. Since leucin and tyrosin are formed in the normal intestine as a result of the action of the pancreatic juice upon proteids, Ulrich attaches much importance to their absence from the urine (with glycosuria), as indicative of disease of the pancreas. Robson finds calcium oxylate crystals in 30 per cent. of all varieties of pancreatic disease.

The *pancreatic reaction* of Robson and Cammidge is a test for glycerine in the urine; it is complicated and recent reports show it is neither constant nor reliable.

Differentiation.—(*a*) *Acute poisoning* is eliminated by the history and by the gastric contents. (*b*) *Intestinal obstruction* is less severe in onset; collapse is usually attended by distention and often by intestinal rigidity or peristalsis above the obstruction, and rectal injections often show its impermeability; indicanuria is usual; epigastric distention alone is unusual; in one pancreatic case, the author saw obstruction lasting nine days. Operation, indicated in either instance, may be necessary for diagnosis. (*c*) *Gall-stone colic* is sometimes hard to differentiate but is rarely as severe as pancreatic pain; collapse or epigastric distention is infrequent. (*d*) In *perforating* duodenal or gastric *ulcer*, the later collapse, pain, and the history, are significant. (*e*) Mesenteric embolism and appendicitis (without induration) must be considered.

Prognosis.—Mild cases may occur, though the malady generally evinces a severe progressive tendency. A fatal attack may be the last of several milder seizures. Fitz has found at autopsy fibrous tissue and crystals or granules of hæmatoidin which he regards as evidence of previous hemorrhage and inflammation. Death may occur within a few hours to three days from collapse, in two or three months from sepsis, or in six months from diabetes (Fitz). Death at an even later period may result from sepsis or diabetes. Koerte and Osler report cases of recovery after cœliotomy had first revealed the true cause of the obstruction. The pancreas may slough into the intestine, as in Trafoyer's and Chiari's cases, and recovery may result.

Treatment.—Rectal feeding and stimulation, morphine hypodermatically, and heat are indicated during the initial shock, from which some surgeons consider that a rally is necessary before operation can be safely performed. Though early operation is dangerous because of shock and hemorrhage, the author considers, with Robson, that it is indicated lest local extension or general toxæmia develop. In 21 cases (Quénu and Duval), 62 per cent. of the acute cases died after operation, 100 per cent. of the hemorrhagic, and 45 per cent. of the acute suppurative cases.

(B). CHRONIC PANCREATITIS.

Etiology and Pathology.—(*a*) *Gall-stones* in the common duct may induce chronic catarrh of Wirsung's duct, thus leading to chronic pancreatitis (Robson). (*b*) *Cancer*, gastroduodenal *catarrh* and *ulceration* (alcoholism, syphilis, and pancreatic calculi) may produce the same results. In these instances *ascending infection* appears to be the patent factor, causing *interlobular fibrosis* (Opie) *i.e.*, fibrosis around the pancreatic lobules; diabetes is a late and uncommon sequence. (*c*) *In liver cirrhosis* and *hæmochromatosis* (see DIAGNOSIS PORTAL AND BILIARY CIRRHOSIS) *an interacinar fibrosis* (Opie) occurs, developing in the lobules, diffusely invading the islands of Langerhans and causing glycosuria; it does not result from obstruction of the common duct. The opposite of this condition was described by J. C. Ohlmacher in a case of degeneration of the liver; there was a compensatory hypertrophy of Langerhans' islands, proving for the first time the mutual relation of the pancreas and liver, in diabetes, and in the disposition of carbohydrates.

Symptoms.—Symptoms may be lacking. Moynihan states that chronic inflammation increases in frequency with increasing thoroughness of operations for gall-stones. They vary with the cause. In catarrh due to gall-stones there is a history of *colic, jaundice*, and sometimes *intermittent fever*. *Tenderness* in the epigastrium, a fulness above the umbilicus, and loss of flesh are noted, and if the pancreatic symptoms predominate, the pain passes from the epigastrium to the left side, to the scapular or renal regions. If gall-stones are not the cause, there may be merely aching or painful attacks which are not at all pronounced; pain may be absent. The gall-bladder may enlarge from obstruction of the common duct, when the liver becomes enlarged and frequently cirrhotic (Robson). According to Robson marked *enlargement of the spleen* is frequent, bile is found in the urine in 60 per cent., *calcium oxalate crystals* in 40 per cent. and glycosuria in but 5.5 per cent. (90 cases). Opie found glycosuria but once in 22 cases. Riedel, Koerte, Robson, Moynihan, and others have described a *tumor-like induration* of the head of the pancreas, which simulates cancer because of the emaciation and icterus which it produces; differentiation may be impossible, even during laparotomy; it is more common in subacute than in chronic cases. As in tumor, excessively copious, pale stools, laden with fat and undigested muscle-fibers, are noted. Polyuria has been reported.

Differentiation from stone in the common duct and from cancer is very difficult.

Prognosis.—Without surgical intervention, death is likely to occur from emaciation, cholæmia, diabetes, or hemorrhagic diathesis.

Treatment.—In severe clear cases and doubtful cases drainage of the gall-bladder or anastomosis is indicated. Robson operated on 62 cases with 13 per cent. mortality, Quénu and Duval on 62 cases with 13 per cent. mortality, and Moynihan on 14 cases without a death. Hemorrhage is a dreaded complication; Robson and Moynihan recommended calcium chloride gr. xx to ʒj t.i.d. before operation to increase the coagulability of the blood.

(C). PANCREATIC APOPLEXY.

Pancreatic hemorrhage has been described by Spiess (1866), Klebs (1870), and Zenker (1876). Draper found 19 instances in 4,000 autopsies and Anders (1899) collected 40 cases. The anæmias, syphilis, alcoholism, obesity, acute infections and local lesions of the pancreas, seem to be *predisposing causes*. Sixty-six per cent. of cases occur *in men*. Doubtless many reported cases are acute hemorrhagic pancreatitis. *Sudden onset*, severe *collapse*, *epigastric pain*, intestinal obstruction, vomiting, restlessness, and normal or subnormal temperature, mark the affection. The course is said to be even more acutely fatal than that of acute pancreatitis; death sometimes occurs within a few hours. If the patient survives the initial stage, suppuration may develop in the pancreas, peritoneum, and retroperitoneal tissues into which the hemorrhage sometimes burrows. Hemorrhagic cysts may form, which should not be confused with cysts into which secondary hemorrhage has occurred. The diagnosis from acute pancreatitis is rarely made, and the treatment of both affections is identical.

(D). LITHIASIS.

Few more than 100 cases of sialolithiasis pancreatica are recorded. The *ætiology* is probably like that of gall-stones, infection of Wirsung's duct (wirsungitis); stagnation of secretion is a predisposing factor, whence the occasional association with gall-stones, pancreatic inflammation and tumor.

Symptoms and Diagnosis.—Confusion with gall-stones is often inevitable, for colic without icterus is equally indicative of either condition and their co-existence is not uncommon. The pain may radiate to the left side and shoulder; Lancereau, Lichtheim, Leichtenstern, Minnich, Holtzmann, Moynihan, and Kinnicutt have recognized cases. In some of them the stones were found in the fæces during life, in others at autopsy. Salivation is reported. In Lichtheim's case there was colic, glycosuria (found in 33 per cent. of the cases) and diarrhœa. The stones are single or multiple (even 300), white-gray, round, oval, irregularly outlined or branching, hard or friable, and vary in size from that of almost impalpable gravel to that of a hazelnut. They consist largely of calcium carbonate and phosphate.

Shattuck described an oxalate calculus. Their sequelæ are (*a*) obstruction and inflammation of Wirsung's duct, (*b*) acute suppurative pancreatitis and possibly pancreatic cancer. The treatment, in the cases which are fortunately diagnosticated, is surgical; Moynihan has both recognized and successfully operated on a case. Gould also operated on a case which did not recover.

(E). PANCREATIC CYSTS.

To Nicholas Senn (1885) belongs the credit of establishing the clinical picture. Bessel-Hagen (1900) collected 149 cases.

Etiology and Pathology.—Probably many reported cases are extra-pancreatic, especially (*a*) the *traumatic* cysts of which Honigmann in 1905 collected 69 cases. (*b*) Duct obstruction (*ranula pancreatica*, Virchow) by calculi, strictures, parasites, tumors, or inflammation in the smaller ducts, is an etiological factor. (*c*) *Cystadenoma* is the cause of most cysts (Lazarus). (*d*) Cysts rarely result from an *old hemorrhage*. In all forms the cyst causes considerable parenchymatous atrophy. (*e*) *Sex*. Traumatic and inflammatory forms are most frequent in men and the cystadenomatous type in women. (*f*) *Age;* over 50 per cent. are found in persons in the third decade of life, though a congenital form, sometimes with cystic liver and kidneys, is found (Richardson).

Symptoms and Diagnosis.—1. In *cysts*, (*a*) a *deep retroperitoneal* swelling is found, (*b*) the location of which is more or less *central*, but is *above the navel* in 87 per cent., where other cysts are most uncommon; it lies behind or between the stomach and colon; it sometimes lies below the colon but rarely above the stomach; very large cysts (which contain 10 to 20 quarts) may fill almost the entire abdomen. A cyst may in rare cases develop in the tail of the pancreas, when it points toward the spleen or left kidney. (*c*) Its form is *round*, *oval*, and *smooth;* (*d*) its consistence often *varies*; (*e*) it is *immobile* on palpation and on inspiration, or at the most can be moved slightly in a vertical direction. (*f*) The *fluid* is alkaline, 1,010-25 specific gravity, usually mucoid, blood stained in 82 per cent. (Koerte), and in 45 per cent. contains one or more pancreatic ferments (diastatic, saponifying, or tryptic) of which the latter is alone characteristic, for the other ferments occur in various exudates. The fluid sometimes contains fat and cholestearin; aspiration is dangerous, because the tension of the cyst is great and fat necrosis or peritonitis may result from leaking after exploratory puncture.

2. The so-called *pancreatic symptoms* are rare, as emaciation, salivation, stools laden with undigested fat or muscle-fibers or glycosuria.

3. Symptoms of *peritonitis* or of *intestinal obstruction* may occur in traumatic or inflammatory types and after rupture. The author saw a case of Murphy and Wittwer in which the picture after rupture

s exactly that of subacute tuberculous peritonitis; after two rup- es the case was successfully operated on.

1. *Pain* like that of cholelithiasis may be present, but it more often liates to the left side, shoulder and neck. Pressure symptoms in- de dyspnœa, vomiting, icterus, emaciation, and polyuria.

Differentiation from ovarian, renal and other cysts is largely a ographical question. Ovarian cysts have their pelvic origin and nnection, but hydronephrosis is movable and originates in the flank which pancreatic cysts seldom penetrate. Fluid in the lesser peri- eal sac or mesenteric, omental, and retroperitoneal cysts are difficult distinguish. Cysts of the liver present respiratory excursion and other viously hepatic symptoms.

Treatment.—Aspiration is never permissible. Enucleation of the c is more difficult and more dangerous than simple drainage. In ssel-Hagen's 149 cases, the operative mortality was 6.7 per cent. iabetes has followed operation (Bull and Kroenig).

(F). TUMORS OF THE PANCREAS.

Cancer of the pancreas constitutes 67 per cent. of all pancreatic eases but possibly this estimate must be discounted because con- sions with benign induration have been frequent until of late years. occurs in 0.6 per cent. (Eppinger) to 1 per cent. (Soyka) of autopsies. 132 cases of pancreatic tumors (Segré) 127 were cancer, 2 sarcoma, ysts and 1 gumma. Sarcoma is usually secondary from contiguous mphosarcoma in the retroperitoneal glands.

Cancer is usually scirrhus, less often of the medullary, colloid, or enomatous type. It most often begins in the head of the organ. In me cases it may be secondary to pyloric cancer.

Symptoms.—(1) *Tumor* is the commonest and surest symptom, curring in 25 to 50 per cent.; it lies *deep*, beside the spine at the level f the navel and between the pylorus and colon. It is tender, prac- cally *immobile*, and varies from the size of a walnut to that of a child's ad though small tumors are commonest; in some instances it may ow propagated pulsation from the aorta or a bruit from its com- ession. Palpation should be made with the stomach and bowels npty. (2) *Icterus* occurs in 72 per cent., increases gradually, and more often *continuous* than remittent. In some cases there may be gors and pain as in biliary calculi; occasionally, just before death, e growing anæmia seems to lessen the jaundice. There may be the ghtly enlarged "icterus liver," but Bard and Pic state the liver is ually rather small. (3) *The gall-bladder* is usually *enlarged* (Bat- rsby, Bard and Pic); see *differentiation*. Cochez holds that it rinks when the hepatic or cystic ducts are obstructed. (4) *Pain* nd *early vomiting* are usual. The pain is epigastric, severe, and metimes neuralgic (neuritis cœliaca) or radiating as in pancreatitis r lithiasis. (5) Cachexia is marked and rapid; great prostration, emaciation, and a syncopal tendency occur in tumor as in other pancreatic lesions. The temperature is usually subnormal. (6) The *stools* may contain much fat (Kuntzmann, 1827 and Bright, 183[illegible] in *steatorrhœa* the fatty acids and neutral fats (Mueller) are more abundant than the fatty soaps; its significance grows if diarrhœa and icterus are not present. The stools also contain many undigested muscle-fibers (*lientery*) and sometimes blood is present. (Sahl[illegible] test for albumin digestion is made by giving salol in divided doses [illegible] glutoid capsules; as the glutoid is not dissolved, no carbolic acid appears in the urine). (7) The *urine* may contain sugar; Miral[illegible] holds that *glycosuria* occurs in 26 per cent., though in Robson's [illegible] cases it was absent. It is said to be an early symptom and to disappear before death or with the increasing icterus. *Albuminuria* is more frequent than glycosuria. The *indican is decreased.* Peptone, maltose (once, by v. Ackeren), and fat (Clark and Bowditch), have been found. Polyuria and bronzing of the skin are rare findings. *Complications* include pyloric or duodenal obstruction; compression of the aorta or cava; retention cysts of the pancreas; and extension by contiguity to the portal vein, colon, peritoneum, ureter or lungs.

Differentiation.—1. Chronic *pancreatitis* with icterus and tumor may not be differentiated even by laparotomy (Riedel) though ascites indicates neoplasm, and recovery means inflammation. The pancreatic cachexia of French writers, occurring in many lesions of the pancreas, may be confused with the cachexia of cancer.

2. In *duodenal cancer* (*q. v.*) the growth is also fixed and is attended by icterus, but cancer above the papilla causes rather the picture of pyloric stenosis; in cancer below the papilla, pancreatic juice may be recovered from the stomach.

3. *Liver disease,* with the frequent icterus, ascites, and splenic enlargement, causes little difficulty in differentiation.

4. Calculus in the Common Duct.—*vs.*—	Compression of Common Duct by Pancreatic Tumor.
1. History of colic usual.	Absent.
2. Tenderness of gall-bladder usual.	Absent.
3. Icterus, often remittent.	Permanent.
4. Liver less large than in—	pancreatic tumor.
5. Intermittent fever, and rigors.	Subnormal temperature.
6. Gall-bladder (see gall-stones) shrunken in 80 per cent.	Dilated in 92 per cent. (Courvoisier's law).
7. Slower course.	Very rapid emaciation.

According to Bard and Pic, the icterus, enlarged gall-bladder, rapid emaciation, cachexia, subnormal temperature and normal or smaller liver of pancreatic cancer can only be confused with some few cases of gall-stones, cancer of immediately adjacent parts, primary cancer of the liver or bile-passages, and primary duodenal or gastric cancer with early diffuse generalization in the liver. Kehr states that obstruction of the common duct is cancerous in 70 per cent., (chronic) pancreatitic in 20 per cent. and calculous in 10 per cent.

Treatment is largely symptomatic; morphine should be given for pain, the gall-bladder should be drained (cholecystotomy) or it should be sutured to the intestine (cholecystduodenostomy). Koerte reports ten operations with six permanent recoveries, and Ruggi reports a successful operation.

DISEASES OF THE PERITONEUM.

(A). ACUTE DIFFUSE PERITONITIS.

Acute inflammation of the peritoneum was at one time far more important as an independent affection than at present; we now regard it as chiefly symptomatic of some other disease, *e.g.*, appendicitis, salpingitis, puerperal sepsis, etc.

Etiology.—The *actual cause* is probably always *mycotic.* Some writers classify peritonitis as bacterial (especially suppurative forms), chemical (serous and sero-hemorrhagic forms) and mechanical (adhesive forms). The Streptococcus pyogenes (puerperal and traumatic forms), and Bacillus coli (forms due to intestinal lesions) are the two most frequent organisms. The staphylococcus, pneumococcus, and tubercle bacillus are fairly frequent and less often the Bacillus pyocyaneus, proteus, anthracis, typhosus, aërogenes encapsulatus, the ray fungus, Bact. lactis, gonococcus, Amœba coli, and pseudodiphtheria organisms. Generally speaking, mono-infections are less common than *mixed infections.*

Factors causing *lessened physiological resistance* include nephritis (in 10 per cent. of cases), alcoholism, cardiac disease, scurvy or gout; in other words peritonitis may be a terminal infection, fatal to reduced subjects.

Atrium.—Compared with pleurisy and pericarditis, peritonitis is seldom primary. Though it may follow inflammation of every abdominal viscus, it is most commonly a secondary manifestation of intestinal disease (*e.g.*, appendicitis), or of pelvic diseases in women. (*a*) Infection may occur through the *alimentary tract;* appendicitis, bowel ulceration (typhoid, carcinomatous, tuberculous), bowel obstruction, perforation, traumatism and infarction may cause it. Gastric are less frequent than intestinal lesions and ulcer and cancer range first. (*b*) The internal *female genitalia;* it may result from gonorrhœa (Noegerrath), extra-uterine pregnancy, puerperal infections and septic abortion; peritoneal infection from diseases of the male genitalia is rare. (*c*)

Liver and bile vessels; local or general peritonitis may result; peritonitis is more common as a result of hepatic abscess and syphilis than of echinococcus, cancer, pylephlebitis, or cholecystitis. (*d*) *Pancreas;* acute pancreatitis. (*e*) *Kidney* and bladder; pyonephrosis, calculi or ulcerations of the bladder. (*f*) Rarer causes are splenic affections, retroperitoneal adenopathies, spinal or costal caries, lesions of the thoracic duct, or the abdominal parietes, or extension through the diaphragm. (*g*) *Metastatic peritonitis* is not common, though it is observed in septicopyæmia and in acute infections. (*h*) In *children* fœtal peritonitis has been observed by transplacental infection; in the new-born the umbilical vein may convey infection, though peritonitis occurs in but 8 per cent. of fatal cord infections. It occurs more commonly through the lymphvessels of the cord; congenital syphilis may in this way cause peritonitis.

Age and Sex.—Most cases occur between the ages of fifteen and forty and women are more often affected than men.

Symptoms.—Symptoms are local and general.

1. Local or Abdominal.—(*a*) *Pain,* the usual initial symptom, is almost invariable, is continuous, begins near the navel and remains greatest there; a causal gastric ulcer may produce pain in the back, salpingitis is attended with early pelvic pain, and appendicitis has its own initial localization, but the pain of diffuse peritonitis is felt at or below the navel. (*b*) *Tenderness* accompanies pain; the tenderness is deep rather than superficial; tenderness and pain are very rarely absent and are caused by inflammation; at the operation or autopsy both the visceral and parietal investments are injected, reddened, lustreless, covered with a thin film of fibrinous exudate which binds the intestinal coils together; not infrequently the peritoneum is dotted with minute petechiæ. (*c*) The *attitude* is characteristic; the knees are drawn up and the head lifted to relieve tension and, in marked cases, to keep the bedclothes from contact with the abdomen. (*d*) *Vomiting* is reflex; it occurs early and greatly aggravates the pain; the vomitus contains food and later bile; it may be green (the *vomitus herbaceus* of the older writers), rarely dark brown or fæcal. It is accompanied by redness and dryness of the tongue, great thirst and singultus which is a sensory phrenic symptom; toward the fatal issue vomiting and singultus cease. (*e*) The *abdomen* on inspection and palpation is at first *tense, retracted,* and *scaphoid,* but *later* becomes *tympanitic* from paresis of the inflamed intestinal coils (Stokes) and decomposition of their contents; the diaphragm stands high and its movement is restricted; in some, especially muscular, subjects, the abdomen may be retracted throughout the course. Exploration by percussion may elicit *dulness,* which is usually due to serous, seropurulent, or perhaps serohemorrhagic exudation, which may be effused in small pockets between fresh adhesions or may fill the entire cavity, amounting to even 30 or 40 quarts and sometimes fluctuating to the hand; *as a rule a tympanitic note* prevails; in some cases dulness suggests the presence of fluid which, however, is not found on exploration; because adhesions, the fluid changes but little on change of posture. On sculation a friction rub is sometimes heard (Beatty and Bright), iefly over the liver. (*f*) *Constipation* is usual and results from in- mmatory infiltration of the intestine (Stokes); in the puerperal treptococcic) and in pneumococcic peritonitides, diarrhœa is com- on.

2. **General Symptoms.**—(*a*) *Shock* is a conspicuous symptom. llapse may occur early and directly from perforation or later from xæmia. The *pulse* is *frequent, hard, wiry,* and averages about 120 more; later it becomes thready and runs to 140 or 170. The *facies ippocratica* is pronounced, with its anxious expression, wrinkled, ummy, cyanotic, lead-colored skin, pointed nose, hollowed temples, d sunken eyes. The voice whispers (*vox cholerica*) to avoid move- nt of the diaphragm and the *breathing* is costal, *shallow*, difficult, d rapid. (*b*) *Fever* is usually but not always present. It may rise ruptly with a chill at the onset but follows no one type; it may be ntinuous or remittent, and there is usually elevation of temperature rectum, even during collapse. The fever and pulse curves may n parallel in peritonitis with a slow course. (*c*) The *lungs and art are crowded* upward by the abdominal distention. The apex- at is high and further to the left as the heart lies more horizontally an in normal conditions. (*d*) The *urine* is scant from lessened ab- rption and from cardiac weakness; albuminuria is frequent; *indi- nuria* is constant from increased putrefaction in the paretic bowel; suria is usual; tenesmus, due to involvement of the vesical serosa, less common than retention.

Course.—Though recovery is possible, the issue is fatal within two ys in the *rapidly fatal toxæmic form* in which there is only roughening the peritoneum without exudation; the slower cases rarely last more an five days. Consciousness is usually preserved to the end, though upor or delirium occasionally intervenes; the pulse becomes thready, e breathing shallow and sighing, the superficial temperature is low ough rectal registration is high, and the patient succumbs to gradual sudden cardiac failure from toxæmia. In exceptional cases death curs within a few hours, or relative recovery results with encapsula- n of pus. Treves found inhalation pneumonia in 17 per cent. of s cases and Tilger found serous pleurisy in 25 per cent.; of the tter, 63.3 per cent. was right-sided, 3.3 per cent. left-sided, and 33.3 r cent. bilateral.

Types.—(*a*) *Peritonitis serosa* usually follows the fibrinous form; e fluid is yellow or light green, its specific gravity is 1,015 or more, contains 2 to 3 per cent. of albumin and flocculi of fibrin, epithelial lls, red cells, and leukocytes. (*b*) *P. purulenta* may occur in pockets as a massive pyoperitoneum, amounting to 30 or 40 quarts. (*c*) *putrida* occurs in septicopyæmia and puerperal fever, but espe- lly in perforating carcinoma; Obermeyer and Schnitzler have oven that fœtid peritonitis may occur without perforation. (*d*) *perforativa* is characterized by two groups of symptoms, (i) those

transverse roll above the navel, and the mesentery is indurated (m
enteritis retrahens) as described under tuberculous peritonitis. As
mesentery retracts, it may gather the intestinal coils into a ball
larger than a child's head, lying centrally or to the right of the med
line. Here and there may be found multilocular serous encapsu
tions, or there may be no serous exudation, at least when the c
comes under observation. There may be palpable tumor-like thi
enings. The spleen, liver, stomach, cæcum, or colon may be part
ularly indurated. In some cases it is but part of a multiple cirrho
(polyserositis, polyorrhomenitis), invading also the pleura and pe
cardium. Miliary fibromata, resembling miliary carcinosis or milia
tuberculosis, may sometimes form around parasitic ova or cholestea
crystals.

(C). LOCALIZED PERITONITIS.

I. Suppurative Forms.

1. Subphrenic Abscess and Pyopneumothorax Subphrenicus.—Subphrenic suppuration was first diagnosticated by Barlow (1845), and was especially described by Leyden (1879). Maydl's monograph (1894) comprised 179 cases, upon which the following figures are based; sixty cases are described by Koerte (1903).

Etiology.—(*a*) *Gastric ulcer* causes 19.5 per cent.; gastric cancer is less frequent. (*b*) *Appendicitis* causes 14 per cent.; (*c*) *trauma,* 14 per cent.; (*d*) *intestinal ulceration,* 7.3 per cent.; (*e*) cholangitis and cholecystitis, 11 per cent.; echinococcus, 7.3 per cent.; liver abscess; (*f*) *metastatic* abscess, 6 per cent.; (*g*) *perinephric* abscess, 6 per cent.; (*h*) occasionally, ulcerative lung lesions, 5 per cent.; (*i*) splenic infarct, abscess or echinococcus, diseases of the spine, ribs, pericardium or uterus are uncommon.

Symptoms.—Pus is always present, is ensacculated beneath th
diaphragm (pyothorax subphrenicus) and is associated in 26 per cen
with gas formation (pyopneumothorax subphrenicus). The *subphren*
abscess is usually right-sided, is limited by the suspensory ligament
the liver and may be intra- or extraperitoneal; in the latter insta
the abscess is most often appendicular or perinephric and lies
back in the abdomen. Cases with simple pus formation are
difficult to localize; their symptoms are those of sepsis with
tenderness, or dulness on the liver convexity, which suggest
abscess. The retroperitoneal forms give most dulness poste
but in intraperitoneal forms it is chiefly located anteriorly; s
and œdema of the chest wall are unimportant but may occur
suppuration; the x-rays may produce a subphrenic shadow.

In *pyopneumothorax subphrenicus* there are signs "of a ca
neath the diaphragm, filled with gas and pus, pushing the di
upward and simulating pyopneumothorax" (Leyden). (*a*)
and *absence of vesicular murmur* and of vocal *fremitus* are fo

e third rib downward, above which is the compressed lung, and low which is (*b*) the *dulness of the liver* whose exposed surface and lge, dislocated downward, are easily percussed and palpated. Somenes the tympany entirely covers the liver dulness. As in pneumoorax (*c*) *succussion* and, on change of posture, the *clearly shifting e of dulness* may be elicited. (*d*) *Puncture* evacuates gas and putrid s; sometimes on a higher puncture serum is found in the pleura r pleurisy develops in 66 per cent. of the cases. The needle ascends ring inspiration and descends during expiration, which is the conrse of the findings in pleural exudation (Fuerbringer). If a manomr is attached to the exploring needle, a rise in the indicating umn during inspiration and a fall during expiration denote a phrenic lesion (Pfuhl). (*e*) *The x-rays* may show movement of diaphragm and a shadow between it and the liver, but in three es the writer found no respiratory excursion. (*f*) *Perforation* into lungs occurs in 16 per cent. (more often in extra- than in intraritoneal forms), into the pleura in 19 per cent., and into the perirdium in 1 per cent. At the beginning, and until perforation, there generally an entire absence of such thoracic symptoms as cough sputum; this is of value in differentiation from true pyopneumoorax. (*g*) The *history* of the subphrenic pyopneumothorax is usually at of some *abdominal lesion* (*v.* Etiology).

Treatment is surgical. In Maydl's series, 74 cases were operated . Koerte's experience with 60 operations gives 66 per cent. of coveries.

2. **Suppuration in the Lesser Peritoneum.**—This may result from stric or duodenal ulceration, acute pancreatitis, etc. Closure of the ramen of Winslow confines the pus; a tumor appears in the epistric, umbilical or left hypochondriac region, bounded above by e stomach, which also in part covers it, below by the colon, and hind by the pancreas and retroperitoneal tissues. Its relations are e those of pancreatic cyst; variability in its size occurs when the mach is filled with gas and fluid. When gas and pus co-exist the aphragm and liver are pushed upward. Treatment is surgical.

3. **Other Forms.**—The *appendicular abscess* has been described. he *pelvic abscess* occurs especially from tubal, but also from uterine d ovarian inflammation; it follows abortion, puerperal fever, gonorœa, tuberculosis, and pneumococcic peritonitis in young girls. Other pes are suppuration around the gall-bladder, colon, and sigmoid, d in other rare localizations.

II. Adhesive or Indurative Forms.

Local thickening or adhesions may occur over any abdominal viscus t especially over the spleen, next over the liver, and less commonly r the intestines; it may bind together two coils or form bands der which loops may become strangulated.

Perisplenitis is most common over chronic malarial, leukæmic, and pseudoleukæmic enlargements of the spleen and less frequent over acute splenic tumors.

Perihepatitis may cover small areas of the liver like soldier's spots in the pericardium, or may engross its entire surface. General perihepatitis was described by Budd (1852), by Curschmann (188[illegible]), who called it the "icing liver" (*Zuckergussleber*), and by Fagge, Hale White, Nichols, A. O. J. Kelly and others. It is frequently associated with deforming proliferative peritonitis. Nichols describes it as a hyaline degeneration, hyaloserositis. Following Rolleston, there are apparently three groups of cases; (*a*) *multiple serositis* (polyorrhomenitis, Concato's disease); this borders closely on Pick's "pseudocirrhosis pericarditica." Kelly has assembled 27 cases in which perihepatitis was associated with adhesive pericarditis. (*b*) *Arteriosclerosis* and *interstitial nephritis* occurred in 86 per cent., in Hale White's series of 22 cases. (*c*) Other rarer factors are syphilis, tuberculosis, malignancy, and alcoholism. Its symptoms may be those of cirrhosis; it will be differentiated under ascites (*v.i.*).

III. Chronic Hemorrhagic Peritonitis.

This is a rare disease and was described first by Virchow, who likened it to pachymeningitis hemorrhagica. It occurs chiefly in the pelvis and is most often circumscribed. This pachyperitonitis is characterized by hemorrhagic inflammation; from the laminæ of the resulting fresh connective tissue, which is deposited layer by layer, repeated fresh hemorrhages arise.

(D). CARCINOMA OF THE PERITONEUM.

Primary malignant growths of the peritoneum are *endotheliomata*, and are very rare.

Cancer is most frequently secondary to cancer of the stomach, ovaries, intestines, pancreas, and liver. Histologically it is that of the primary growth. Its gross forms are (*a*) *miliary carcinosis*, resembling miliary tubercles; (*b*) *vegetative;* (*c*) *ulcerative;* (*d*) *cystic* and (*e*) *infiltrative*. The primary tumor may be quite latent clinically, so that the peritoneal complication is apparently primary. The secondary growths spread by contact, by the lymphatics, and by implantation of free particles. They may be felt in the omentum, mesentery, and Douglas' *cul de sac* and in some cases may be confused with tuberculous masses. The diagnosis is usually reached by the symptoms and signs of the initial growth or by those of an ascites, which is serous, hemorrhagic, pseudochylous, or adipose; its progress is rapid and is attended by cachexia. Groups of cancer cells may be found in the aspirated fluid, and secondary cancer may develop at point of puncture. Differential features are tabulated under the t topic.

(E). ASCITES.

scites (hydroperitoneum) is a symptom and sometimes the chief ptom of various diseases.

tiology.—(*a*) It may result from ***stasis***, of which the chief varieties valvular and other *cardiac* lesions, pulmonary, mediastinal or ral disease, and *portal* stasis, due to disease of the liver or of the tal vein. Stasis causes increased pressure in the portal system, wed by malnutrition of the vessels and pouring out of serum. (*b*) *dræmia or cachexia* causes malnutrition and increased permeability he vessel walls; it occurs in amyloidosis, chronic nephritis, marantic ditions, profound anæmias, and occasionally in acute affections. *Inflammation*, as simple, tuberculous, suppurative or cancerous tonitis, may be an etiological factor. (*d*) *Abdominal* tumors, æmic spleen, etc., may cause ascites. In many cases more than factor may operate; tuberculous peritonitis may complicate liver hosis or ascites may be due to the hydræmia of nephritis, accentu-l by weakness of the heart.

ymptoms.—1. Inspection.—This shows a *symmetrical enlargement* he abdomen, which, in the average case, presents rather more lateral n anterior bulging. The abdomen resembles that of a batrachian *ntre de batracien*). The skin is pale, tense, striated, œdematous; *pouting navel* and diastasis of the recti muscles are noted. A *ut Medusæ* may be noted in the cases of cirrhosis. Dilated veins er the ribs and sides of the upper abdomen indicate compression of cava inferior. Sometimes the heart, beating upon the fluid, im-rts systolic waves to it.

2. Palpation.—Fluctuation (*ballottement*), due to the transmission a fluid wave from side to side, is usually present but may be absent cause of tense abdominal walls or great accumulation of fluid. eudofluctuation may be caused by accumulation of fluid other than itic, as intestinal contents, or by lax, obese abdominal walls; in latter condition a third hand placed in the median line of the domen will break the deceptive wave, which is due to superficial parietal vibration. Fluctuation is felt in ascites even where great npany exists. In women the *water-pillow* fluctuation (Landau), y be elicited *by vaginal examination*, and Tripier remarks that the rus seems remarkably movable. *Thrusting palpation* may disclose outline of the liver, spleen, or possibly some neoplasm even through abundant fluid: it is performed by placing the fingers vertically the abdomen and suddenly thrusting them inward, thus anticipating rigidity of the muscles and pushing aside the ascitic fluid.

3. Percussion.—At least a pint to a quart (500 to 1,000 c.c.) of fluid st be present for clinical detection. With small effusions, the ient should be examined in the genupectoral position—which is the best method, according to Leube. Elevation of the buttocks causes scanty fluid to gravitate to the flanks where its presence is more easily demonstrated. Classically, the *fluid* in dependent parts *gives dulness*, and the *supernatant intestines* are located by *tympany* in the highest parts of the abdomen. These areas *vary with change of position*, always giving, in uncomplicated cases, *tympany above* and *dulness below*, in any posture the patient may assume. When the patient lies upon his back, the tympanitic area is central and elliptical. This rule is void when the mesentery is retracted and does not allow the gut to float, in which event there is dulness over the highest areas on light percussion, though tympany can be elicited on deep percussion over the same area; adhesions between the intestinal loops prevent shifting of the fluid; two lines of tympany in the flanks mark the ascending and descending colon; when the gut is adherent anteriorily, there is permanent tympany in that location, and the author knows of an instance of ascites in which, ignoring this possibility as well as the physical signs, a physician perforated the intestines, though happily without unfavorable issue; immovable tympany in the flanks in ascites may be due to extensive adhesions of intestinal coils to the lateral abdominal parietes. Light percussion is employed in the determination of ascites, because heavy tapping elicits tympany from the adjacent or more deeply-situated intestine.

A large quantity of free fluid may, in very rare instances, exist without dulness. The author has withdrawn two gallons of ascitic fluid from a patient in whom no dulness could be elicited in any position.

Œdema of the subcutaneous tissues renders percussion of the abdomen difficult, as do fatty abdominal walls, fatty omentum, and fatty mesentery. The manometric pressure in the abdomen amounts to 20 to 40 mm. of mercury.

Diagnosis.—Although ascites is usually one of the most easily demonstrable physical conditions, nevertheless many excellent clinicians have mistaken it for other affections. *Fæcal accumulations* cause dulness in either flank, simulating fluid, but the history of the case, the immobility of the dulness, and the use of enemata or catharsis, preclude error. Large quantities of *semifluid fæces* in the ascending and descending colon may closely resemble free fluid; on thrusting palpation a splashing sound may be elicited over the colon, and Kuessner publishes a case in which a large quantity of fluid in the intestines was mistaken for ascites. Leube confused ascites with an enormously *dilated stomach*. Preperitoneal lipoma, hydronephrosis, hydatid cyst, pregnancy, dilated bladder, ovarian cyst and tympanites are frequently, though unnecessarily, sources of error.

Rostan (1837) noted that tympany might be absent in ascites when the intestines contained little or no gas, a point sometimes forgotten in differentiation between ascites and *ovarian cyst*, the latter of which may also thrust itself in between the coils of gut and simulate ascites; ovarian cyst usually produces a central flatness with tympany in the flanks, and neither the dulness nor tympany shifts with change of po-ture. Adami has collected 42 cases of fatty tumor (*fibrolipoma* and *myxolipoma*) in which ascites is very closely simulated.

Etiological Diagnosis.—1. Stasis.—In *cardiac disease* the dyspnœa develops early, but that caused by ascites pressing upward on the diaphragm is a late symptom; swelling of the feet usually antedates the ascites, though Strümpell (1883) pointed out that pericarditis and valvular disease may exceptionally produce ascites without anasarca. It must be borne in mind that a marked ascites may crowd the heart upward and outward. The *liver* is peculiarly an etiological factor in ascites (see DIFFERENTIAL CHART under LIVER DISEASES). Portal ascites commences almost invariably in the peritoneum; the legs swell secondarily from pressure on the vena cava inferior or from cardiac weakness due to great ascites (Giovanni); the legs may swell disproportionately, cicatrices may involve the portal vein and cava simultaneously at the notch in the posterior surface of liver, and finally the legs may swell first. In some cases the ascitic fluid may lie between the diaphragm and the liver, which latter is then pushed downward and may appear large though it is merely dislocated. An attempt should be made to exclude *perihepatitis*, which, according to Fagge, causes one death to every five fatal cases of cirrhosis, and is more apt to be attended by albuminuria (see TABLE given below).

2. Hydræmia. (Cachexia, Increased Vascular Permeability).—In renal disease the eyelids often become œdematous before other symptoms develop and the nephritic ascites is seldom great unless the liver and heart are involved. Cachectic ascites is not uncommon in leukæmia and kindred affections. Slight serous effusions occurring in acute infectious diseases in children may come under this title. The author has seen three instances of ascites in typhoid convalescence.

3. Peritonitis.—The specific gravity and the percentage of albumin in the fluid are important factors in differentiating between exudate and transudate. Three divisions are distinguished: (*a*) a specific gravity of 1,010 or lower indicates a *cachectic transudate, e.g.*, in nephritis with less than 1 per cent. albumin. A very low specific gravity and percentage of albumin occurs in amyloidosis. (*b*) A *hypostatic transudate* has a specific gravity which ranges between 1,010 and 1,014, and has between 1 and 3 per cent. of albumin. (*c*) An *exudate* has a specific gravity of more than 1,015 (or 1,018) and the albumin reaches 4 to 6 per cent. The lower strata are heavier, and the specific gravity should be measured several times during the paracentesis. When there is less than 1 per cent. of albumin, no disease of the peritoneum or portal vein exists. Reuss' formula enables us to compute the albumin percentage from the specific gravity. It is subject to an error of less than $\frac{1}{4}$ of 1 per cent.; the percentage of albumin = $\frac{3}{8}$ (specific gravity minus 1,000) minus 2.8. A specific gravity of 1,017 to 1,020 positively indicates an exudate. There are certain exceptions to this rule, and certain confusions may arise; thus an ascites due to a carcinoma in the liver substance has a low specific gravity and albumin is present to 1 or 2 per cent. If the carcinoma spread to the peritoneum (carcinomatous peritonitis), the specific gravity rises and 5 per cent. of albumin, or more, is found. An ascites due to nephritis has a low specific gravity but the figure rise when stasis due to weak heart or intercurrent inflammation develops. In many cases of simple transudation (ascites and hydrothorax) the characters of an exudate may develop, due to the long-continued irritation of the serosa.

Ascitic fluid always contains the constituents of the blood—albumin, globulin, urea, xanthin, hypoxanthin, guanin, uric acid, sugar, etc. (*a*) Ascitic fluid due to stasis is clear yellow or yellow-green, opalescent, and alkaline, and microscopically contains some lymph cells, erythrocytes, and peritoneal endothelium. (*b*) Exudates show a microscopic picture varying with the cause; fibrin threads, pus, blood, adipose, chylous or chyliform fluid, intestinal contents, cancer cells, tubercle bacilli, diplococci or pyogenic organisms, may be found. In exudates are found the polymorphonuclear leukocytes and some erythrocytes; lymphocytes and red cells are detected in transudates and tuberculous exudates (see PLEURISY); the fluid must be examined at once or formalin should be added for the cells readily degenerate and shrink. Rieder, Dock and others describe cells in carcinomatous peritonitis in which asymmetrical karyokinetic figures appeared. Quincke states that red disks, lymph cells, and peritoneal endothelium are found in every ascites.

Primavera differentiated between transudates and exudates by means of glacial acetic acid; a single drop will leave a white cloud as it falls to the bottom of a vessel containing an exudate, while its addition to transudates gives no reaction.

The French authors, especially, assert the existence of a primary ascites due to cold, excessive catharsis, drinking freely of water, suppression of menses or checking of hemorrhoidal bleeding. This so-called "essential" ascites is usually a chronic serous or tuberculous peritonitis.

Differentiation Between Serous and Tuberculous Peritonitis.—Hench believes that chronic serous peritonitis occurs in children though West and Courtois-Suffit think such affections are tuberculous. In tuberculous peritonitis (*q.v.*) there are usually temperature, emaciation, and bacilli in the exudate. According to Tepret and Vierordt, chronic nontuberculous peritonitis occurs especially in women, the causes of which are Bright's disease, trauma, portal stasis, alcoholism, and heart affections. The ascitic forms of nontuberculous peritonitis may pass into the membranous or adhesive variety (peritonitis deformans or pachyperitonitis). The diagnosis of simple nontuberculous peritonitis is most difficult to establish, even at autopsy, and the diagnosis in doubtful cases of a tuberculous peritonitis is by far more frequently justified.

Differentiation Between Tuberculous Peritonitis and Ascites.—Mechanical (portal) ascites is accompanied by other circulatory disturbances, as splenic tumor, piles, or caput Medusæ. The spleen is rarely enlarged in tuberculous peritonitis. The specific gravity and percentage of albumin are almost final in determining between peritonitis and ascites. The fluid is generally clear in ascites and usually cloudy in peritonitis. Pain and fever usually indicate encapsulation. The liver is small in the terminal stage of atrophic cirrhosis, which is also marked by various urinary changes; these are evidences of incomplete discharge of the hepatic function. In tuberculous peritonitis, the *hydrops ascites saccatus* gives an irregular form to the abdomen from meteorism produced by adhesions and from irregular nodules sometimes simulating carcinoma: it was described by Hippocrates. Free tuberculous exudations differ according to Thomayer, from simple ascites in that the tympany in tuberculous forms is often right-sided, due to retraction of the mesentery (mesenteritis retrahens) to the right side, carrying with it the small intestine. Mesenteritis retrahens is usually either tuberculous or carcinomatous, but may sometimes be due to simple deforming peritonitis. The author recalls several instances in which right-sided tuberculous masses pushed to the left both the tympany and the fluid. Hemorrhagic ascitic fluid occurs frequently in tuberculous peritonitis, and rarely, if ever, in cirrhotic transudate.

The "apparent tumors" (Bamberger) of tuberculous peritonitis are always suggestive where the nature of an ascites is in question. In two instances observed personally, the omentum was so retracted and so peculiarly adherent to the normal liver as to resemble most closely the edge of an enlarged cirrhotic liver. Intestinal or pulmonary tuberculosis, adhesive pleuritis, and meteorism from intestinal adhesions, or an irregular form of the abdomen with a "doughy" feel on palpation, indicate tuberculous peritonitis. A good working rule is that a peritoneal exudate is tuberculous, when cancer, sarcoma, and trauma can be excluded. Exploratory incision is often the only direct route to diagnosis and appropriate therapy. In the case of Heidenrich, in which the fluid was encapsulated, each of about 20 separate sacs was filled with fluid differing from that found in the others; in one was clear serum; in another paler fluid; in others, the effusion was brown, purulent, thick, or sanguinolent.

Nichols, in an excellent monograph ("A Somewhat Rare Form of Chronic Inflammation of the Serous Membranes [Multiple Progressive Hyaloserositis]," *Royal Victoria Hospital Reports*, vol. i, No. 3) gives a differential table, presented on the following page.

4. **Tumors.**—Tumors, other than peritoneal, may cause ascites. Uterine fibromata produce hydroperitoneum, which is not usually great, is lemon-colored, and but rarely sanguineous. There is no relation between the size of the tumor and the amount of fluid transuded, because very small uterine myofibromata may excite considerable peritoneal effusion. There is, nevertheless, a direct relation between the malignancy of the tumor and the amount of the fluid found (Terillon). As a general statement, ascites is infrequent in uterine tumors (Cruveilhier) but when observed is most common in

	"Zuckerguss-leber"; chronic perihepatitis.	Atrophic cirrhosis of liver.	Chronic tubercular peritonitis.	Carcinoma o[illegible]f peritoneum.
Age.	Occurs about middle life or later.	Oftenest about middle age.	Commonest between ages of 20 and 40.	Occurs late in li[illegible]fe.
Sex.	Both sexes equally liable.	More frequent in males.	Predominates in females.	More frequent i[illegible] fe-males.
Previous history.	Often a history of acute pericarditis or perihepatitis.	History of alcoholism, syphilis or digestive disturbances.	Often a chronic cough, diarrhœa, or genital tuberculosis.	In some cases a [illegible] history of cance[illegible] of stomach or ov[illegible]ies.
Alcoholism.	No influence.	Frequently present.	Unimportant.	Unimportant.
Syphilis. Heredity.	No influence. No influence.	Occasionally. Unimportant.	Unimportant. May be family taint.	Unimportant. Unimportant.
Incidence.	Acute becoming chronic or insidious from first.	Insidious.	Onset may be acute or insidious.	Insidious.
Chronicity.	Cases last for 2–10–16 years.	May last for years.	Prolonged.	Fairly rapid co[illegible].
Fever.	Generally absent except during exacerbation or some complication.	May be afebrile; when present is slight.	Usually slight; often absent.	Rarely absent; [illegible]ue to complicatio[illegible]
Pain.	Indefinite and trifling.	Trifling.	Apt to be troublesome.	Variable.
Digestive disturbance.	Trifling or none.	Constant; dyspepsia, nausea, vomiting, gastric hemorrhage, melæna.	Fairly common.	Often marked.
Ascites.	Constant and extreme; nutrition long maintained—Osler's case tapped 121 times; may resemble peritonitis, albumin 3 per cent., fibrin.	Constant.	Never extreme, may be absent, may be hemorrhagic.	Moderate [illegible] may be he[illegible] rhagic or p[illegible] chylous.
Anasarca.	Constant but slight.	Relatively slight.	Trifling.	Slight.
Jaundice.	Absent in pure cases.	Occurs in 27 per cent. of cases.	May occur exceptionally.	Common when [illegible]ver is enlarged.
Liver.	Not cirrhotic; at first enlarged, then small; smooth.	Cirrhotic; at first enlarged, then small and warty.	Often enlarged.	May be enlarg[illegible]d, with nodules.
Spleen.	Gradual enlargement.	Gradual enlargement.	Nothing special.	Nothing special.
Omentum.	Thickened and contracted.	Normal.	Often matted up.	Often matted u[illegible]

adenomyoma (Freund). It is more often seen in papillomatous cys[illegible]; in glandular cysts whose walls rupture by fatty degeneration and po[illegible]ur out their contents into the abdominal cavity, serum is secreted by t[illegible]he peritoneal surface because of the irritation, even though the ruptur[illegible]ed cyst be very small (Quénu). The fluid in cysts contains more soli[illegible]ds (60 to 50 pro mille) than does the ascites of renal disease (25 p[illegible]ro mille, Méhu). Ascites is most often observed in solid ovarian tumo[illegible]. In an ovarian sarcoma successfully removed by operation, an ascit[illegible] existed whose specific gravity was 1,024; the case recovered and remains well after nine years.

Treatment.—The treatment of ascites varies with its cause, for which reference should be made to valvular heart disease, atrophic cirrhosis, tuberculous peritonitis, and nephritis.

Paracentesis in peritoneal carcinoma and chylous ascites (*v. i.*) hastens the issue. Hale White asserts that cases of cirrhosis survive but few punctures, while cases of perihepatitis may be punctured more than 200 times. Lécanu reports a case of ascites in which 885 tappings were performed in fifteen years. In puncture, surgical antisepsis is most necessary, for reduced subjects are easily infected. The bladder should be emptied by catheterization. The trochar should be introduced in the *linea alba* to avoid injury to the bloodvessels. It should be pushed carefully and is felt to penetrate the parietal peritoneum by the sense of something giving away. A dull-pointed probe should be sterilized, with which to push back the omentum if it should prolapse against the canula. The fluid should be evacuated slowly to obviate collapse which may result from dilatation of the abdominal veins, if they are suddenly relieved of the pressure of the effusion. A cat-o'-nine-tails bandage may be applied as the tapping progresses, to compress the abdominal contents.

Chylous and Adipose Ascites.

The peritoneal cavity, much more frequently than any other serous sac, is the seat of those unusual exudates and transudates known as chylous, chyliform, lactiform, oily, or adipose ascites. The first authentic case is Poncy's (1699). During the last two centuries the literature gives about 200 cases. Seven have come under the author's observation.

Much confusion has arisen from inaccurate nomenclature. *Chylous ascites* properly designates an effusion of chyle. *Adipose ascites* contains a large percentage of fat with no chylous admixture. Some authors use the terms "adipose," and "chyliform" interchangeably. Boulengier prefers the term "chyliform" and "lactiform" to "chylous" on the ground that the fluid is rarely chyle. Chyliform ascites is a chyle-like fluid in which lymph or chyle is mixed with exudates or transudates.

1. **Chylous Ascites.**—Straus' case is the clearest example, in which chemically typical chyle was extravasated through two fistulæ. Ingested butter was recognized in the fluid withdrawn by paracentesis. Senator's latest case gave similar results with the administration of olive oil. Chylous ascites contains sugar, which is said to be diagnostic when diabetes is excluded. Sugar when primarily present may disappear later (Whitla). Its presence is very rare and very suggestive; it has been detected not more than two dozen times. Of late many clinicians discount the importance of the presence of sugar as a diag-

nostic test; Bock found sugar (0.04 to 0.07 per cent.) in all cases of hydrops, and Eichhorst in ten out of seventeen cases of pleural exudate. A small amount of fat (0.9 per cent.) indicates chyle. Chylous ascites is rich in solids, mineral salts, and albumin. Albumin and fat occur in small punctiform granules, susceptible of chemical differentiation. The escape of chyle may occur through a visible rupture or by transudation through the walls of the chyle vessels; among the most frequent causes of lymph or chyle obstruction are compression of the thoracic duct or lymph system by glands, neoplasms, peritonitis, occlusion of left subclavian vein, pressure efforts, lifting or coughing, filarial disease, or occlusion of the thoracic duct. Rupture may occur in any part of the lymphatic system; in the thoracic duct, receptaculum, lacteal vessels, lymph glands, and chylous cysts. Chyle may extravasate into a serous effusion by mural alteration of the lymphatic vessels.

2. Adipose or Chyliform Ascites.—Adipose ascites is characterized by the absence of sugar and a higher percentage of fat. The degree of opacity is no index of the amount of fat, because turbidity may be due to emulsionized albumin. Fat is found in most cases; its greatest amount is 5.25 per cent. (Schmidt), and in one of the author's cases 6.5 per cent. The granules of albumin and fat are much coarser in adipose than in chylous ascites. Hydropic and fatty carcinoma cells may, if numerous, cause a creamy layer, and their microscopic detection favors the diagnosis of hydrops adiposus. Red blood disks may occur, also fibrin, filaria, casein, mucin, sodium alkali albuminate, bile, acetone, hydropsin, peptone, lecithin, cholestearin, fibrinogen, diastatic ferments, urea, and various inorganic compounds. Among the numerous etiological conditions are tuberculosis of the peritoneum and glands; carcinoma of the glands, peritoneum, and lymph vessels; chronic peritonitis; liver cirrhosis, heart disease, and sarcoma of the omentum or mesentery.

Diagnosis.—A diagnosis has never been made prior to puncture, except in Morton's early case. The local signs and symptoms do not differ from those of serous ascites. It is probable that some cases diagnosed as the vulgar ascites, healing without treatment, are instances of chylous hydrops; this explains the increasing frequency of these affections. It is not easy to differentiate between chylous and adipose effusions, even at necropsy.

Prognosis.—About 90 per cent. of both types die. Continuous chylous fistulæ are always fatal. The immediate prognosis is seemingly more favorable in the adipose form.

Treatment.—Surgical interference is chiefly indicated in tuberculous peritonitis. Paracentesis should be avoided as much as possible for it is weakening, and precipitates the fatal issue.

SECTION V.

DISEASES OF THE KIDNEY.

(A). ACUTE NEPHRITIS.

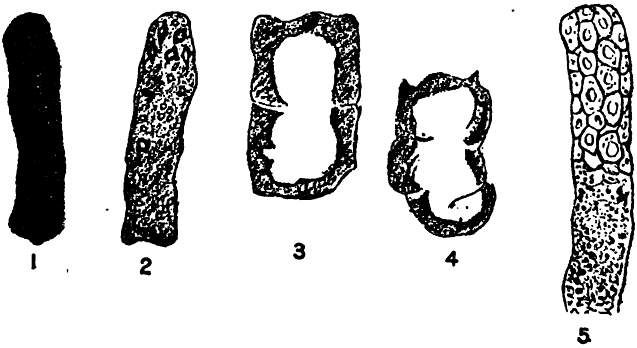
Etiology.—(*a*) Acute nephritis may result from *infections*. The *scarlatinal* nephritis (*q.v.*) is the most frequent type, and develops during the third week after desquamation. It also occurs in diphtheria, at the height of the disease; it is found in a large percentage of cases of acute enterocolitis in children; it may result from malaria, variola, sepsis, ulcerative endocarditis, or pneumonia; there is scarcely an infection in which acute nephritis may not occasionally develop, as typhoid, typhus, measles, tonsillitis, or epidemic meningitis; it is rare in German measles, varicella, or purpura. It may develop in tuberculosis and syphilis. Infections operate (i) more often by their toxins than (ii) by the causal microbe, though the typhoid bacillus, recurrens spirillum, pneumococcus, streptococcus, and others, have been found in the kidneys and urine; (iii) in some instances (nephritis hæmoglobinurica) hæmolysins develop and apparently cause acute nephritis. (*b*) *Toxic substances*, taken internally or applied to the skin, may induce acute nephritis; as turpentine, salicylic compounds, carbolic acid, potassium chlorate, cantharides, and kindred substances. (*c*) Exposure to *cold and dampness* (with alcoholism) is frequently the sole apparent cause, though its mechanism is obscure; possibly it lessens the tissue resistance or acts as a hæmolysin, as in the hæmoglobinuric type. (*d*) *Skin diseases*, burns, eczema, and psoriasis, may be etiological factors, either by lessening the skin functions, by producing hæmolysins, or by too active medical treatment (nephritis medicinalis). (*e*) *Pregnancy* (*v.i.*) may result in nephritis. (*f*) The cause is often *obscure;* chronic Bright's disease, cardiac affections, and the dyscrasiæ, predispose to acute nephritis. (*g*) Acute neph may occur independently of any known cause; this primary f may be epidemic.

Pathology.—*Gross changes* may not be apparent in mild ca though in some the kidneys are swollen from marked intersti exudation, and are dark and heavy. In other cases the kidne yellow-white, and perhaps mottled from small hemorrhages. T œdematous capsule strips readily and the cut section drips blo On section the cortex is swollen and granular, and its striations blurred and pale in contrast with the dusky medulla; the glomer are frequently visible as red or later as yellow dots. Without t microscope, lesser degrees of acute nephritis may be confused wi fatty or parenchymatous degeneration. *Microscopic changes* a usually diffuse. (*a*) *Glomerular* changes. Glomerulonephritis, esp cially described by Klebs, is caused by toxins reaching the tufts throug the blood current. It is best exemplified by the scarlatinal form (*q. v.*). The glomerular capillaries show infiltration with leukocytes, an albuminous or cellular exudate escapes into the capsule, and the epithelium of Bowman's capsule degenerates, desquamates, and later proliferates; in some cases numbers of red disks escape with the serum and leukocytes. (*b*) *Tubular* changes. Cloudy swelling, hyaline and fatty degeneration occurring especially in the cortex, may be difficult to differentiate from febrile and other degenerations, though interstitial exudation occurs in every case of nephritis. In severe cases some cells necrose entirely and desquamate; of this the diphtheritic form is the best type. The swelling in and around the convoluted tubules is one cause of the kidney enlargement. (*c*) *Vascular* and *interstitial* changes are essential to the pathological diagnosis and are more focal than diffuse. Serum exudes into the connective tissue, tufts, and tubules, and coagulates in them; it is probably the cause of the casts; leukocytes escape into the parenchyma and interstitium, and in some case red cells also (hemorrhagic nephritis). Councilman describes Unna' plasma cells, which he thinks are conveyed to the kidney from th spleen and marrow.

Symptoms.—The *onset* may be *sudden*, especially in cases followi exposure. In other cases it develops *gradually* in the course or co valescence of the causal infection as scarlatina, diphtheria, etc. many cases it may be unrecognized unless frequent analyses of urine are made, or until uræmic symptoms develop.

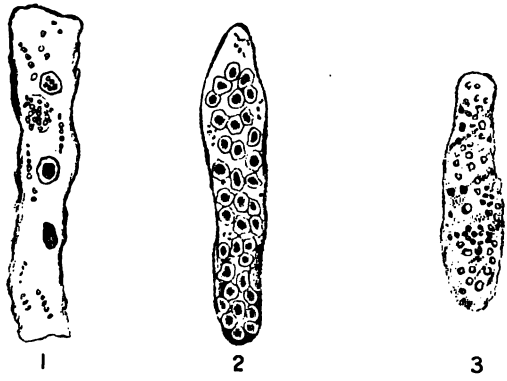
1. Urinary Findings.—(*a*) The *urine is decreased* to ten or tw ounces (a few hundred c.c.) or in severe cases is *suppressed;* th due to glomerulitis or stoppage of the tubules by coagulated alb and desquamated epithelium; the decrease usually runs paralle the intensity of inflammation and hydrops. During convalescen urine becomes abundant and clear. (*b*) Its *color* may range fro of febrile urine to a *dark* smoky tinge; it is seldom red or a bloody; in the hæmoglobinuric form it is chocolate-colored. specific gravity averages 1,025 to 1,030 and (*d*) the reaction (*e*) *Albuminuria* is almost invariable; the albumin amounts to $\frac{1}{2}$ to 1 per cent. and one to two drams (5 to 8 gm.) daily. There is serum albumin, considerable quantities of globulin, and if there are many cells as in tubular nephritis, nucleo-albumin. The author has seen six fatal cases of acute diffuse nephritis, confirmed by autopsy, in which there was no albuminuria. Similar cases are reported by Quirolo, Henoch, Senator, Fermi, Sanné, and others. (*f*) *Urea* is *decreased* even to $\frac{1}{6}$ its normal quantity, as are also the *chlorides* and phosphates; the uric acid remains normal, and the xanthin (alloxur) bases may be increased; for the molecular constitution of the blood and cryoscopy, see CHRONIC NEPHRITIS. (*g*) The *sediment* in mild acute tubular nephritis consists of many *epithelial cells*, some red and *white corpuscles*, and hyaline *casts*, with crystals of oxalate of lime and uric acid, and sometimes hæmoglobin granules, which may be also found as free granules. In diffuse glomerular nephritis there are almost always *red cells*, mono- and polynuclear leukocytes, epithelium, fatty, hyaline and granular casts, granules of fat and often of hæmoglobin, uric and

FIG. 45.



Casts. 1–4, amyloid; 5, cast, epithelial in its upper, and granular in its lower, part.

FIG. 46.



Urinary casts; 1, hyaline, with fat droplets and cells; 2, hyaline, with leukocytes; 3, cast with fat droplets.

calcium oxalate crystals, and sometimes various micrococci. Senator maintains that the leukocytes from the kidney are mononuclear. Waxy and epithelial casts are found only in the severest types. Blood and casts may appear before and may also outlast the albuminuria. The significance of casts (cylindruria) will be considered under chronic parenchymatous nephritis.

2. Hydrops (Anasarca).—This is often the first symptom to attract attention. (*a*) It occurs *about the eyes* early in the morning and later in the day in other parts, as the limbs and external genitalia. As the process advances it may become general. (*b*) It changes its location readily, and with *anæmia* gives a rather *characteristic facies.* (*c*) Anasarca occurs most commonly in cases due to *scarlatina and exposure,* rather less commonly in those following pregnancy, malaria, alcoholism, and skin diseases, and seldom in those developing in sepsis, pneumonia, typhoid, or diphtheria. (*d*) It is caused by *inflammation* of the *skin vessels,* is identical with the inflammation in the glomerular vessels, and is nearly always associated with glomerulitis. (*e*) Identical changes may occur in the *serous sacs* (pleura, pericardium, or, far less often, peritoneum, meninges, and joints), *mucous membranes* (glottis or intestinal tract), or viscera (pulmonary or cerebral œdema). (*f*) It parallels the intensity of the nephritis and the decrease of urine.

3. Anæmia.—Anæmia is often an early symptom and with the dropsy is almost suggestive of acute nephritis. The red cells and hæmoglobin are reduced, the specific gravity of the serum is lowered, and the freezing point is raised (*v.* CHRONIC NEPHRITIS).

4. Cardio-Vascular Symptoms.—The right heart may become dilated. Hypertrophy of the left ventricle is very uncommon, though it was observed by Riegel and Leube within ten days of the beginning of the disease. The pulse is usually slow and tense; the sphygmogram shows increase of the tidal and decrease of the dicrotic wave, and the sphygmomanometer shows the blood tension increased even to 180 or 200 mm.; these phenomena are due probably to vascular contraction induced by metabolic products retained in the blood. The second aortic tone may become accentuated, and, if heart fatigue develops, the gallop-rhythm is heard.

5. Temperature.—Fever and rigors are exceptional.

6. Pain.—Occasionally there may be pain and tenderness in the back, frequent and painful urination, emaciation, and epistaxis. Constipation is the rule. The skin is generally dry.

Diagnosis.—The urinary findings are characteristic; *albuminuria* (*q.v.*) may result from other causes, as fever, amyloidosis, or stasis; casts, leukocytes, epithelia, and even red cells, may also occur in chronic nephritis, but the history and evolution determine the diagnosis (*v.i.* DIFFERENTIAL TABLE OF DIFFUSE DISEASES OF THE KIDNEY); cases without urinary findings are extremely rare. The greatest danger lies in the diagnosis of an acute nephritis, when the condition is only an *acute exacerbation of a chronic process;* fully half of the so-called idiopathic acute cases which the author has seen belong to this category.

In *renal hæmaturia* the urine contains no casts (save perhaps red cell casts), few leukocytes, and practically no epithelia; there is no anasarca; the urine is clear at one time and is laden with blood at another. *Anasarca* (*v.s.*) occurs in certain types only and in various cachectic states.

Diagnosis of Types.—(*a*) The *kidney of pregnancy* usually develops in the last half of gestation, and infrequently before the third month. Its pathogenesis is yet vigorously disputed, though toxæmia due to the double task imposed upon the maternal kidneys seems to be the most probable cause. It has been suggested that decreased antitoxic activity of the thyroid gland is a causal factor. The placenta is thought by some to elaborate toxins (syncytiolysins). The urine is decreased, pale, and of low specific gravity; it contains much albumin and not much sediment, though red and white cells, lymphocytes, fatty epithelia, and sometimes casts, are found. The attendant anasarca, like that of stasis, develops from below upward. When retinitis develops its prognosis is more favorable than it is in other nephritides. The convulsive seizures (eclampsia) are generally uræmic, though Ingerslev collected 106 cases in which there was neither clinical albuminuria nor renal alteration at necropsy. Fehling remarks on the slight anatomical changes in the kidney and on the fact that but 5 per cent. of pregnant patients having nephritis develop eclampsia. The exact explanation of the convulsions remains obscure, though it seems that the brain centres during gestation are unduly susceptible to irritation. The embolism of placental cells in the brain or lungs (Schmorl) is considered by some rather as a result than a cause of eclampsia. Zweifel maintains that the oxydation of the albumins is decreased, the nitrogen output is decreased, the ammonia (as in diabetic coma) is increased, and that sarcolactic acid is found in the blood and urine. The maternal mortality is 25 to 30 per cent., and that of the child over 50 per cent. Very few cases develop chronic nephritis. (*b*) The *cholera nephritis* is parenchymatous and chiefly tubular. Various factors are operative, as toxæmia, arterial anæmia, venous hyperæmia, and heart weakness. By some it is considered renal ischæmia rather than nephritis. The urea, chlorides, calcium and magnesium salts are decreased, and much indican, aromatic sulphates, acetone, and ammonia, are found. The anuria may last three or even fifteen days. (*c*) *Hæmoglobinuric nephritis* is probably due to hæmolysins, which disorganize the blood and irritate the kidneys by the hæmoglobin thus set free. It is found in burns, acute infections (typhoid, pernicious malaria, scarlatina, yellow fever, Winckel's disease of the new-born), exposure, hæmoglobinæmia, and various drug poisonings, notably that of chlorate of potash. Hæmoglobinuria, red disks in the urine, casts, leukocytes, epithelial cells, and abundant albuminuria, are indicative of hæmoglobinuric nephritis. (*d*) *Acute recurrent* (chiefly hemorrhagic) nephritis is uncommon.

Prognosis.—The prognosis of acute nephritis is always dubious. The following factors are important: (*a*) Recovery is usual in *light* *forms* not involving the glomeruli. Atypical forms are generally favorable. (*b*) The prognosis varies according to the *causal affection*, the scarlatinal type has a mortality of 33 per cent.; the virulence of the epidemic is an important factor, as are also heart complications. (*c*) *Complications* may affect the prognosis, as (i) intercurrent *inflammations*, such as pneumonia, pleuritis, pericarditis, or erysipelas; (ii) *œdema* of the pharynx, larynx, lungs, or serous sacs; and (iii) *uræmia*, which will be fully covered under chronic interstitial nephritis; uræmia is more often an adult than an infantile, and a later rather than an early, complication, though convulsions may be its first symptom; headache, delirium, dyspnœa, convulsions, coma, and vomiting, are among the salient signs of uræmia.

Course.—The course is seldom fatal within a few days. In general there are more recoveries than deaths. The average duration is less than two weeks, though one or two years may elapse before recovery. Convalescence is usually complete, though gradual transition into chronic nephritis sometimes results.

Treatment.—Most of the points mentioned will be considered more fully in the treatment of chronic nephritis. (*a*) *Prophylaxis* is of limited value. Drugs which irritate the kidneys should be carefull avoided in acute infections or in pregnancy. In fevers, with or without albuminuria, water should be given freely by mouth or by enema; hydrotherapy is prophylactic, save in scarlatina, when all prevention is seemingly futile and cold baths are contra-indicated. Induction of labor is indicated in severe cases in pregnancy. (*b*) The *diet* should be regulated. Milk is a moderate diuretic, is non-irritant, and contains little or no extractives or salt. It should be given as in typhoid (*q.v.*). Thoroughly cooked carbohydrates, as gruels, make a good combination with it. Broths and beef-tea are to be avoided, and meats should be administered only late in convalescence. Alcohol is contra-indicated, save as exceptionally necessitated by the causal disease. (*c*) *Absolute rest in bed* is imperative; rest with the body covered, protects the skin and spares the products of the tissue waste, which are eliminated by the kidneys; the patient should lie between flannel blankets and should be dressed in flannel nightgowns. (*d*) There is *no drug* which affects the nephritis; tannin and ergotin (of each five grains three times daily) seem to restrict renal hemorrhage. Salt should be largely withheld, because it increases albuminuria and œdema (see TREATMENT OF CHRONIC NEPHRITIS). (*e*) The best *diuretics* are *water and milk*, which flush out detritus from the kidneys. The alkaline charged waters seem to search out waste substances in the tissues and facilitate their removal by the kidney (but excessive quantities are often given); potassium citrate and sodium benzoate are said to have the same effect; the citrate may be given in doses of half a dram with sweetened weak lemonade. Sweet spirits of nitre ʒss to ʒj, q.i.d., is diuretic and also dilates the bloodvessels. All stronger diuretics are to be avoided. (*f*) The *skin* should receive consideration. *Warm baths* are diuretic and stimulate some vicarious elimination through the skin.

Careful covering and rest in bed keep the skin relaxed and not only materially aid its functions but also relieve renal hyperæmia. (*g*) Management of the *bowels* is as important as that of the skin. *Saline catharsis* lowers arterial tension and aids compensatory elimination. As salts often derange the stomach, an ounce should be given by rectum with two ounces of glycerine and one of water. One-half to one dram of compound jalap powder or $\frac{1}{12}$ grain of elaterin is indicated when uræmia is imminent. (*h*) *Treatment of uræmia* (see TREATMENT OF CHRONIC NEPHRITIS). For suppression of urine a full warm bath lasting fifteen to twenty minutes should be given; a hot pack should be applied followed by wrapping in blankets and covering heavily with a rubber sheet; or the alcohol sweat or hot-air sweat may be given as described under TREATMENT OF CHRONIC NEPHRITIS. Cups applied to the back may divert blood away from the kidneys, because the vessels of the loin anastomose with those of the kidney. Pilocarpin hydrochlorate, gr. $\frac{1}{6}$ to $\frac{1}{8}$ in adults, and one-half as much in children, causes profuse sweating and salivation but long ago the author abandoned its use after witnessing two deaths from sudden pulmonary œdema in patients with apparently strong hearts; of course it should never be employed when the heart is weak. Active, rapid catharsis is effected by giving two drops of croton oil on the tongue; this may be repeated in two hours if necessary. If uræmic symptoms (in an œdematous patient) appear slowly, an incision two inches long should be made over the lower tibiæ down to the bone, thereby directly draining away the serum with its retained toxins; this is better than the use of small punctures, or Southey's capillary tubes, which are attended by all the dangers of puncture-wounds. If uræmia develops suddenly, the legs should be incised and a pint or more of blood should be withdrawn from the veins of the arm; whether venesection relieves the circulation mechanically or removes toxins is still a matter of theory, but practically, relief is often obtained. It should be followed by subcutaneous normal salt infusions—or plain water without the salt—which stimulate the heart and kidneys and lower the molecular concentration of the blood. Pleural or ascitic hydrops should be aspirated. The heart must be stimulated by strychnine and digitalis as in cardiac insufficiency, though hypodermics are better than oral administration. A hypodermic of morphine is beneficial and harmless when there is much dyspnœa and restlessness. Chloral and bromides modify sudden convulsions (*v.* CHRONIC NEPHRITIS). Œdema of the lungs (*q.v.*), vomiting, and diarrhœa, are caused by several factors, *viz.*, cardiac stasis, uræmia, œdema of the alimentary mucosa, and the vicarious elimination of urea, which breaks up in the digestive tract into irritating ammonium; vicarious vomiting and diarrhœa should not be checked at once; if persistent, the vomiting is treated as outlined in chronic gastritis (*q.v.*) or typhoid; gastric lavage is beneficial, and dilute hydrochloric acid, ♏x to xv, neutralizes the ammonium. (*i*) *In convalescence*, iron and other mild tonics should be given, irritating foods and beverages interdicted, exercise carefully restricted, and cold and exposure should be avoided by sending the patient, if possible, to a warm climate.

(B). CHRONIC NEPHRITIS.

Our knowledge of chronic Bright's disease dates from Richard Bright's communications in 1827. Aetius, Avicenna, Cotugno (1770), and other writers, mentioned the association of hydrops and albuminuria, but to Bright is due the credit of correlating renal disease, albuminuria, and dropsy; he also noted the etiological importance of alcohol and cold, as well as the cardiac hypertrophy, uræmia, brain hemorrhage, coma, convulsions, blindness, and the tendency to serositis.

The following classification of nephritis is given as covering typical cases, though it must be borne in mind that *combined* or *mixed types* are equally frequent. As a cause of death, chronic nephritis ranks sixth, after pneumonia, tuberculosis, heart disease, endocarditis, and unknown causes.

I. Chronic Parenchymatous Nephritis.

Definition.—Chronic Bright's disease, with special involvement of the parenchyma (though the interstitial tissue is always affected), with much albuminuria, and with abundant formed elements in the urinary sediment. The affection constitutes 3 per cent. of all cases of disease.

Etiology.—It may result from (*a*) *infections*, as tuberculosis (which causes 25 per cent.), malaria, and syphilis; (*b*) from exposure to *cold and dampness;* (*c*) from toxic factors, as alcohol and lead, and less frequently others, as mercury or arsenic; (*d*) from *cardiac disease;* 14 per cent. of these cases develop nephritis. (*e*) Exhausting *discharges*, suppuration, and ulceration may cause it. (*f*) *Acute nephritis* seldom becomes chronic, though it is a somewhat predisposing factor. (*g*) *Age and sex* are of etiological importance. Most cases occur between twenty and fifty years of age; it is rare in children. Males are affected more often than females.

Pathology.—*Three types* of the disease exist, besides the mixed forms.

1. The "Large White Kidney" or "Inflamed Fatty Kidney."—This is never decreased in size and may weigh 10 to 12 ounces (300 to 360 gm.) and measure two or three times the normal size.

Gross Characters.—The capsule is loose and non-adherent; the surface is smooth, yellow-gray, with prominent stellate veins, and sometimes has dots of hemorrhage upon it. On section the cortex is broader than normal, the striations are poorly marked, and when cut streaks of fat may show; the glomeruli and convoluted tubules are yellow, and contrast with the gray bands of newly formed connective tissue. The medulla shows fewer changes, and is red, in sharp contrast with the light cortex.

MICROSCOPIC CHARACTERS.—The most essential change is hyaline and fatty degeneration and atrophy, chiefly in the convoluted tubules, in which lie desquamated epithelia and coagulated albumin. The glomeruli are always diseased, but to a varying degree; they are enlarged, their vessels show hyaline and fatty degeneration and nuclear multiplication; albumin is found in their capsules, and the epithelium is swollen, fatty, or even necrotic. The connective tissue is always increased, which histologically separates chronic nephritis from acute nephritis and mere degeneration. These changes are never equally diffuse.

2. The "Large Red" or "Variegated Kidney."—GROSS CHARACTERS.—It is also large, but is firmer than the kidney just described or that of acute inflammation, for it has more connective tissue, which may cause some tearing and loss of the cortex when the capsule is stripped off. The surface may be perfectly smooth. The cortex is swollen, indistinctly striated, dotted yellow from fatty glomerular change, or red from minute ecchymoses, and sometimes streaked yellow and red from similar tubular changes.

MINUTE CHARACTERS.—These are essentially those of the large white kidney, with the exception that fatty degeneration is less marked, and connective tissue alteration and hemorrhages are more conspicuous.

3. The Secondary Contracted Kidney.—This form may develop after a year or two from the above forms. Some consider it a primary, independent form.

GROSS CHARACTERS.—It is seldom decreased in size and may be as large as the "large white" or "large red kidney"; the name refers rather to its connective tissue than to any visible renal shrinkage. The capsule is adherent in places, and, when stripped, tears off small areas of the renal substance. The surface is rough and shows in some places reddish depressed areas (fibrous contraction or granulations, and parenchymatous atrophy) and in others yellow areas (which are more normal, though fatty). Section shows decrease in the cortex, corresponding to the reddish foci. It differs from the genuine contracted kidney (primary interstitial nephritis) in the following respects: it is secondary, much more rapid in development, contains fewer cysts, is more yellow, and its red granulations and yellow prominences are larger.

MINUTE CHARACTERS.—Epithelial changes resemble those of the two forms described. The membrana propria of the tubules thickens, adheres, and obliterates many tubules. The glomeruli collapse, thicken, and shrink to small dark-gray granular bodies, which sometimes calcify. The retracted areas, capsular adhesions, and cortex shrinkage, correspond to connective tissue contraction.

Symptoms.—The *onset* is in most cases *gradual* and *insidious*, the earliest symptoms being loss of strength, pallor, emaciation, simple decline of health, indigestion, anorexia, headache, shortness of breath on exertion, or chronic bronchitis. Sometimes there is tenderness over the kidneys.

1. **Urinary Findings.**—Except in the secondary contracted kidney (*a*) the *urine is decreased* to seven ounces or a quart (200 to 1,000 c.c.); (*b*) the color is *dark* yellow-red, opaque, or rarely smoky; the urates are held in suspension by the albumin; sometimes its surface is shimmering from fatty cells and casts; (*c*) the specific gravity is 1,020 to 1,040 and (*d*) the reaction is acid; (*e*) *albumin* is almost never absent, is *more abundant in the day time* than at night, and amounts to 1 to 3 per cent. and one-half to one ounce (15 to 30 gm.) daily; it coats the bubbles formed by agitation of the specimen and preserves them for a long time. (*f*) The absolute amount of *solids* is generally *decreased*; for example, the urea (which normally constitutes 90 per cent. of the nitrogen output and amounts to about an ounce [35 gms.] daily), kreatin, and sodium chloride, are decreased, and the freezing point of the urine is lowered (*v. i.*). (*g*) The *sediment* is *abundant*, and hyaline, granular, fatty, and epithelial *casts abound* (first noted by Simon and Nasse); coarsely granular and waxy casts appear late in the disease; the granular type is due chiefly to fat particles, which may darken the casts. They have been long regarded as significant of nephritis. Key, Nothnagel, Rosenstein, and others afterward, observed their occurrence in the urine of otherwise normal individuals, and of late there has been a tendency to underestimate their importance. According to Senator, casts originate in the kidney chiefly from epithelial cells, and always indicate some renal disease. They may be dissolved in the bladder by the pepsin contained in the urine (Sehrwald). Leukocytes and fatty epithelia, and, in some forms of nephritis, red cells, occur in considerable amounts. In Ackermann's case no casts were found in the urine, but were found in masses in the renal pelvis at necropsy.

In the secondary contracted kidney the urinary findings are the same except that the urine amounts to three pints or two quarts (1,500 to 2,000 c.c.), is clearer, is somewhat lower in specific gravity, and contains somewhat less albumin and casts.

2. **Œdema.**—Œdema is very frequent. Its absence argues for little glomerular involvement. It commences *about the eyes, changes* its location readily, and is at first evanescent, but soon *becomes general, obstinate*, and *ominous*. It is more pronounced in the "large white kidney" than in other types and increases as the urine decreases. The puffy eyes, pale swollen cheeks, dull expression, distended abdomen, and shapeless wrists and ankles, often tempt one to a snap diagnosis. The *serous* and *mucous membranes* are similarly involved, though to a lesser degree, and, also, as possible terminal events, pulmonary or cerebral œdema may develop. Œdema is caused (*a*) by hypalbuminosis and hydræmia of the blood (Bright's theory), or (*b*) by alterations in the vessel walls, *i.e.*, abnormal permeability, a theory advanced by Cohnheim and Lichtheim.

3. **Anæmia.**—Anæmia distinguishes chronic parenchymatous nephritis from the genuine interstitial type. There is usually retention of urea, uric acid, and chlorides; sometimes the blood is cloudy.

4. **Cardiovascular Findings.**—The pulse is more often small, soft, rapid, and associated with a weak apex, indicating dilatation, than tense and associated with a loud second aortic tone and strong apex, indicating hypertrophy. Secondary contraction and mixed nephritis are likely to develop findings similar to those of the genuine interstitial nephritis; Senator finds hypertrophy with dilatation (eccentric hypertrophy) in parenchymatous, and simple or concentric hypertrophy in interstitial nephritis. Myocardial degeneration is common.

Complications, and Causes of Death.—(*a*) *Marasmus* results from the anæmia, indigestion, diarrhœa, œdema of the alimentary mucosa or its irritation by decomposed urea. (*b*) Intercurrent *infections* may result, as inflammation of the pleuræ or pericardium, cellulitis, and especially pneumonia; in one instance the writer saw acute necrosis and sloughing of the scrotum, after which the patient improved remarkably; many pneumonia deaths really result from nephritis. Other complications are (*c*) *œdema* of the lungs, larynx, serous sacs, and extremities, impeding respiration and obstructing the arterial flow in the peripheral vessels; (*d*) *hemorrhages* (in 8 per cent.) in the brain or other organs; (*e*) *retinitis* (in 18 per cent.); (*f*) *uræmia,* with its numerous nervous, circulatory, and digestive symptoms (*v.* INTERSTITIAL NEPHRITIS); (*g*) *acute exacerbations,* particularly in the chronic hemorrhagic type; and (*h*) *cardiac insufficiency.*

Diagnosis.—The diagnosis rests upon three cardinal symptoms: (*a*) the *urinary,* which are characteristic when twenty-four-hour specimens are carefully and repeatedly examined in every case coming under our care; (*b*) the *anasarca,* which is usually present, though sometimes absent in secondary contracted kidney and in Wagner's hemorrhagic type; and (*c*) the *anæmia.* (For *differentiation,* see DIFFERENTIAL TABLE OF DIFFUSE RENAL DISEASES, pages 796–797.)

Prognosis.—Though some cases which suggest an arrest of the process are found at autopsy, and some cases may recover after a year or two, the clinical course is usually progressive and fatal. Dropsy usually causes death in a few months to a year, though the author has seen three typical cases apparently recover after two and a half years; these cases did not result from acute infections like the cases of recovery after one to two years reported by Senator and Rosenstein. The outlook is slightly better in children than in adults. The "large, white kidney" causes death in one-half to one year, the "large, variegated kidney" in one and a half to three years, and the secondary contracted kidney in one and a half to three years. The treatment is considered under chronic interstitial nephritis (*q.v.*).

II. Chronic Interstitial Nephritis.

Definition.—A primary, interstitial, contracting sclerosis of the kidney, characterized clinically by abundant urine of low specific gravity, with small amounts of albumin and few cellular elements; and by marked cardiac hypertrophy, arteriosclerosis, retinitis, and uræmia.

This genuine contracted kidney (primary contraction) must not confused with secondary contracted kidney, arteriosclerotic contr tion, embolic contracion or the contraction resulting from ascendi infection—*e.g.*, cystopyelonephritis.

Frequency.—In ten years Eichhorst treated 31,562 cases, of whi 409 (1.4 per cent.) had contracted kidneys; he finds this form six a a half times as frequently as the parenchymatous form, though wi us the latter is at least equally frequent.

Etiology.—The etiology is essentially that of *arteriosclerosis*, wi which contracted kidney has a three-fold relation: (*a*) arteriosclero may cause the arteriosclerotic kidney; (*b*) contracted kidney m cause arteriosclerosis; or (*c*) both may result from a common cau The noxa or toxin reaches the kidney through the blood stream.

The disease may result from (*a*) chronic *alcoholism*, especially wh combined with exposure or over-eating; (*b*) chronic *lead-poisoni* proven to be a cause by clinical and experimental evidence (Lancerea and Ollivier); (*c*) *gout* (Todd), caused by uric or oxalic acid a the alloxur bodies; (*d*) *syphilis;* (*e*) *diabetes* (Senator), caused sugar, acetone, oxybutyric acid, or kreatin; the diabetes lessens ceases when induration develops; (*f*) *age;* it begins between the ag of *thirty and fifty*, and is clinically manifest after fifty years of a Sawyer collected 24 cases in children, 13 of whom were under t years. (*g*) *Sex* is an etiological factor. The disease is two to thr times as frequent in *men* as in women. In the infantile form mo cases occur in girls. (*h*) Other *infrequent causes* are acute nephrit the kidney of pregnancy, hereditary tendency as in Dickinson's seri through four generations, and endocarditis.

Pathology.—It is undecided whether the process is inflammato or atrophic; Weigert believes that cellular degeneration occurs fir and induration follows.

Gross Pathology.—(*a*) The kidneys are *contracted* even to one-thir their original size and may weigh but 1.6 ounces (50 gm.), or 0.6 ounce (21 gm.); contraction is rarely symmetrical in both. (*b*) Their *con sistence* is *increased.* (*c*) The *capsule* is *thick*, tendinous, wrinkled vascular, and *adherent*, and strips off particles of cortex when removed (*d*) The *surface is uneven;* there are small reddish or sometimes pale prominences, measuring 0.5 to 5 mm. and representing the mo normal tissue; the paler retracted areas are fibrous retraction. (Small *cysts* are frequently found, usually with clear contents; the vary in number and their size ranges from that of a pin-point to tha of a cherry; they apparently result from glomerular and tubula constriction by connective tissue (retention cysts). (*f*) On section th *cortex is shrunken*, even to 1 mm., is irregular and gray from fibrilla connective tissue. The medulla is less changed, though also shrunken pale fibrous lines alternate with red dilated vessels; uric acid o calcium salts are deposited. (*g*) The pelvis is large and much fa surrounds the kidney.

Minute Pathology.—The changes are largely cortical, focal, asymmetrical, and interstitial. (*a*) The *glomeruli* early contain round cells, nuclear increase, desquamated cells, and albumin; later they *atrophy* from degeneration of the afferent artery; multiplication of cells is noted between the loops, and hyaline change occurs in the glomerular vessels; in the extreme stage the glomeruli become *hyaline and fibrous;* some tufts remain normal and others may visibly hypertrophy, Sabourin's compensatory adenomata. (*b*) The *epithelium* is always changed, though much less than in parenchymatous forms; in the sunken areas many tubules disappear, and degenerated cells are seen in the prominent surface granulations; in the medulla the collecting tubules are larger, even cystic, and are filled with colloid material. (*c*) The *connective tissue* is increased everywhere, but more in the cortex than in the medulla; round cell infiltration occurs early and induration late; the vessels are sclerotic in all their coats, but especially in the intima, and many writers consider that the vascular changes are the earliest and causative alteration.

Symptoms.—*Long latency* for years, *insidious onset*, and *protracted compensation* by *heart hypertrophy*, are characteristic of this type. Many cases are first discovered during an acute infection, as pneumonia; they may be found on examination for life insurance, or at the coroner's autopsy after accidents. It may appear to be a physiological, intermittent, or cyclic albuminuria. Polyuria, cardiac symptoms, retinitis, indigestion, or depreciation of health, may be the first symptoms. The œdema, pronounced urinary findings (in a single specimen) and anæmia of chronic parenchymatous nephritis, are seldom conspicuous and almost never early symptoms in this type. The condition may be first declared by heart incompetence, uræmic coma, or convulsions.

1. **Urinary findings** are generally the earliest signs. Twenty-four-hour specimens should be examined (with a fresh specimen for microscopic examination) repeatedly in doubtful cases. (*a*) The *amount*, which is at first normal, is gradually increased (*polyuria*); this is a frequent cause for consulting the physician. The urine is also passed too frequently (*pollakiuria*), especially at night; this is always suspicious when the prostate is normal. In well-developed cases it totals two to three quarts (2 to 3,000 c.c.), rarely more, though 8 to 10 quarts (8 to 10,000 c.c.) were passed in a case of Lecorché and Talamon and 12 quarts in Bartel's case. (*b*) It is *pale* and (*c*) *weakly acid.* (*d*) The specific gravity averages 1,010 (1,005 to 1,012). (*e*) A *permanent foam* due to the albumin is frequent. (*f*) *Albumin* is present *in most cases*, but is *not abundant;* the merest traces are found up to 8 grains (0.5 gm.) pro mille, and a daily total of one-half to one dram (2 to 5, rarely 10 gm.). It is often absent in single specimens, especially in the morning, whence a twenty-four-hour specimen should always be examined. Delicate tests (*v.* ALBUMINURIA) are often necessary. It may appear only after eating, exercise, excitement, or fatigue, whence its greater frequency toward night; it may increase after

dyspepsia or bronchitis. Senator's statement that its alleged absence is due to an arteriosclerotic kidney and not to interstitial nephritis is not invariably correct, according to personal experience. Albuminuria may be absent for weeks or months. (*g*) *Solids are decreased, i. e.*, the urea, uric acid, ammonium, chlorides, and phosphates. The determination of the freezing point, *cryoscopy*, was first employed by Korányi to estimate the amount of soluble molecules in the blood and urine. The blood, when there is deficient renal excretion, freezes at a lower point, because its molecular concentration is greater; this point is 0.56° C. (with 0.02° physiological variation in either direction), lower than that of distilled water. The urine freezes at a higher point than normal, as its molecular concentration is less. Normal urine freezes at 0.9° C., lower than distilled water; in chronic nephritis the urine freezes at 0.5 or even 0.2° below that of water. The smaller the molecules, the greater their influence in lowering the freezing point; the chloride molecules are more potent, therefore, than those of the urea group. Increased molecular concentration of the blood usually (not always) indicates disease of both kidneys. Cabot recently concluded that cryoscopy is of no great value in medical cases. (*h*) The *rapidity of elimination* is decreased. Methylene blue was employed by Achard and Castaigne (1897) to test this point. One cubic centimeter of a 5 per cent. aqueous solution is injected subcutaneously; normally it should stain the urine in one-half to one hour, reach a maximum in three to four hours, and cease in thirty-six to seventy-two hours; elimination may be nearly or even more rapid, in chronic parenchymatous, but it is much slower than normal in interstitial nephritis (Bard). The permeability of the kidneys may also be tested by a hypodermic injection of one cubic centimeter of a one-half per cent. solution of phloridzin (Achard), which produces glycosuria in normal kidneys; phloridzin glycosuria is reduced or absent in interstitial nephritis. A dilution test may be made. Drinking of large amounts of water raises the freezing point of the urine to 0.3, 0.2, or 0.1°, in interstitial nephritis and in health, but does not raise it in parenchymatous nephritis. (*i*) *The sediment is scant*, absent, or can be obtained only by the centrifuge. Hyaline, granular, and epithelial casts, are found in small numbers when the light is moderated by the diaphragm; the granular form is found in some cases only after repeated search; the granular casts are often wide, originating in the dilated tubules of the medulla. A few epithelia, leukocytes, and crystals of uric acid and oxalate of lime, are found. Red cells are infrequent.

The urinary findings are somewhat modified by intercurrent uremia, fever, and heart failure, all of which concentrate the urine, raise its specific gravity, and deepen its color.

2. **Cardiovascular symptoms** were noted by Bright, but their significance was first established by Traube. They appear in 50 to 90 per cent. of cases, and later than the urinary findings. *Cardiac hypertrophy* is more quickly detected clinically than pathologically, especially in the young, because the urine is increased early before the physical

signs of hypertrophy prevail, and again because concentric hypertrophy is present long before dilatation appears. Emphysema may obscure the precordial evidences of hypertrophy, such as the *heaving apex beat*, and one must therefore depend on the evidences presented by the *tense pulse*, the hardness of the arteries, the sphygmographic tracings with a square apex, the loud ringing second aortic tone, or other manifestations of increased arterial tension, as epistaxis, vertigo, headache, tinnitus, palpitation, or cardiac asthma. Strassburger, Sahli, and Fellner, estimate the normal systolic blood pressure at 125 mm. and the diastolic at 29 mm.; in interstitial nephritis the systolic pressure may mount to 200 mm. or more. High tension is one of the most constant and early symptoms; its early occurrence leads some writers to incorrectly regard contracted kidney rather as a disease of the arteries than of the kidneys. The cause of the hypertrophy is not definitely known; theories of mechanical obstruction to the blood-flow by the renal contraction, and of vasomotor constriction excited by retained waste substances, have been advanced. Though hypertrophy should be looked for in interstitial rather than in parenchymatous nephritis, it must never be forgotten that the two types often mingle with the urinary findings of the "large white kidney" and the cardiovascular findings of a renal contraction. In the last stage of contracted kidney the hypertrophied heart weakens, the pulse tension falls, the apex becomes weaker, a systolic murmur appears from myocardial insufficiency, and Potain's gallop-rhythm, a sign of heart fatigue, is heard.

3. **Retinitis** may be the first declaration of the disease, and may come under the observation of the ophthalmologist. It occurs more frequently than in any other renal lesion, 22.4 per cent. (Groenauw) to 28 per cent. (Eates). Its various forms are stellate white areas of fatty and fibrous tissue, a white zone surrounding the disk, degeneration of the disk, retina, or of both together, "flame-like" hemorrhages, and the choked disk whose large veins are compressed where the arteries cross them. (See Plate VI, Fig. 8.) The retinitis albuminurica is not specific, identical findings occurring in other conditions.

4. **Uræmia** is the most dreaded and frequent complication; its nature and immediate cause are unknown; it can only be stated that it is an auto-intoxication, due to disturbance of renal function, probably with retention within the blood of nitrogenous substances. Further explanations are wholly theoretical, such as decreased alkalinity of the blood (acidosis); failure of the normal "internal secretion" of the kidney; the toxic action of abnormal products of metabolism or breaking up of the proteids, resulting from destruction of the renal tissue (nephrolysins, Ascoli); it is not due to retention of urea alone, nor to mechanical conditions alone, as œdema or anæmia of the brain. It occurs most often in chronic interstitial, next in acute, then in chronic parenchymatous nephritis, and is much less common in other diseases of the urinary tract, as pyelonephritis, etc.

Uræmic *symptoms* may be acute or chronic, but will be considered as nervous, respiratory, cardiac, and digestive.

NERVOUS SYMPTOMS.—(i) Acute *convulsions* (eclampsia) are due to irritation of the cortex by toxins; they closely resemble the epileptic attack. There are usually prodromes, as pain or pressure in the head, migraine, neuralgia, insomnia, vertigo, visual disturbance, tinnitus, nausea, vomiting, or dyspnœa; there is usually no "epileptic cry." In less than a day clonic or tonic convulsions appear, usually generalized (sometimes Jacksonian) and involving especially the flexor or extensor muscles of the arms and legs; the abdomen is retracted; there are dyspnœa, cyanosis, and involuntary evacuations; the reflexes, especially the patellars, are increased; the pupils are wide and without reaction; the pulse is full and slow before the attack but becomes rapid during the convulsions. The skin becomes covered with sweat and the temperature is usually elevated. The convulsions cease after fifteen to thirty minutes and coma intervenes, during which the convulsions may recur. Death is the usual outcome, but recovery is possible, as in one of the author's cases in which the anterior third of the tongue was bitten off. (ii) *Other motor manifestations.* The most concise general statement regarding uræmic nervous phenomena is as follows: *paralysis* affects especially the sensorium, to a lesser degree the special senses, and rarely motility; *irritation* seldom affects the sensorium (*e.g.*, produces delirium), it principally acts upon the motor paths (convulsions) and very seldom upon the special senses. Uræmic convulsions are usually general but may resemble focal symptoms, such as *convulsions of only half the body,* for instance, of one-half the tongue, of the left arm and leg, or deviation of the head to the left. They may rarely occur with convergent strabismus, facial spasm, Jacksonian epilepsy, nystagmus, tremor, localized trembling and twitchings, muscular cramps, grinding of the teeth, tetanic convulsions, trismus, convulsions on one side and contractions on the other, rigidity of the neck or opisthotonos. In seven cases, seen by the author, the symptoms were those of an acute focal brain lesion; in two, which were treated by lumbar puncture, relief was immediate.

Paralyses in uræmia are infrequent, are chiefly transitory hemiplegias and are due to *cerebral œdema,* capillary hemorrhage, softening, apoplexy, inflammatory foci or cortical overstimulation by toxins. Hemiplegia may occur with aphasia, amnesia, deafness, crossed oculomotor paralysis, glossoplegia, or conjugate deviation of the eyes. The author has reported five cases in which uræmia resembled meningitis very closely. Other cases have been suggestive of brain tumor. (iii) *Acute coma* often occurs without antecedent eclampsia, being preceded by headache, stupor or apathy; coma may last for days or even months. (iv) *Psychical symptoms.* One of the most common chronic symptoms is a *characteristic restlessness and anxiety.* Obstinate *headache* of a dull, throbbing nature is common; it often begins in the early morning; it may be occipitial, or hemicranic. *Insomnia* is common, though during the day the patient is often sleepy. Delirium and mania are more frequent in chronic nephritis in adults than in juvenile cases; melancholia and delusional insanity occur; these mental states may assume medico-legal importance as to the testamentary capacity of the patient. (v) *Special senses.* Sudden, complete and bilateral blindness is cortical in origin; this *amaurosis* may be an initial symptom; there are usually no retinal findings and the condition lasts but a few hours, though in exceptional cases œdema of the disk (Schmidt), or even choked disk is found. The pupils are wide and without reaction in acute eclampsia, but are small and mobile in chronic uræmia. There may be sudden or gradual uræmic deafness and tinnitus aurium.

RESPIRATORY OR CARDIAC SYMPTOMS.—*Dyspnœa* is very common. It is sometimes toxic and central, occurring in close parallelism with cerebral manifestations. In other cases asthma uræmicum develops when the mind is absolutely clear, and it is then correctly referred to the heart. *Prodromal slowing* of the heart to 40 to 60 beats is frequent in incipient uræmia. The dyspnœa may be continuous or paroxysmal and is often nocturnal. Cheyne-Stokes breathing may develop in the day time or during sleep and is always an unfavorable sign, though recovery is possible. Obstinate hoarseness and a rare form of laryngeal spasm are recorded.

DIGESTIVE SYMPTOMS.—These rank second to the nervous symptoms in frequency. The breath is often offensive and sometimes has an odor of urine, which condition Senator refers to trimethylamine; he considers it of poor prognostic value, for it often precedes eclampsia. The tongue is foul, the mouth is inflamed and the saliva is frequently increased. *Nausea and vomiting* are partly central and partly local from decomposed urea, eliminated vicariously into the stomach; the same may be said of *hiccough* and *diarrhœa.* All these symptoms may be very acute. Diarrhœa may be serous or dysenteric from the Bac. dysenteriæ (Flexner). Mathieu and Roux in a recent study of 23 cases of "uræmic ulcers" found them chiefly in cases of advanced nephritis in subjects under twenty years old; they rarely occur in the stomach; diarrhœa is common, but bowel hemorrhage and constipation are rare. They are seemingly produced by necrosis due to toxæmia, though sometimes secondary to submucous hæmatomata. Healing is very rare and the prognosis is almost always unfavorable.

5. Other Complications and Symptoms.—(*a*) Various *hemorrhages* occur; of the brain in 7 to 17 per cent. of cases; also in the dura, skin, nose, bronchi, stomach, bowels, uterus, conjunctiva or retina. The author has observed 9 cases of profuse gastro-intestinal hemorrhages in uræmic subjects. (*b*) *Inflammations* may develop. Chronic bronchitis always suggests kidney disease. Pericarditis, pleuritis, and pneumonia especially, may develop as terminal infections. A large number of deaths from pneumonia is due to nephritis which was previously unnoticed. Laryngitis and acute (inflammatory) œdema glottidis may occur. (*c*) *Œdema* is a late symptom and results not from the nephritis unless it is of the mixed type, but from failing of the heart. Ascites also is nearly always cirrhotic or cardiac. (*d*) The *skin* is usually dry and harsh and is sometimes covered with urea crystals—the "urea frost" or uridrosis. Itching, erythema, urticaria, eczema and numbness or "dead fingers," unusual susceptibility to cold (cryæsthesia), neuritis, rheumatoid pains or clubbing of the fingers are probably toxæmic.

Diagnosis of Chronic Interstitial Nephritis.—There are *three cardinal findings, cardiovascular, urinary* and *retinal;* and as Senator says, he who invariably examines the urine and heart in every instance rarely fails to diagnosticate nephritis correctly.

Cardiac Symptoms.—The very interdependence of cardiac and renal changes may prove to be a source of clinical confusion; thus primary cardiac disease may cause renal congestion, embolism or even acute or chronic nephritis; again alcohol or syphilis may be a common cause of arteriosclerosis, myocarditis and nephritis; and finally, the heart lesion may be wholly secondary to renal disease. Simple renal stasis is considered in the differential table given below. In terminal cardiac weakness caused by contracted kidney, the hydrops, dyspnœa, rales or systolic murmur may suggest myocarditis with renal stasis; gallop-rhythm is more common in the heart of renal disease than in primary cardiac affections.

The Urinary Findings.—In *diabetes mellitus* there is an increased flow of urine, in which the high specific gravity, glycosuria and less prominent heart findings are distinctive; in *diabetes insipidus*, the absence of albumin, casts and cardiac changes are characteristic; and *pyelitis* is characterized by albumin and pyuria but not by casts or cardiac changes. In the *arteriosclerotic kidney*, arterial and cardiac antedate the urinary changes (the converse is true in interstitial nephritis), the specific gravity is higher, albumin less in amount and in frequency of occurrence, and the course is much more rapid. For differentiation from other DIFFUSE RENAL DISEASES SEE TABLE, pages 796–797.

Diagnosis of Uræmia.—Skull-fracture, miliary tuberculosis, leptomeningitis, cerebral abscess, sepsis and other diseases may be falsely diagnosticated uræmia, simply because nephritis is also present. Senator has said that without a previous history we can often only diagnosticate a renal disease and suspect the presence of other (latent) affections. The danger of mistaking uræmic symptoms, as hemicrania, nervous symptoms, dysentery etc., for independent disease is greater in direct ratio to the difficulty of diagnosis of the nephritis itself and many authors have remarked on the lack of correspondence between the clinical and pathological findings in nephritis. According to Leube, uræmic intoxication is assumed to be the cause of nervous symptoms only when other causes may be excluded, because in the course of renal disease, organic nervous lesions, as hemorrhage and meningitis, may simulate uræmia. Though the urine is usually decreased before an attack of uræmia, Liebermeister instances a case in which, immediately before a uræmic seizure, both urine and urea were increased two-fold, hours before uræmia developed. Bierme reports an anuria which lasted two hundred and twenty-two hours before uræmia developed.

Apoplexy is probably present when pressure symptoms, as vomiting, or slow, tense pulse, complete hemiplegia, or conjugate deviation, exist; it is rarely completely simulated by uræmia. Meningitis, insolation, or the coma resulting from alcohol or opium are rarely simulated; in very exceptional instances focal symptoms and fever may cause diagnostic error. The author has seen a few cases in which uræmia simulated *typhoid or sepsis* by the constant high temperature, though the autopsy disclosed nephritis, and two cases in which there was no bacteriological evidence of secondary or terminal infection.

Prognosis of Interstitial Nephritis.—Because the disease is insidious in onset, it is too often detected only when the patient suffers from cardiac insufficiency, beginning uræmia, apoplexy or intercurrent inflammations; for this reason this incurable malady is rather seldom treated in its early stages. The clinical course usually runs five to ten years after albuminuria is found; a longer duration is not uncommon; a course of twenty years is reported by Senator and Aufrecht, of twenty-three years by Oppolzer and of thirty by Rosenstein. Much depends on the docility and financial ability of the patient. Marked arteriosclerosis naturally hastens the fatal issue. *Retinitis* is usually an ominous symptom; in Belt's 419 cases, 72 per cent. died in one, and 90 per cent. in two years. Retinitis increases the gravity of the outlook four-fold. Staub's and Gruening's figures are almost identical. Webster, Wert, Hare, Mix, and the author have seen cases which lived from four to ten years after retinitis was found. Uræmia, pneumonia, apoplexy, and other complications are decidedly unfavorable.

Treatment of Chronic Nephritis, Parenchymatous and Interstitial.—Both forms are considered together, because they often blend (mixed nephritis).

1. **Prophylaxis.**—Prevention and treatment of the cause have a very limited practical application. Irritating foods, excessive eating, all forms of intemperance, over-work, neglect of infections, and dyspepsia come under this head, but are usually treated too late.

2. **Rest, Exercise.**—The kidneys should be relieved by rest in the horizontal posture, because exertion and even the erect position, which *per se* may cause albuminuria, only increase preëxisting albuminuria. If in severe cases exercise seems advisable, it should be given passively with the patient in the recumbent posture. The rest should be absolute as long as the heart is weak, the urine scanty, or hydrops conspicuous. The importance of rest in bed was recognized by Bright and Bartels. When there is moderate renal involvement, or in the intervals of improvement, relative restriction of exercise should be enforced. Rest in bed lessens the amount of waste products to be eliminated by the kidneys and so protects the skin and dilates its vessels that the cutaneous functions become more active and relieve the vascular

hypertension, just as a patient with a cold should remain in bed, in order to increase the activity of the skin and promote that of the kidneys. The author has treated two severe cases of parenchymatous nephritis by rest in bed for a year, and absolute recovery resulted. It is a question if lying covered in bed is not one of the reasons why nephritics pass more urine at night than in the day time. Fatigue, dampness, and cold must be avoided; these indications are best carried out by rest in bed during the time of severe symptoms or of acute exacerbations, and by relative rest and warm clothing in cases of moderate severity. Practitioners do not sufficiently insist on psychical rest; in men leading a strenuous life, moderate nephritis has often decreased and sometimes disappeared when nervous tension was relieved; too often these subjects take physical exercise or courses of training after they are already exhausted by head-work; in any event exercise should supplant work and not be taken after hard nervous strain.

3. Springs, Climate.—"Cures" at water resorts have the general advantage that the importance of diet and hygiene is often much impressed on the patient; the psychical or suggestive element is also an important therapeutic agent. Diffuse nephritis (with considerable parenchymatous and interstitial change) is most benefited. Only patients without œdema or serious heart involvement should be sent to such places as Poland Springs, Bedford Springs, Saratoga, Vichy, or Neuheim. A stay in the California, Mississippi, or North Carolina climate during the winter at least will materially benefit.

4. Diet.—The diet must be non-irritative, must not especially tax the kidneys in its elimination, must not burden the circulation, and must maintain nutrition. Disease of the glomeruli alone may be compensated by vicarious excretion of water through the lungs, skin, and intestines, but disease of the tubules whose function is elimination of the end-products of albuminoid metabolism, cannot well be compensated by other structures, whence the indication is for *limitation of albuminous foods* and their replacement by fats and carbohydrates. The amount of albumin lost through the kidneys is small and can easily be offset by a glass or two of milk daily; in this respect nephritis differs from diabetes in which the system is irritated by unappropriated sugar and loses a vast number of units of heat and energy each day. Albuminous food is restricted only to spare irritation of the excreting renal cells, and no absolute rule can be laid down for all cases. Chittenden has recently shown that we eat twice as much meat as we need. It is an obvious mistake to guage the severity of a nephritis by the degree of albuminuria, or to estimate improvement by the influence of diet upon the albuminuria alone. Fats as butter, olive and cod-liver oils, and cream, are very important, because they supply energy and heat units in place of the restricted albumin. As in diabetes, the restriction of diet must not merely concern renal irritation, but also, and most of all, the maintenance of nutrition, especially when the patient suffers from parenchymatous nephritis, very form in which restriction of diet is theoretically most needed; emia and malnutrition are the most distinguishing features of the enchymatous as contrasted with the interstitial type of nephritis. e individual patient must be considered more than the disease *per se* he type of the disease.

The *milk diet* is free of extractives, is easy of digestion, and is a diu- c. It has its restrictions; it is bulky, over four quarts being neces- y to maintain nutrition, and it therefore may cause dyspepsia; acks iron, but this can be easily compensated; four quarts contain excess of about 60 per cent. of proteid; it contains too much water, reby possibly overloading the blood and straining the heart; and phosphates and chlorides tax the kidneys. One to one and a half rts of milk with a pint of cream, fats and carbohydrates (baby foods, o, rice, or potatoes), will meet the needs of metabolism; and, some- es over half of the phosphates of the milk can be precipitated and t in the intestine (and away from the blood and kidneys) by giving cium carbonate gr. v to x with the milk (von Noorden). It should taken slowly, in sips, and it should be remembered that it is a food d not a beverage. Milk was one of the chief articles of diet of the er masters in medicine; a very short time ago we heard that it tained an excess of water; now we are told it is excellent because contains so little salt. Part-truths are misleading.

In regard to a *meat diet*, there is much liberality at present, though bably the reaction in favor of the meat diet is extreme, as Strubell's ent experiments demonstrate that a meat diet promotes uræmia. e average daily amount allowed by von Noorden is three ounces 0 gm.). There is absolutely no difference between red and white ats, as to extractives, or purin bodies (Walker Hall, Kaufmann, ohr and others). The eating of some meat is preferable to over- ding the stomach and bowels with a bulky vegetable diet, save en toxæmia (uræmia) is imminent.

Raw *eggs* increase (perhaps induce) albuminuria; not more than o daily should be given. *Substances excreted with difficulty* include e following, according to von Noorden; urea (in meat), creatinin meat broths, extractives and to a less extent in eggs), urinary gments (in hæmoglobin), hippuric acid (in cranberries, fruits with s, prunes, plums and gages), phosphates (in milk) and inorganic lphates (in meat); uric acid and alloxur bodies, contrary to general inion, are, according to von Noorden, eliminated readily; (they are nd in glandular organs,—such as the thymus, sweetbreads, liver d kidney,—in broths, and according to Taylor, in coffee). The *lorides* are especially to be avoided in parenchymatous nephritis th œdema. As pointed out by Widal, Lemerre, Javal, Mercklen d Courmont in 1903, administration of salt (which is retained in blood) increases the body-weight, induces œdema and aggravates uminuria, while "dechlorination" of the food reduces weight, œdema, d albuminuria. Probably the entire truth is not yet known; Rumpf 05) found cases with œdema, uræmia and retinitis in which the chlorides of the blood were decreased. *Irritants,* as cheese, relishes, horseradish, onions, rhubarb, asparagus, spices, sauces, broths, beef-tea and alcoholics should be absolutely avoided; tea and coffee must be taken with great moderation because they may aggravate or produce nephritis (Penzoldt, Hubach).

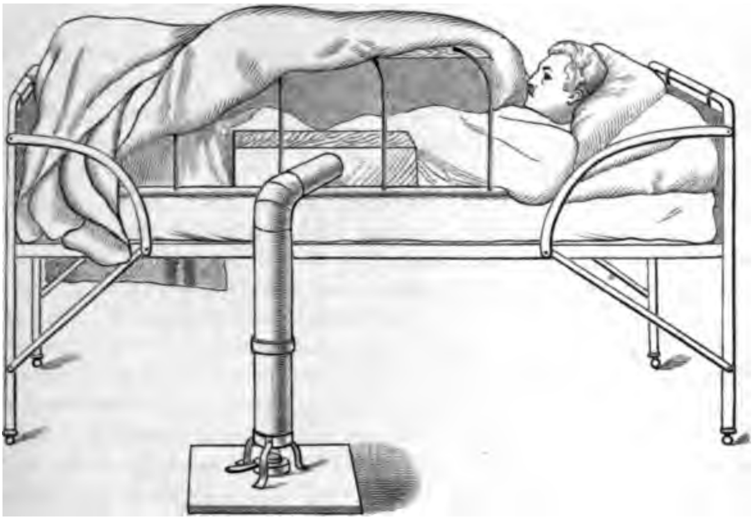
5. Water.—In regard to giving water, the same extremes of opinion are noted; fluids were once excessively restricted, but more lately undue quantities were recommended; von Noorden states that flooding the vascular system with water overtaxes and damages the heart; "water can only be excreted by the kidney if the blood pressure is increased; it is difficult to demonstrate that the heart is damaged by excessive water drinking, but indirectly this can be shown, for many patients having weak hearts give a history of excessive water drinking for long periods of time; in such cases improvement is frequent if the ingestion of water is limited; about three pints (1,500 c.c.) should be the average amount." Nevertheless relief of high arterial pressure and minor uræmic symptoms may follow copious drinking of water.

The following therapeutic measures are but symptomatic.

6. Diuretics.—These are generally useless and should be, for the most part, avoided. Diuretin is said to produce no renal hyperæmia but to act directly on the epithelium and, according to Gruenwald, to sometimes reduce the albuminuria; it may be employed and sometimes with success (*v.* TREATMENT OF VALVULAR DISEASE). The use of diuretics is indicated by increase of hydrops, scanty urine, and imminent uræmia. The refrigerants (cream of tartar, ʒj) or potassium citrate (gr. xx to xxx) are the only safe diuretics; they are thought to abstract water from the tissues and to convert urea, uric acid, and similar products into more readily excreted substances; they are apparently most active in the effervescent form. Basham's mixture (liq. ferri et ammonii acetatis) as a tonic and diuretic should be given in doses of but 1 or 2 drams. Diuretics which act through the circulation will be considered below.

7. Hydrops.—Hydrops necessitates treatment because it prevents movement, threatens suffocation by pulmonary or laryngeal œdema, increases the danger of secondary infections, as erysipelas or cellulitis, embarrasses the circulation by pressure on the afferent capillaries and efferent lymphatics and venules, interferes with digestion, and increases the danger of uræmia. (*a*) Though *cathartics* may eliminate fluids as well as solids, drastics must be given with due care for the digestion and nutrition, pulv. jalapæ co. ʒss to ʒj at night, or Epsom salts ℥ss to j in the morning. (*b*) *Sweats* are generally given, possibly less so of late. Though the functional relation between the skin and kidneys cannot be overestimated, profuse sweating eliminates fluid rather than solids. Leube holds that sweats concentrate the blood and predispose to uræmia; he states that when a patient sweats one quart (1,000 c.c.) only 7 to 15 grains (0.5 to 1 gm.) of urea are evacuated. The author has more often seen aggravation of symptoms, or a fatal issue result from sweating, than relief and hence prefers the old-fashioned full warm bath, recommended by Osborne and Liebermeister, to the more active methods. In this form of treatment an ice-bag is placed on the head to prevent brain congestion. Commencing with water at body temperature, this is raised five or six degrees. After twenty or thirty minutes the patient is removed and wrapped—without drying—in blankets. A hot-air bath may be given by placing a lamp under one end of a stove-pipe which runs by an elbow into a tent built over the patient, as illustrated in the following cut. Another method is the alcohol sweat, in which hot bricks protected by flannel are placed around the heavily covered patient; equal parts of alcohol and water are poured on the bricks and the patient is covered again; the heat produces a steam vapor which is tolerated fairly well unless the heart is weak, when fatal collapse may ensue. It is often unsafe to give pilocarpine (*v.* ACUTE NEPHRITIS) because it produces profuse salivation, vomiting, diarrhœa, syncope, and pulmonary œdema. (*c*) Mechanical relief by means of *incision* is superior to all other measures which must first bring the fluid with all its urea and extractives into the circulation, with the ever present danger of inducing uræmia; *i.e.*, the already impure blood is again contaminated by the toxic deposits in the œdematous tissues. If the kidneys and other emunctories cannot relieve the blood, how can they remove the additional refuse in the anasarcous fluid, which maybe amounts to quarts, and comes to the blood from the tissues? The author has experimented with mild cases, making deep incisions, three inches long, over the lower leg (not over the malleoli where infection from the feet is likely) whenever the hydrops is at all extensive. In testing the fluid evacuated,

FIG. 47.



Sweat bath employed in nephritis.

there has frequently been 0.1 to 0.2 per cent. urea which may serve as a measure for other retained products of kindred nature; when quarts seep out during the first twenty-four hours, one is well justified in incising the legs, despite the slight danger of occasional infection. Sweats and drugs never achieve equal results. Free incision is obviously less dangerous than punctures or Southey's capillary tubes.

8. The Circulation.—Treatment often resolves itself into the care and stimulation of the heart. Mental excitement, nervous strain, physical exertion, constipation, and tobacco, coffee, alcohol, and exposure to heat must be avoided. The more the heart hypertrophies the better is the outlook, although ultimate cardiac dilatation is the inevitable result of every hypertrophy. In the uræmic seizures, with precisely the same symptoms in other respects, the heart may vary; it may beat as strongly as in an apoplexy, or very weakly. (*a*) *With cardiac over-action* observed early in uræmia, the angiospasm or the high tension must be corrected by *vasodilators*. Iodides are beneficial when there is no urgency; they should be given in doses of grs. v to x t. i. d.; iodism, and especially salivation, may result from renal retention. When extreme tension and imminent uræmia exist, tr. veratri viridi ♏v to x every half hour for three or four doses may be given (though the author considers that the drug is injurious) or large doses of nitroglycerine, gr. $\frac{1}{50}$, until results are produced. The author believes that the vasodilators are often given to excess, as hypertrophy and increased arterial tension are less pathological than compensatory conditions. *Bleeding* may so relieve the intracranial blood pressure as to remove for a while the uræmia, but too often it does not; salines may be given afterward, either subcutaneously or by rectum; they serve as a "lavage of the blood" and stimulate the kidneys if enough sound tissue be left to act upon. Increased intracranial tension may be relieved by *lumbar puncture;* the normal pressure of 120 mm. (as indicated by the sphygmanometer) is often increased, sometimes to five or six hundred mm.; during the last year the author has seen marked and sometimes protracted relief in medical and eclamptic cases; the headache, vomiting, convulsions, and coma are often decreased or entirely relieved. (*b*) When the *heart is weak*, rapid, irregular, or galloping, cardiac stimulation by digitalis and strychnine is indicated. Infusion of digitalis by mouth may cause vomiting and ♏ij to v of the fluid extract may then be given hypodermatically or by rectum. Its combination with squills or calomel is inadvisable; the latter may cause the most intense salivation; in one case necrosis of the entire tongue occurred. Cardiac unrest, palpitation or dyspnœa should be relieved by a hypodermic of morphine, by application of *ice to the heart,* or by champagne, nitroglycerine, bromides, and valerian. Venesection lessens the molecular concentration of the blood (*i.e.*, its toxicity) and relieves the laboring heart; infusions act in the same way; an excess of salt may be given (*v.s.* "dechlorination") in the infusions; when they are repeated, the salt should be left out, for it may produce œdema of the lungs. Pilocarpin and sweats are distinctly contra-indicated, because they over-tax or fatally depress the heart. Von Ziemssen gave champagne and digitalis by mouth as long as the patient could swallow, and then digitalis by rectum or hypodermatically and camphor subcutaneously.

9. **Vomiting.**—Vomiting and diarrhœa are as often vicarious as toxæmic and should not be checked at once. Gastric lavage and colonic flushings are beneficial.

10. **Convulsions.**—For convulsions, hypodermics of morphine should be given, as advised by Loomis and Mackenzie; the author never saw harm result from its use, though medical opinion is generally against it. Chloral (gr. xv every hour for not more than four doses), aids the morphine, while bromides generally act too slowly.

11. **Operation.**—Surgical treatment is still *sub judice.* It is certain that many cases of "decapsulated" kidney were not genuine nephritis, but rather albuminuria from floating kidney, or ascending infection. Again, it is still a question whether chronic nephritis is merely a renal disease or perhaps a result of a general condition of the blood; this idea was advanced by Semmola, and is not yet disproven.

(C). PASSIVE CONGESTION—INFARCTION.

1. Passive Congestion.

Venous hyperæmia is an important renal lesion, contrasting with arterial hyperæmia, of which latter little is definitely known, though it occurs in renal inflammation, left ventricle hypertrophy, diabetes, perhaps also in nervous lesions and after the administration of diuretics.

Etiology.—(*a*) Among the chief causes of *general stasis* are cardiac insufficiency from valvular disease, myocarditis, and pulmonary emphysema. (*b*) *Local stasis* is much less important, and has scarcely any part in renal pathology (Cohnheim). Berkmann (1859) described renal stasis and thrombosis of the renal veins occurring in the newborn or in nurslings after severe diarrhœa; when unilateral, there is renal pain, great albuminuria, some hæmaturia, swelling of the kidney and decreased urine; most cases of bilateral thrombosis die from anuria and uræmia. Thrombosis of the cava, and pressure by large tumors may cause passive congestion in the renal veins.

Pathology.—Anatomical changes result from slowing of the arterial flow, lessened arterial pressure, and to a less degree from venous hyperæmia, which changes alter the integrity of the secreting renal structures, notably the glomeruli. In experiments, arterial insufficiency causes initial glomerular alteration, while venous obstruction causes primary congestion in the medullary portion.

Early Changes.—The kidney is swollen, dark red and firm, and its stellate veins are prominent. On section it expands beyond its close fitting capsule, which is readily stripped off. The organ is very vascular and may drip blood. The glomeruli are seen as dark red points and the medullary bloodvessels are clearly defined. Microscopical the conspicuous changes are hyperæmia, occasionally with puncts hemorrhages, fatty and cloudy degeneration of the epithelium of t tufts and convoluted tubules, escape of albumin into them, formi hyaline casts in the tubules, and finally some thickening of the vesse

Late Changes.—Prolonged stasis leads to greater atrophy of t secreting structures and to increase of connective tissue; this "cyano induration" has been rather overdrawn in clinical descriptions, b may result from extreme and protracted stasis. The kidney becom pale and firm, the cortex shrinks, the capsule may become somewh adherent, and connective tissue develops in the medulla. Micr scopically, fibrillar connective tissue, thickening of Bowman's capsu and epithelial degeneration are found.

Symptoms.—Aside from the causal heart or other lesion, and t signs of stasis in other parts, the urinary findings are of chief intere The urine is decreased, and its amount follows directly the hear activity. It is acid, dark, concentrated (specific gravity 1,025 to 1,03 and it has a lateritious sediment of urates and often of uric acid cry tals. Albumin and globulin are present, usually in moderate amount Urobilin may be present. Microscopically a few leukocytes, epitheli cells and hyaline casts are found; granular casts are seldom fou and should arouse suspicion of nephritis; red cells are usually co sidered common, but Leube insists that they almost always indica nephritis or infarction.

Diagnosis.—The urinary findings are significant in association wi the causal lesion; œdema begins in the legs; the nutmeg liver, dyspnœ cyanosis and pulmonary stasis are present. The percentage of album runs parallel with the strength of the heart, increasing with its failu or decreasing if digitalis or strychnine improve its action. If cardiac increase the urine, and if abundant albumin and casts still remai there is probably coincident nephritis. (See TABLE pages 796 and 79

The *treatment* is that of chronic valvular disease (*q.v.*).

2. Infarction—Embolism.

Though infarction may rarely occur from local disease of the re arteries, acute or chronic valvular disease of the left heart is its p disposing cause. Sixty-six to 75 per cent. of all embolisms are ren Stoppage of a renal terminal artery causes the well known wedg shaped anæmic infarct in twenty-four to thirty-six hours; the lo ischæmia causes fatty degeneration, necrosis, and a finely granular tra sudation into the connective tissue and glomeruli. After a while fibro substitution occurs with ultimate shrinkage and deformity. Af repeated infarctions, the irregular "embolic contracted kidney" is se As in other infarctions, the process may be innocent (mechanical) less commonly suppurative.

Symptoms.—In most cases there are no symptoms. Exceptionally a diagnosis is possible, based on (1) the valvular lesion; (2) sudden onset with fever; chills and vomiting; (3) lumbar pain and tenderness, and (4) hæmaturia and albuminuria.

There is no *treatment.*

(D). AMYLOID DEGENERATION OF THE KIDNEY.

Etiology.—The etiology of amyloid kidney is that of amyloid liver (*q. v.*); its three principal causes are *ulcerative tuberculosis, protracted suppuration* and *inveterate syphilis.* The most plausible theory is that toxins produce, either in the blood or in the organ involved, an albumin which is deposited in and about the bloodvessels. Amyloid kidney was first described by Rokitansky (1842) and its symptomatology was developed by Wilkes, Todd and Traube.

Pathology.—Amyloidosis seldom occurs in the kidneys without associated changes in the liver and spleen. To the naked eye the kidneys generally appear *enlarged,* weighing together even as much as 26 ounces (870 gm., Johnson); when amyloid degeneration or infiltration is slight, there may be no alteration in size or weight; in some cases the kidneys are large from coincident chronic parenchymatous or small from interstitial nephritis. The capsule strips readily, showing a pale, smooth surface. On section the *cortex is enlarged,* glistening, waxy, and pale and its *edge translucent;* the *glomeruli stand out* as *glistening points,* and become brown when treated with Lugol's solution. Microscopically the amyloid substance can be seen clearly; it is so named because of its starch-like reaction, though chemically it is an albuminoid. It is either deposited from the blood in the small vessels, or more probably formed *in situ,* whence it is found especially in the glomeruli, then in their afferent vessels and in the straight vessels of the medulla. The renal interstitium is involved secondarily, and the epithelial cells never suffer directly, though the *membrana propria* may be affected and the cells may suffer secondary compression, degeneration and atrophy. As the changes indicate, amyloid disease cannot be classified with Bright's disease, though their association (*v. i.*) is not uncommon.

Symptoms.—Its symptoms are variable. (*a*) *Urinary symptoms* are exceptionally absent (Rosenstein and Litten). The urine is generally *limpid and increased,* but may be decreased when amyloid degeneration is associated with parenchymatous, or greatly increased when associated with interstitial, nephritis; some writers place rather undue importance upon variability in its amount. The specific gravity is 1,010 to 1,015. *Albuminuria* is *usual,* being absent only in those exceptional cases which invade the vasa recta and avoid the glomerular capillaries; albuminuria results from increased permeability of the glomerular vessels and may amount to 1 or 2 per cent.; Senator has remarked that there is frequently a large amount of globulin in the urine. There is little or no change in the solids as a result of the amyloid itself; indicanuria is not infrequent. Microscopically hyaline casts may be present and perhaps a few leukocytes and epithelia but the sediment is scanty in most cases; the so-called waxy or am loid casts found in severe nephritides have no relation to amyloido of the kidney. (*b*) Cardiac hypertrophy, uræmia and retinitis do n occur in renal amyloidosis, except when it is associated with nephrit (*c*) Amyloid degeneration of both the *spleen and liver* and sometim also of the alimentary tract are associated in 66 per cent. of case these and the casual disease are necessary for diagnosis. (*d*) *Œdem* and *anæmia* which is almost an actual cachexia, are usual in cases diffuse developed amyloidosis. Hydrops of the serous sacs, especial *ascites*, is quite common though less so than œdema, and usual occurs as a late or terminal event. *Differentiation* (*v.* Table on pag 796 and 797.)

Prognosis.—Amyloid disease is generally incurable, though stated under amyloid liver, light grades may recede. Amyloid of t kidneys in most cases runs its course in a year or less, particula when associated with parenchymatous nephritis. Cases may endu for several, even fifteen years, but these are generally complicated contracted kidney.

Treatment.—Treatment consists chiefly of prophylaxis; with mod ern surgical treatment, amyloid disease is less common than formerly Early treatment of tuberculosis and syphilis decreases the percentag of amyloidosis; for the developed disease there is no treatment.

(E). MALFORMATIONS OF THE KIDNEY.

Malformations and Structural Anomalies of the Kidneys.

To malformations and structural anomalies in general, but little clinical importance is attached. Complete absence or rudimentary development of both kidneys occurs only in unviable infants.

One kidney, usually the left, may be *absent or rudimentary*, whereon the normal kidney vicariously hypertrophies; the enlargement may be mistaken for malignant disease, whence its removal is fatal. Before operation it is imperative, at least in doubtful cases, that cystoscopy be employed to determine the presence of two ureters or that a preliminary incision be made to ascertain that there are two kidney Two ureters may come from one kidney. Ballowitz collected 1 cases in which one kidney was lacking; 61 per cent. occurred in mal and in 63 per cent. the left kidney was lacking. According to Gérar unilateral absence of one kidney is usually associated with unilater malformation of the internal genitalia but this is not true in unilater renal atrophy nor in cases where two kidneys fuse.

Supernumerary kidneys, double kidney, deformation from lacing, scoliosis, are of pathological interest only; 15 cases of supernumerar kidneys are recorded (Graser) and in two cases there were four kidneys.

The "*horse-shoe*" kidney (*ren arcuatus*) due to the fusion of both kidneys, is fairly common. Errors in diagnosis are common especially if the kidney is dislocated, and are inevitable if the renal pelvis is inflamed or dilated. Cases are reported in which the *ren arcuatus* has been mistaken for a tumor and extirpated, obviously with fatal results.

Movable Kidney.

Congenital dislocation occurs twice as frequently in men as in women, and is usually left-sided, in which respects it differs from the acquired form. It frequently coincides with congenital anomalies of the genitalia and intestines, and is of more anatomical than clinical interest. The kidneys may lie one upon the other (dystopia renum cruciata), of which Chatelin could collect but 15 cases. The kidney may lapse into the pelvis. The ureters are sometimes quite short.

Acquired dislocation, described by Rayer nearly a century ago, is much more common and important. It is variously called movable or floating kidney, ren mobilis seu migrans, ectopia renis acquisita, nephroptosis.

Etiology.—(*a*) *Sex;* 88 per cent. occurs in *women*, as shown in Kuttner's collection of 667 cases; Dietl claimed that women constituted 99 per cent. of the cases. (*b*) *Age.* Most cases occur between the twentieth and fiftieth years. Comby reports 18 and Abt 5 cases in children. (*c*) The *actual cause* is disputed. The kidney is supported by the fat around it, the peritoneum in front of it and by its own bloodvessels. Among the alleged causes are; atrophy of the perirenal fat; the presence of a *mesonephron* (Gérard) surrounding the kidney to a variable extent; *weakening of the abdominal walls* by pregnancy and other causes; alterations in the intra-abdominal pressure by *malpositions of the uterus, e.g.*, prolapse (Landau); pressure exerted by *corsets* on the liver (Cruveilhier); *congenital laxness of the supporting structures*, in connection with which Stiller claims that the tenth rib is usually "floating" like the eleventh and twelfth ribs (costa fluctuans decima); tumors of the kidney, which drag it downward; trauma or sudden efforts, as in lifting. Several of these causes are also thought to produce enteroptosis, in connection with which floating kidney is very frequently observed.

Symptoms.—Symptoms are absent in many, probably in most cases; they may date from the time when the physician incautiously remarks upon the presence of "a floating kidney." There are *two main symptoms*, the subjective pain and the objective tumor.

Pain.—Pain may be merely a dragging sensation or may amount to severe renal colic, which is said to result from stretching of the renal nerves; it may be lumbar, abdominal or may shoot toward the heart, thigh or sciatic region; it may occasion intercostal neuralgia, even on

DIFFERENTIAL TABLE FOR THE DIAGNOSIS OF THE DIFFUSE DISEASES OF THE KIDNEYS. (MODIFIED FROM LEUBE.)

		Passive Congestion.	Acute Nephritis.	Chronic Nephritis.			Amyloid Kidney.
				Chronic Parenchymatous Nephritis.	Secondary Contracted Kidney.	Genuine Chronic Interstitial Nephritis.	
Etiology.		General stasis (heart, liver, kidney) or local stasis (thrombosis renal veins, cava, etc.).	1. Cold, exposure. 2. Poisons, *e.g.* cantharides. 3. Acute infections, scarlatina, sepsis, pneumonia.	1. From acute nephritis. 2. Cold and moisture. 3. Toxic. 4. Toxæmic, phthisis, malaria. 5. Heart disease.	Secondary contraction may follow, but even then the kidney remains large.	Gout, alcoholism, diabetes, plumbism, syphilis, arteriosclerosis.	1. Protracted suppuration. 2. Old tuberculosis. 3. Inveterate syphilis.
Pathology.		Kidney, larger (may shrink), cyanotic, fatty, cloudy change, infarcts.	Some swelling, exudation of cells and serum; glomerular, epithelial, and interstitial changes.	1. Large, white kidney. 2. Large, red (variegated) kidney. Fatty changes in tubules. Some connective change, especially in 2.	Contractions less than in genuine interstitial form, less cysts, larger prominences, larger granulations, and more yellow, and smooth.	Very small, atrophied, indurated kidney with fibrous hyperplasia, epithelial atrophy, adherent capsule, small granulations, etc.	Large, homogeneous waxy, vascular infiltration.
Urine	*Quantity.*	Scanty: varying with heart's activity, whence prognosis from amount, and from action of digitalis.	Decreased (glomerular changes), suppression even.	Decreased (tubular and glomerular change): 200 to 1,000 c.c.	Somewhat increased, 1,500 to 2,000 c.c.: at least more than normal.	Very abundant, polyuria, pollakiuria usually 2–3,000 c.c. (rarely 4–5,000).	Normal or increased; variable.
	Color.	Dark red.	Turbid (urates or blood)—pale red to dark red.	Opaque: "meat water" appearance; urates suspended in albuminous urine.	Moderately clear.	Pale, clear.	Clear yellow.
	Sp. Gr.	High: 1,025–30.	High: 1,025–30–40.	Normal or somewhat higher, 1,020.	Somewhat under normal.	Low, 1,010 being the average.	Normal or less.
	Albumin.	Due to arterial anæmia, moderate, varying with condition of heart.	Much: ½ to 1 per cent.	Much: 1½ to 3 per cent. (very rarely absent).	Considerable.	0.5 per mille, slight, intermittent latent or long absent.	Usually abundant.
	Sediment.	Moderate, urates, hyaline casts, few red cells.	Great: much urates, red and white cells, blood, epithelial, waxy casts.	Great: red and white cells; casts, all kinds, [illegible] and fatty, especially fatty cells.	Moderate—many casts of all types.	Scanty: few hyaline (granular) casts.	Usually none, unless co-incident nephritis

					phates.	
Urine Continued — *Blood.*	Absent, save for stray red cells; blood from infarct.	Much.	Usually some: more in large red kidney (chronic hemorrhagic nephritis).	Little or none.	Absent.	None.
Heart Hypertrophy.	Changes depending on valvular lesion, etc., present.	None as rule (very rarely early hypertrophy).	Occasional, eccentric hypertrophy; especially in diffuse nephritis.	Usually present.	Almost invariable (concentric hypertrophy).	None in pure cases.
Hydrops.	From stasis; stationary, mechanical, in feet first then abdomen, pleuræ, etc.	Usually marked, rarely absent; mostly in scarlatinal and exposure nephritis; from vessel changes.	Usually extreme and obstinate (in skin, cavities, and viscera); marked vascular change; somewhat less in large red kidney.	Moderate; less than in large white kidney but more than in true contracted kidney.	Absent, unless in stage of cardiac incompetence.	Marked, from cachexia or vessel infiltration.
Anæmia.	Often present when is usually associated with cyanosis.	May be some.	*Very* frequent; a cardinal symptom with the urinary findings and hydrops.	Moderate.	Usually none (except in young subjects).	Marked.
Uræmia.	Absent.	Frequent; especially when due to cold and scarlatina.	Moderately frequent.	Frequent.	Most frequent.	Never.
Visceral Symptoms.	Lung congestion, stasis liver, infarcts spleen, etc., dyspnœa.	Pulse somewhat tense; often latent; fever due to other causes.	Anæmia; retinitis; bronchitis. Fairly frequent.	Retinitis, bronchitis, etc.	Retinitis and arteriosclerosis more than in any other forms.	No retinitis. Amyloid degeneration of liver, spleen, and alimentary tract.
Course.	Depends on age and extent of lesion; may be long.	Average duration 1½ weeks; usually wholly disappears if patient lives; sometimes becomes chronic (scarlatina).	½ to 1½ years (may be longer in chr. hemorrhagic nephritis).	Slower than in large white kidney, but faster than in the genuine contracted kidney; 1½ to 3 years.	Much longer than in other nephritides, 5 to 10 years.	Depends on causal disease.
Cause of Death.	Cardiac dilatation, embolism, coronary disease.	Death from causal disease; uræmia, hydrops (glottis) œdema of lungs, inflammation (pericardium, pleura, lung, etc.).	Uræmia, œdema glottis, hemorrhages, marasmus, sepsis, acute exacerbations, but mostly (visceral) inflammations.	Uræmia, cardiac insufficiency, myocarditis, hemorrhage (cerebral), retinitis, inflammations (pleural, pericardial, pneumonic, cutaneous) very common.		Causal disease, hydrops, cachexia.

the opposite side of the body. Pain may be constant or intermittent, mild or severe or like a crisis (*v. i.*).

Objective Kidney Findings.—On bimanual examination with one hand over the kidney in front and the other pushing it up from behind, the findings vary; (*a*) frequently the *kidney* is not painful nor tender, but *palpable*, which condition has no significance especially in women; (*b*) the kidney may be *movable*, descending on deep inspiration, maybe to the navel, where it can be retained by pressure during expiration; movable kidney is found in 20 per cent. of women (Tuffier); (*c*) the kidney may be *floating* (the most extreme type), in which it may fall or wander to the lower abdomen or pelvis. In 76 *per cent. of cases the right kidney* is involved, in 11 per cent. the left, and in 13 per cent. both kidneys (Kuttner); the right kidney has longer vessels, and the left renal vein is more firmly connected with the suprarenal vein and the pancreas. When the kidney is grasped a sickening sensation sometimes results as when the testicle is compressed. The *tumor is smooth* and is usually recognized by its shape. In rare cases the pulsating renal vessels are felt (v. Frerichs and Eichhorst). If not clearly felt when the patient is lying on the back, palpation is made in the standing posture or with the patient lying on the left side. Little confidence can be placed in percussion along the spine, though occasionally tympany can be found over the normally dull area along the spine occupied by the kidney; the renal dulness merges with that of the liver and spleen above and extends outward 2.5 inches (6 to 7 cm.) on the left and 3.5 inches (8 to 9 cm.) on the right side. In thin subjects the sinking in of the back and the tumor in front may sometimes be clearly seen. In rare instances the kidney may be found in a hernial sac.

Other Symptoms and Complications.—(*a*) The same train of *neurasthenic*, hysterical or hypochondriacal symptoms are present as in enteroptosis; Tuffier believes that these symptoms are causal rather than sequential. Nervous dyspepsia is particularly common. (*b*) Dietl (1864) described attacks of sudden severe pain and collapse known as "*Dietl's crises*"; they are marked by fever, chill, vomiting, collapse, severe pain, sometimes with swelling and tenderness of the kidney, scanty urine containing blood, pus, uric acid and oxalates, and in some instances peritonitic manifestations; Dietl's theory that the kidney became "incarcerated" between the connective tissue and peritoneum is now abandoned; Gilewski maintains that the ureter is twisted, forming acute renal retention and hydronephrosis, and in one of Sutton's cases, the ureter was found twisted three times. Landau holds that the renal vessels suffer torsion, and Mosler explains the crises by acute nephritis, ureteral stoppage or peritonitis. (*c*) *Dilatation of the stomach* is neither causal or sequential. It is only a coordinate and not at all a common coincidence. (*d*) *Dysmenorrhea*, constipation and enteroptosis are common complications. Colitis mucosa is reported by Morris. Compression or actual stenosis of the intestine, icterus, abdominal aneurysm by tugging through the renal ɜls upon the aorta, compression of the cava inferior and adhesions ıe gall-bladder are rare (and questionable) sequences. Albumin- may occur periodically, on palpation of the kidney or when the ɜnt stands (orthostatic type).

iagnosis.—The diagnosis is rarely difficult. The finding on biman- examination of the sensitive kidney-shaped movable tumor is more ɔrtant than the inconstant subjective or ambiguous neurasthenic ptoms. Severe pain may be confused with a tabetic crisis, appen- is or the passage of gall- or kidney-stones. The kidney may ibly be mistaken for some other movable tumor, as omental, ric, intestinal, and gall-bladder tumors or corset liver. Morris reported cases in which an enlarged gall-bladder has been mis- n for movable kidney; both tumors are movable, may induce us or dyspeptic disturbances, and both may be attended by colic a dull (or resonant) note over the tumor. Doubtful cases are lly determined by repeated examinations, perhaps under anæsthesia. icular reserve is necessary in the diagnosis of left-sided floating ey.

ognosis.—Death is an extremely rare occurrence, though it may t from "incarceration" and peritonitis.

eatment.—The patient should never be told of a movable or ing kidney, because the symptoms, chiefly the neurasthenic, may from the diagnosis. (*a*) A *rest cure* may develop adipose tissue relieve the nervous symptoms. (*b*) A well-fitted *bandage*, with ling to prevent renal descent, is excellent in many cases. (*c*) bstinate and extreme cases, *nephrorrhaphy* or nephropexy, as nmended by Hahn, is indicated. According to Czerny and Giess, ɔ 85 per cent. are cured. Tuffier gives results of 163 cases as ws: absolute recovery 53 per cent.; improvement 30 per cent.; orary improvement 5 per cent., and failure 12 per cent. Frank s the results of Hahn's clinic as 70 to 80 per cent. complete recovery, ɔ 20 per cent. decidedly improved, and 5 to 10 per cent. failures; nortality was under 1 per cent. Nephrectomy is seldom indicated. The crises are treated by morphine, reposition and later by ation.

(F). ALBUMINURIA AND OTHER ANOMALIES OF RENAL SECRETION.

I. Albuminuria.

lbuminuria was discovered by Cotugno in 1770. It results (*a*) ı leakage of albumin through the glomeruli and tubules (*albumin- vera, renalis*) or (*b*) from admixture of blood, pus, semen or fluid umors (*albuminuria spuria*). The genuine form will be first sidered, and the spurious or accidental form later under pyuria hæmaturia.

The most common albumin is serum albumin (or simply, albumin) with which serum globulin is frequently associated.

Etiology.—(*a*) Its incidence in the *diffuse renal diseases*, the nephritides, amyloidosis and passive congestion has already been discussed. (*b*) *Blood alterations* are frequent causes; as (i) blood diseases proper as anæmia, leukæmia, pseudoleukæmia, pernicious anæmia, scurvy or purpura in which the poverty of the blood or toxins induce renal degeneration; (ii) toxæmic states, as the acute and chronic infections, in which "*febrile albuminuria*" (Gerhardt) evidences the renal degeneration; this condition borders very closely on or merges into mild cases of acute nephritis; (iii) diabetes, icterus, and poisoning by lead, mercury, ether, etc. (*c*) *Nervous maladies* possibly act by lowering the local blood pressure, as epilepsy, exophthalmic goitre, migraine, and the psychoses. (*d*) Affections of the *digestive tract*, as intestinal incarceration, hepatic diseases, and lead colic may produce albuminuria. (*e*) In the *new-born*, it was first noted by Virchow. It is almost regular in the first week or ten days of life (Ruge, Martin) and occurs with hyaline casts, urates and epithelium. It is hardly explained by change of metabolism or of circulation or by uric acid infarcts. Ribbert holds that it is due to imperfect development of the glomeruli. (*f*) *Pregnancy* is an etiological factor; it occurs in 40 per cent. of cases in the last few months of gestation (from increased tax on the kidneys, and from pressure) and in the same percentage after delivery (from effort and strain). (*g*) Is there a "*physiological albuminuria*"? Various types are described, as Pavy's cyclical albuminuria, which appears and disappears; intermittent albuminuria; adolescent albuminuria, in young anæmic subjects, often with vasomotor disturbances; orthostatic albuminuria, also termed postural or orthotic, which appears only when the subject stands. Some practitioners bring under this caption albuminuria after heavy eating, severe exercise, coitus, mental strain or cold baths. Certain clinicians as Senator, Bradford and Grainger Stewart, whose names alone carry weight, insist that there is a physiological albuminuria, in which signs of nephritis, as casts, or cardiovascular changes are absent. The author regards every case of albuminuria as pathological; its cause may be an insidiously developing nephritis, a receding nephritis, stasis, or maybe "constitutional," as Martius calls it, but it is always a sign of glomerular or tubular lesion (see PROGNOSIS), however slight it may be. The amount of albumin in this type rarely exceeds 0.4 to 0.5 pro mille (Senator).

Detection.—The urine must be clear and acid; if turbid it should be filtered and if it then remains turbid it should be cleared by shaking with magnesia usta; it must be acidulated, if necessary, with acetic acid; if very concentrated, dilution with distilled water is advisable.

1. Boiling Test.—A test-tube, two-thirds filled with urine, is held by its lower part and the upper layers are boiled. Albumin produces clouding, which may also result from phosphates and carbonates (dissolving when nitric acid is added), from balsams (dissolving in alcohol) and, it is said, from urotropin and formaldehyde. Nitric acid should not be added before boiling, for a soluble acid-albumin may result.

2. **Nitric Acid or Heller's Test.**—Urine, somewhat diluted, is placed in a test-tube or conical glass, and nitric acid is run under it by a pipette. The fuming or nitroso-nitric acid is obtained by heating nitric acid in which a small bit of wood is placed (part of the stick of a match). Albumin, globulin, mucin, and albumose are precipitated at once or after a few minutes at the contact point; albumose dissolves by heating, the others do not. These tests are very satisfactory.

3. **Ferrocyanide Test.**—To a test-tube half full of urine, 10 drops of acetic acid are added, which may precipitate uric acid, urates, oleoresins or nucleo-albumin (mucin). These are filtered out and one dram (5 c.c.) of a 10 per cent. solution of potassium ferrocyanide is added. A precipitate indicates albumin, globulin or albumose, which latter alone dissolves on heating.

4. **Acetic Acid and Salt Test.**—Adding to the urine *acetic acid* to an acid reaction, and then an equal volume of *saturated sodium chloride* solution, precipitates albumin and albumose, but the latter dissolves on heating. The above tests detect albumin in dilutions as low as one part in 10,000.

5. **Spiegler's Test.**—This should be used if more delicate tests seem necessary. Nucleo-albumin (mucin) is removed by adding 15 drops (1 c.c.) of dilute acetic acid and filtering. To the urine is added a quantity of Spiegler's reagent (bichloride mercury 8, tartaric acid 4, glycerine 20, and distilled water 200,) when albumin or albumose develop in the contact zone. One part in 350,000 may be detected. Jolles' modification: bichloride 10, succinic acid 20, sodium chloride 20, and 100 parts of distilled water.

Globulin occurs chiefly in renal congestion, acute and chronic nephritis and amyloid degeneration. It dissolves in an excess of acetic acid. It is precipitated by boiling. Flocculi of globulin are thrown down, when 2 drops of dilute acetic acid are added to 50 c.c. (1.6 ounces) of filtered urine diluted 10 times with distilled water.

Nucleo-albumin (formerly called *mucin*) occurs in acute desquamative nephritis, icterus, poisonings causing necrosis of the renal cells, and regularly in catarrh of the urinary passages. It is precipitated by boiling and by acetic acid, and unlike globulin, does not dissolve in an excess of acetic acid. Shreds of mucus, voided with colic, have been observed by von Jaksch and Baumueller; the condition seems analogous to colica mucosa and spasmodic asthma.

Peptone or albumose is of little clinical importance; the terms include albumins which do not coagulate by heat. When the urine is boiled, the albumin is precipitated and filtered out. Albumose in the filtrate causes clouding and precipitation on cooling. What Kühne once called peptone is now termed albumose. (*a*) *Bence Jones' albumose* is rather a peculiar albumin than an albumose; described by him in 1848, albumosuria has also been called Kahler's disease. Muffat (1905) collected 38 cases. Its significance is uncertain; it has been found in osteomalacia, and in multiple myeloma. It is precipitated by cold nitric acid, dissolves on heating and reappears on cooling. (*b*) Albumose may be found in various acute infections (meningitis, rheumatism, pneumonia), septicæmia, peritonitis, empyæma, in acute yellow atrophy, etc. Albumin in the kidney, bladder or urinal, may be converted by pepsin into albumose.

Diagnosis.—The diagnosis of albuminuria is easily determined by the tests enumerated; blood or pus may give albumin reactions (accidental or spurious albuminuria, *v.* hæmaturia and pyuria).

Prognosis and Treatment.—These are etiological. The outlook is not favorable, though not necessarily bad. In seemingly "physiological" cases, the condition of the heart must be watched and casts carefully looked for. In youth and adolescence albuminuria may be "physiological" but in middle and later life it is usually nephritic. Shattuck and Osler, however, hold that "albuminuria is more common apart from Bright's disease than is commonly supposed, that it increases with advancing years, and that a small amount of albumin with finely granular casts of small diameter in those over fifty years of age is of small importance." Life insurance companies are suspicious of "physiological" albuminuria, for figures show that it entails a death-rate which is double the average. Albuminuria is said to result solely from high arterial tension, but the author is inclined to regard these cases as incipient arteriosclerosis or contracted kidney. Hawkins reported a case in which albuminuria lasted, without any unhappy symptoms, for forty-three years after Bright had given a fatal prognosis.

II. Hæmaturia.

Etiology.—Like albuminuria, blood in the urine (hæmaturia) is only a symptom, and is observed in many diverse conditions. (*a*) It is *most frequently renal.* (i) *General conditions* may cause it, as intoxications (by quinine, carbolic acid, turpentine, or salicylic acid), acute infections (of which malaria is the most common, acute exanthemata, particularly the hemorrhagic types) or the blood diseases (purpura, scurvy, hæmophilia, leukæmia, and the anæmias). (ii) Other causes are *local renal conditions,* as trauma, vascular maladies (thrombosis of renal veins, infarct, aneurysm, varicose veins, arteriosclerosis, and rarely passive congestion); inflammation (acute, chronic, hemorrhagic and suppurative nephritis); rarely amyloidosis or parasites (distoma, filaria, nephrophages sanguinarius); tumors; tuberculosis; calculous ulceration and pyelitis; sometimes floating and cystic kidney, especially after bimanual examination. (iii) Hæmaturia may be "*idiopathic*" *or* "*essential.*" In some very rare cases it occurs in "apparently healthy kidneys," to which phrase there has been a just objection. This condition has been called renal epistaxis (Gull), angioneurotic hæmaturia (Klemperer) and renal purpura (Senator). M. L. Harris collected 18 cases in which simple nephrotomy gave relief. In Eshseries of 48 cases of unilateral renal hæmaturia, nothing was at operation in 33 per cent., floating kidney existed in 12.5 ›nt., renal pelvic changes in 19 per cent., and diffuse renal dis- n 24 per cent. (*b*) It may occur from *lesions in the lower urinary* The chief types of these lesions are stone in the ureter or bladder; red varices anywhere in the urinary tract; stasis from liver cir- or thrombosis of the iliac veins; inflammation (pyelitis, ureteritis, s, prostatitis or urethritis, sometimes called "Russian gonor- "); and tuberculous, malignant, or other ulcerations.

nptoms and Diagnosis.—Hemorrhage may be macro- or micro- :. The urine may be clear or turbid, red or smoke-colored. It ıally acid, but may be alkaline when there is copious vesical rrhage. The color is generally brighter when the urine is alkaline. ısion with concentrated, icteric, carbolic, fuchsin, hæmatoxylin enna urines is possible. The presence of blood is positively nined (*a*) by the *microscope;* the red cells remain normal for a ime in acid urine, but may become pale in dilute, and mulberry- in concentrated urine; (*b*) by the *spectroscope;* if no red cells resent with positive spectroscopic findings, it indicates hæmo- ıuria (*v.i.*); and (*c*) by *chemical tests* as Heller's (boiling with c potash, which throws down a rusty phosphate and hæmatin ›itate), the Almén guaiac test (acidulating the urine and adding ırpentine and fresh guaiac) which gives a blue reaction (also ced by pus) and Teichmann's test (treating the sediment with n chloride and glacial acetic acid, slightly heating and cooling, hæmin crystals are deposited).

gnosis of the origin and cause of hæmaturia is often most diffi- (*a*) In cases of *renal* origin, the blood is intimately mixed with ine, as much with that first voided as with the last. The color ›n that of water in which raw meat has soaked ("beef water" Clots are infrequent in renal hæmaturia, save from trauma, s, aneurysm, or varicosities. The red cells are seldom seen in ux-form and, according to Gumprecht, are fragmented from of the renal cells on the red disks; this does not occur in vesical rhage. If there is some other renal lesion, as nephritis, there re albumin than the red cells would account for, and albumin sent after careful filtering; according to Goldberg, if there is in with a red cell count, by the hæmocytometer, of less than per c.mm., the albuminuria is renal. Blood casts, granular casts enal epithelium are indicative of renal origin. (*b*) Hæmaturia ating in the *pelvis and ureter* is more often marked by clots and ly by caudate epithelium. (*c*) Blood from the *bladder* is more clot, and the last part voided is more bloody than the first. symptoms, as tenesmus, alkaline urine reaction and perhaps of tumor, very often indicate a vesical origin. (*d*) *Urethral* rrhage stains most deeply the first urine voided. It must always me in mind that the presence of blood may be accidental, from rual or other contamination.

Treatment.—The treatment is etiological and can be but briefly considered here. Rest, and sometimes morphine, are indicated. Aside from the local treatment of vesical disease, urethritis, and calculous pyelitis, tannic acid, ergotin and calcium chloride āā gr. v. t.i.d. may check bleeding. Aromatic sulphuric acid ♏x, well diluted, is also given at three-hour intervals. Adrenalin solution 1 to 1,000 parts, given by the catheter, may stop vesical hemorrhage. In essential (idiopathic) renal hæmaturia, simple incision of the kidney is frequently efficacious.

III. Hæmoglobinuria.

This is hæmoglobin in the urine without hæmaturia. Though first described by Dressler (1854), most of the early accounts are English, from Harley, Dickinson, Gull, and Pavy. There are two main groups, the toxic, and the paroxysmal.

Etiology.—The *toxic form* may follow transfusion of blood, injections of glycerin, poisons organic and inorganic, as potassium chlorate, phosphorus, mercury, quinine (in malarial cases only), carbolic acid, carbon monoxide; it may also result from burns, severe icterus, pernicious anæmia, scarlatina, acute nephritis, Winckel's disease in the newborn, and æstivo-autumnal malaria. The *paroxysmal form*, of which Gilman Thompson recently collected 206 cases, occurs largely in England, France, and Germany; 96 per cent. are males, chiefly between *thirty and forty years* of age. *Cold* seems to be the most important factor; exercise, excesses, or menstruation, may precipitate an attack. It is observed in horses after cold and exertion; hæmoglobinæmia and hæmoglobinuria appear, paresis of the hind legs develops and death results from disturbed circulation and respiration. In cattle and sheep epidemics occur in which Babes, Krogius and others have found a hæmatococcus. In the majority of human cases, hæmoglobinuria is preceded by hæmoglobinæmia, though genuine renal hæmoglobinuria (without hæmoglobinæmia) seems to occur in some cases of nephritis and from certain intestinal nematodes.

Symptoms.—Symptoms occur only after destruction of at least one-sixth of the blood. After a chill, fever, lumbar pain which radiates to the legs, cyanosis, oppression and gastric symptoms, the urinary findings appear; the urine is dark and contains clumps of blood pigment, casts and epithelium stained with hæmoglobin or methæmoglobin, albumin and sometimes oxalate of lime crystals and bile pigments. In most cases the hæmoglobin is already altered to methæmoglobin, as shown by the spectroscope. There may be slight jaundice, œdema, or urticaria. The blood shows increased coagulability and diminution in the number and redness of the red cells, which do not form in rouleaux. There may be swelling of the liver and spleen from hæmoglobin deposits in them. The paroxysm lasts one-half to several hours, and in the interval the general health is fairly good.

The course is most chronic. No death is known from the paroxysm itself and very rarely from the disease.

Treatment.—Prophylaxis in regard to colds and exertion may modify the attacks. Chovstek was able to abort attacks by giving nitrite of amyl inhalations. Warmth may modify the seizure. Mercury, iodides, adrenalin, calcium chloride and thyroid extract have been recommended.

IV. Pyuria.

Etiology.—(*a*) *Pus* may originate from *pyelitis* or pyelonephritis. It may occur intermittently as in pyonephrosis, or continuously as in calculous or tuberculous inflammations. The urine is usually acid in tubercle and colon bacilli infections; it is acid in proteus and in some infections ascending from the bladder; the staphylococcus may render the urine less acid than normal or alkaline (*v. i.* pyelitis). (*b*) Pyuria may result from *cystitis;* the urine may be acid or alkaline and contains ropy mucus, and triple phosphates. Pus is seen especially in the last part of the urine voided. (*c*) *Urethral* pyuria, which is usually gonorrhœal, is greatest in that part of the urine first evacuated. (*d*) *Accidental* pyuria occurs from leucorrhœal contamination, and is easily excluded by examining specimens obtained by catheterization. Under this heading may be brought pyuria from rupture into the urinary tract of appendicular, pelvic, or psoas abscesses; much pus is voided at first, but it gradually lessens and disappears.

Symptoms.—The urine in pyuria is yellow-white, turbid, and has a sediment which is sometimes ropy and becomes gelatinous when caustic potash is added. The *reaction* is often ammoniacal, due to germs decomposing the urea, as the proteus, and some staphylococci. Gas may be voided with the urine (pneumaturia); this occurs particularly in enterovesical fistula and in diabetic cystitis. Alkalinity was formerly thought characteristic of cystitic, and acidity, of pyelitic, pyuria, but this distinction has been dropped. *Albumin* is found, the amount of which in simple pyuria corresponds with the number of pus cells. Albuminuria may be absent if there are not over 6–800 pus cells to the c.mm. It is less or may disappear when the urine is filtered. The question often arises whether the albuminuria is simply accidental or is combined with renal albuminuria, for instance, whether cystitis is coincident with contracted kidney. Besides consideration of the amount of urine, its specific gravity, cardiovascular findings, and retinitis, besides washing out the bladder and ureteral catheterization, a study of the characteristics of the pus cells and their number is of great value. Senator has shown that leukocytes coming from the renal parenchyma in nephritis, are mononuclear, and those from suppuration are polymorphonuclear. Goldberg found that when (*a*) there is more than 1 part pro mille of albumin in the clear urine, as determined by Esbach's method, with (*b*) 50,000 pus cells per c.mm. as determined by the hæmocytometer, the albumin is partly renal. De Lint altered Goldberg's method by gravimetric determination of the albumin; when there is one part pro mille of albumin, and less than 7,000 pus cells, the albuminuria is (*a*) renal, and (*b*) it results from the pus alone, when there are more than 40,000 pus cells; no conclusion can be drawn if the pus cells number between 7,000 and 40,000 per c.mm.

Treatment.—The treatment is that of the fundamental tuberculosis, calculus, pyelitis, cystitis, etc. Salol and urotropin āā gr. x t.i.d. are excellent antiseptics for the urinary tract.

V. Chyluria.

The endemic or *tropical form* is found in Florida and South Carolina, Brazil, the Antilles, Zanzibar, India, Egypt, and Japan. It is due to the filaria, which was first recognized by Bancroft and Leurs in the scrotum, by Wucherer in the urine, and by Lewis in the blood. It enters the body by the drinking water in which mosquitoes have upon their death liberated filaria embryos. The filaria plug the lymph vessels, which dilate and rupture in the kidneys, as proven by Mackenzie's autopsy. They are found in the blood at night (see ANIMAL PARASITES). The *non-tropical forms* are not understood and have rarely occurred in Europeans who have never been in the tropics; the rare autopsies have shown no marked renal alteration (Roberts).

The *urine* resembles chyle, is weakly acid or neutral in reaction, may present a *fatty, creamy surface* and contains 2 to 10 per cent. albumin (serum albumin, globulin or albumose), cholestearin, lecithin, fat, and stearic and palmitic acids. Its specific gravity is normal. Sugar is seldom found. Microscopic findings are fat globules, leukocytes, sometimes red disks and sometimes the filaria parasites, usually in the fibrin clots, which sometimes form and occasion dysuria. Casts are never found. Chyluria is often intermittent; it occurs especially at night and may be initiated by exercise and changes of posture. In some cases there is considerable free fat in the blood (*lipæmia*). Its course is chronic, covering months to years. Recovery is the rule, though death may occur from anæmia and marasmus. *Treatment* is unsatisfactory. Potassium picronitrate gr. $\frac{1}{8}$ t.i.d. is said to accelerate recovery. Prophylaxis regarding drinking water must be considered in the tropics.

VI. Lipuria.

Lipuria or adiposuria is symptomatic of (*a*) *lipæmia*, or fat in the blood. The clearest instance is after bone fractures, contusions of the liver, or after delivery in which fat enters the blood from the pelvic cellular tissue. Fatty foods, as cod-liver oil; acute yellow atrophy; diabetes; diseases of the pancreas, and various poisonings and tumors are possible causes. (*b*) It is also symptomatic of *renal lesions* (from fatty epithelial cells, degenerated tumors, or fatty pus cells).

Under the *microscope* small fat globules are seen, which stain black with osmic acid, or red with the alcanna test. The fat may be saponified by alkalis, emulsified by albumins or dissolved by alcohol, ether, xylol or chloroform. Lipuria is often simulated by bacteria or phosphates, or by oil from càtheters, suppositories, or greasy urinals.

VII. Phosphaturia.

Phosphoric acid is chiefly excreted in combination, mostly with sodium and potassium forming the alkaline phosphates, and with calcium and magnesium forming the earthy phosphates. The sodium exceeds the potassium, and the calcium is in excess of the magnesium salts. Phosphoric acid is chiefly derived from the food, but some is derived from decomposition of lecithin, nuclein, and protagon. It averages thirty to forty-five grains (2 to 3 gm.) daily. Amounts over one dram (4 to 5 gm.) or alteration of the normal relation of the phosphorus to the nitrogen (17 or 20 to 100) are pathological. An excessive deposit which occurs in dyspepsia, is not synonymous with an actual increase. Phosphaturia may be observed in neurasthenia, pulmonary tuberculosis, diabetes (largely from the diet), acute yellow atrophy, leukæmia, and in anæmia. As much as two drams (7 to 9 gm.) daily have been reported by Teissier and Ralfe, who termed the condition *diabetes phosphaticus*. Earthy phosphates may cloud the last part of the urine, which is mistaken by the laity for "spermatorrhœa." The addition of an acid rapidly clears the urine. The *diet*, recommended for this condition, consists of meat, fish, bread, potatoes and apples. Coffee, nux vomica, arsenic, cod-liver oil, or ammonium benzoate (gr. x to xxx t.i.d.) are also given.

VIII. Lithuria.

No subject has occasioned more clinical absurdities than uric acid. It normally bears to urea a ratio of 1 to 70. Futcher has found a ratio in gout of 1 to 300 or 500 (*v.* GOUT). It is deposited in larger amounts than normal when the urine is very acid and the mineral salts or pigments are decreased. An increased precipitation is far more common than an absolutely increased excretion of uric acid. The sediment is clearly seen as red granules resembling red pepper. Uric acid combines chiefly with ammonium and sodium, and less with potassium, lithium and calcium, which form *urates* (*sedimentum lateritium*) of the pink or "brick-dust" color so frequently seen in febrile urine and urine of high specific gravity or of excessive acidity. They are soluble when heated and precipitate on cooling.

IX. Oxaluria.

Oxalates, first detected by Donne (1838) in the normal urine, amou to less than 10 mg. daily. Quantities larger than this daily are pat ological. Helen Baldwin, in Herter's laboratory, decided that they a not formed during normal metabolism; they must then be taken with the food. The actual conditions of oxaluria are in doubt. has been observed in gout, emphysema, dyspepsias (especially in hype acidity and increased intestinal putrefaction), neurasthenia, icter and diabetes. Oxalates occur as octahedral and dumb-bell crysta which according to Klemperer occur in 50 per cent. of renal calcu Cantani and others maintain that oxaluria is associated with *nervo depression, irritability,* lassitude, hypochondriasis, *lumbar pain,* swe and nervous *dyspepsia,* though the concensus of opinion is against a definite clinical picture or diathesis. Klemperer has recently advised diet of meat, fat and vegetables (except spinach and cabbage); excludes milk and eggs and administers magnesium sulphate ℥ss on daily. Nitromuriatic acid ♏ij with tincture of nux vomica ♏x is old and excellent empirical treatment.

X. Indicanuria.

Potassium indoxylsulphate is usually present in small amounts in normal urine. It is colorless as a rule, but may be voided blue or actually give a violet hue in alkaline urine. When acid or some oxydizing substance is added, it decomposes and frees indigo. The steps in its production are as follows: the action of the pancreatic juice on albumins produces indol, which is absorbed, oxydized in the tissues to indoxyl and unites with potassium sulphate to form potassium indoxylsulphate. Its absence in some pancreatic diseases has been discussed. It has been rarely found in urinary calculi. It is increased in cachexiæ in which albuminous substances are decomposed, in intestinal obstruction particularly of the small bowel, and in carcinoma, suppuration, and peritonitis. Boiling the urine and add nitric acid, as in the ring test for albumin, gives a blue ring; add half a dram (2 c.c.) of chloroform and shaking brings down the vio or blue-stained chloroform.

XI. Other Anomalies.

Alkaptonuria was discovered by Baedecker. It is a crystalli aromatic body, probably derived from proteid decomposition, wh chief clinical significance is that (*a*) it reduces Fehling's soluti thereby simulating glycosuria, though it is negative to polarization fermentation tests; and (*b*) it imparts to the urine a dark brown c ially apparent only after it has stood or after caustic potash is added captonuria by derivation means urine which "absorbs" an "alkali" ly). Adding a drop of a very dilute solution of ferric chloride ses a dull green color which at once disappears. It is said to be nogentisic or uroleucinic acid. Sometimes it occurs as a familial ction. Garrod collected 40 cases, 73 per cent. of which were les; in 32 congenital cases, 19 of them occurred in 7 families. Vir-w (1866) described a case of *ochronosis*, a discoloration of the tilages and ligaments. Osler described 2 cases associated with aptonuria, making in all 9 cases in the literature; it may occur h pigmentation of the face, chronic arthritis and deformation of the bar vertebræ.

ydrochinon, from carbolic acid poisoning, may cause a somewhat ilar discoloration.

æmatoporphyrinuria, discovered by Hoppe-Seyler, imparts a dark r to the urine. It occurs in pulmonary tuberculosis, rheumatism, stinal hemorrhage, sulphonal and lead-poisoning, etc. Hæmato-phyrin resembles hæmatin, save that it contains no iron.

cetonuria is of clinical importance, chiefly in diabetes (*q.v.*).

or fuller details the reader may consult works on clinical diagnosis, as the recent treatises by Simon, Emerson and others.

PYELITIS—PYELONEPHRITIS—SUPPURATIVE NEPHRITIS.

Pyelitis.

yelitis and pyelonephritis are inflammations of the pelvis, and pelvis kidney respectively. They were first described by Rayer.

tiology.—Pyelitis is almost invariably *mycotic*. The kidneys ally eliminate bacteria without injury to their structure but when general vitality or the local resistance of the kidneys is lowered, lammation of the pelvis or of the kidney may result. Various cteria (*v.i.*, SUPPURATIVE NEPHRITIS) may be found, including the ercle bacillus and other organisms. (*a*) *Foreign bodies*, as large, rd, or sharp calculi, less often cancer or tubercle, and still more rarely rasites, promote infection. (*b*) *Ascending infections* from the lower nary tract are peculiarly dangerous when associated with urinary structions, as urethral stricture, hypertrophied prostate, or perhaps pressure of a pregnant uterus. When the bacteria which produce itis cause decomposition of urea, the ammonia evolved acts as an litional eroding factor. According to Rosving, certain streptococci in this way, but not the tubercle or colon organisms. Infection ally ascends from cystitis along the ureters, producing ureteritis, n pyelitis and perhaps a pyelonephritis, but it may reach the kidney ng the lymph vessels, the microbes entering through erosions in vesical mucosa. James Goodhart has shown that 50 per cent. of es with obstruction to the flow of urine die of renal suppuration.

(*c*) Infection may be *hæmatogenous* as in typhoid, the exanthemata, or in the intestinal catarrhs of infancy. (*d*) Less common factors include *traumatism*, extension by *contiguity* from neighboring inflammations, as pyelonephritis (pyelonephritis is usually an ascending process, in which the kidney is secondarily involved, and only rarely is it a process descending from the kidney to the pelvis), toxic factors, as acrid, irritating diuretics such as turpentine, and hemorrhagic diseases, as black measles or scurvy; cold, venous stasis, and diabetes are somewhat predisposing factors; arteriosclerosis promotes the form of pyelitis described by Ollivier, which is attended by hemorrhage and the formation of fibrin coagula. Pyelitis is most prevalent in males and in adults.

Pathology.—Pyelitis may be acute or chronic; solitary or combined with cystitis; catarrhal, suppurative or ulcerative; unilateral or less often bilateral. Acute catarrhal forms present all the characters of mucous membrane inflammation, as redness, œdema, desquamation, formation of mucopus and minute ecchymoses. Chronic forms show grayish to reddish-brown pigmentation and thickening. In any form, croupous or diphtheritic inflammation or even gangrene may develop. Urates and triple phosphates may precipitate. Small cysts may form in the pelvis or ureter; this is caused by retention or perhaps by sporozoa; the small lymph nodes may enlarge, and in rare cases the epithelium may hypertrophy and thicken, causing cholesteatoma (*v. i.*, issues).

Symptoms.—(*a*) There are no certain symptoms in the rare cases in which a suppurative nephritis descends to the pelvis. (*b*) In the ascending type, early vesical symptoms antedate and often obscure the pyelitis. (*c*) The symptoms are most clear in primary pyelitis from calculus or from acute hæmatogenous infection.

Pyuria is the chief symptom. The amount of pus is usually proportionate to the albumin (*v.* PYURIA). Its amount varies as well as its constancy; when the ureter is obstructed, pyuria is suspended, and when the obstruction is removed the pus escapes in increased amounts. *Polyuria* and a low specific gravity are usual in chronic cases; the urine may be doubled or tripled in amount, simulating diabetes insipidus; polyuria results from decreased absorption of water from the medullary portion or from compensatory hypertrophy of the sound kidney, or sometimes of the heart. In acute cases the urine is decreased. There may be *other urinary findings*. The urine is *usually acid* but quite often alkaline (from coincident or causal cystitis, or from bacteria decomposing the urea). It is often *cloudy*. It is true that large, caudate, club-like epithelial cells, often overlapping each other like shingles, are present in moderately severe pyelitis, but they may occur, though less frequently, in ureteritis and cystitis. When pyelitis extends to the papillæ or higher (pyelonephritis), cylindrical plugs of pus cells, epithelium, but chiefly of cocci, are seen (Fischl). Red cells (calculus), triple phosphate or uric acid crystals, fibrin (croupous or diphtheritic pyelitis), and various bacteria (perhaps tubercle bacilli) may occur. *Pain* over the kidney is usually dull, but in acute cases may suggest stone. There is a desire—probably reflex—to urinate frequently; this symptom may lead to an erroneous diagnosis of cystitis. *Fever and rigors* are not uncommon, especially in severe calculous or tuberculous types or those invading the kidney substance. Temperature is sometimes sudden and high ("irritative") and at other times is intermittent from septic absorption.

Complications.—(*a*) There may be extension to the renal substance (*pyelonephritis*) and it may be reduced to a pulpy inspissated and even calcified mass. (*b*) Prolonged inflammation may lead to fibrosis of the kidney (Ackermann, Albarran); this is known as the "*ascending contracted kidney.*" (*c*) Plugging of the ureter may occasion distention of the pelvis by retained urine (*hydronephrosis*) or pus (*pyonephrosis*), both of which conditions will be described below. (*d*) *Anuria,* described by Morgagni and later by Israel, is occasioned by reflex suppression of the sound kidney, or by extensive lesions in both kidneys. (*e*) *Amyloidosis* may be a complication. (*f*) *Intoxication* may occur by bacterial toxins and renal inadequacy. The condition resembles uræmia in some respects, but œdema, retinitis, and convulsions are absent. Senator described a dyspnœa like that of diabetic coma. Treitz and v. Jaksch believe that intoxication results from absorption of ammonia (*ammoniæmia*) from decomposed urea. (*g*) Perforation outward causes the *paranephritic abscess* (*v.i.*). In some cases an interesting condition called "spinal paraplegia" has been described; though it was once considered reflex, it probably results from an ascending neuritis.

Prognosis.—The prognosis is influenced by the basic infection (whether simple, tuberculous, or calculous) and by the complications as described. In acute cases the outlook is generally good. Chronic cases without much necrosis may last many years and may even recover.

Diagnosis.—Much depends on careful analysis of pyuria (*q.v.*). *Cystitis* may be confused with as well as combined with pyelitis. Their differentiation is often difficult from examination of the urine alone; the albumin and pus correspond in both, and possibly there is rather more albumin in pyelitis; no absolute reliance can be placed on the reaction, acidity, or alkalinity occurring in either condition, though pyelitic urine is probably more often acid. The tailed, clubbed, or shingle-like epithelia are more common in pyelitis, though not pathognomonic. Von Bergmann's method consisted of thorough vesical irrigation and then gentle compression of the suspected kidney to force into the bladder, and thence into the catheter, pus or cells; pain over the kidney and extending downward is significant; the only certain method is cystoscopy and ureteral catheterization. In *nephritis* the urine contains more albumin than is explained by the pus, though the amount may be slight in interstitial nephritis; the leukocytes are mononuclear and the presence of casts, cardiovascular changes, and the results of cryoscopy are usually decisive.

Treatment.—*Prophylaxis* involves care in the use of catheters, [illegible] irritating diuretics, the expectant treatment of gonorrhœa, etc. *Water*, *milk* and mineral waters should be used freely to flush out the urinary tract. *Pain* is relieved by hot applications to the kidney and by giving five grains of powdered nutmeg; opium is seldom indicated. For *pyuria*, salol and urotropin āā gr. x p.c. are most efficient; the salol somewhat influences the pain. Methylene blue gr. j to ij, t.i.d. may be also given, but digestive disturbances must be watched, for. Potassium chlorate gr. x to xx t.i.d. may lessen ammoniacal fermentation but may cause intoxication. These remedies are superior to lead acetate, sandal oil, or turpentine. *Surgical interference* may be necessary when ureteral drainage is incomplete. Nephrotomy is the best operation, nephrectomy being seldom indicated.

Suppurative Nephritis.

Its *etiology* is partly that of pyelitis. The microörganisms encountered are the staphylo-, strepto- and pneumococcus, Bacillus coli, gonococcus, proteus, typhoid and tubercle bacillus, and actinomyces. (*a*) The *embolic form* results from suppurative processes elsewhere and represents merely one phase of septicopyæmia of more pathological than clinical interest. The suppurative foci are nearly always *bilateral* and are chiefly *cortical;* the glomeruli are filled with microörganisms and, as the foci enlarge, they show as yellowish areas through the cortex; some deposits may also occur in the medulla; the kidneys are enlarged and lax. Clinically, *pyuria*, hæmaturia and cylindruria are *seldom* seen, and a diagnosis can seldom be made; anuria or oliguria may result from multiple glomerular involvement. (*b*) The *ascending type* is essentially a cystopyelonephritis, which has been already described, and forms the so-called "surgical kidney"; infection and urinary obstruction are its usual antecedents; pyuria, polyuria and albuminuria its usual symptoms; and suppuration in the medullary rays its usual anatomical evidence; very rarely necrotic renal tissue is evacuated. (*c*) Suppuration of the kidney may result from its *invasion by contiguity* from adjacent pus foci, as appendicular, peri-uterine or vertebral foci; much pus may be voided at once with later gradual cessation of the pyuria.

The diagnosis is always uncertain, save when sequestra come away, the prognosis always ominous and the treatment always expectant and surgical.

(H). PERINEPHRIC ABSCESS.

A distinction is sometimes made between perinephritis, inflammation of the fibrous capsule, and paranephritis, inflammation (suppuration) of the fatty capsule and surrounding tissue.

Etiology.—(*a*) The most common cause is *suppurative* disease of the *kidney*, as pyelitis, pyelonephritis, pyonephrosis (*v. i.*), and tuberculosis, from which extension occurs by actual rupture or along the blood or lymph vessels. (*b*) *Extension by contiguity* may occur from vertebral caries, appendicular, pelvic or pancreatic suppuration, liver abscess, cholecystitis or even from the lungs and pleura. (*c*) It may result from *trauma* plus infection. (*d*) The so-called *idiopathic cases*, once attributed to cold, are probably *embolic*. Most cases occur in persons between thirty and forty years of age, and 70 per cent. occurs in males.

Symptoms.—(*a*) The *onset is acute*, with a rigor and sudden temperature in the embolic (so-called primary) cases, but it is *insidious* in the secondary group. (*b*) *Fever* of a continuous or remittent type is usual, though it may be absent in some cases, *i. e.*, absence of fever does not exclude suppuration. (*c*) *Pain and tenderness* are experienced over one kidney, somewhat more often over the right than the left. Sometimes, though seldom without coincident involvement of the kidney itself, pain shoots along the ureter to the pubes or thigh. To relieve tension the patient frequently lies on the back with the *knee flexed* and the thigh rotated outward. It may be here remarked, though we are discussing suppuration about the kidney (paranephritis) that pain may arise from simple inflammation of the fibrous capsule (perinephritis); this is relieved by simple nephrotomy. (*d*) *Local findings* may be marked or absent. In some patients there are lumbar *œdema*, redness and *swelling*. The physical signs anteriorly are seldom marked, as suppuration usually occurs behind the kidney. The surgeon finds at operation a diffuse phlegmon, and less often diffuse serous infiltration or circumscribed abscess; the pus may be odorless; it may smell of urine when the abscess has arisen from perforation outward of a pyelitic lesion; a fæcal odor results from infection (without perforation) from the bowel or from appendicular abscess. Necrotic fat and sometimes altered blood are voided. (*e*) The *urine is negative*, unless there are (i) a pyelitic origin and (ii) rupture of the abscess into the urinary tract. (*f*) The chief *complication* is *rupture*, which occurs in about a quarter of the cases; in 15 per cent. it occurs upward (Küster), into the lungs and pleura, and in 8 per cent. it is downward, chiefly into the intestine. Burrowing to the peritoneum, groin, pelvis, or perineum may follow; according to Gerota, it occurs along the ureter toward the pelvis, when the abscess is anterior to the renal fascia and toward the lumbar region or iliac fossa when the abscess is behind the fascia. *Amyloidosis* may result from neglected or obscure cases of paranephritis. Serous pleurisy, subcutaneous emphysema, compression of the portal vein, icterus, and septicæmia may result.

The *course* averages two to four weeks, though a longer duration is not uncommon.

Diagnosis.—Diagnosis depends on (*a*) local pain, (*b*) œdema or swelling, (*c*) fever and (*d*) exploratory aspiration. Lumbago causes no serious confusion. Other affections causing occasional confusion are psoas abscess, appendicular abscess behind the kidney or rupture downward of empyema though these are more often etiological factors than separate questions for differentiation.

The only *treatment* is incision and drainage. In the cases of Schede and Schmieden complete recovery occurred in 73 per cent.

(I). HYDRONEPHROSIS.

Definition.—Hydronephrosis is a sac-like dilatation of the pelvis and calices, with accumulation in it of a watery fluid; obstruction to the urinary flow is its cause and atrophy of the renal substance is its result. It was first described by Tulp (1674) and named by Rayer.

Etiology and Pathology.—There are two forms, the congenital and acquired. (*a*) The *congenital form* is most often due to anomalies in the ureters; these include atresia, stenosis, valve formations, twists, compression by peritonitic bands, vestiges of Müller's or Gärtner's ducts, abnormal bloodvessels, imperfect insertion into the bladder, or insertion too high into the pelvis, in the lower part of which the urine accumulates. Of the latter form the author has seen two cases in young men and J. B. Murphy has observed four others. When there are double ureters, one may end blindly in the bladder wall, and dilating, may compress its fellow of the same side. In some few cases no obstruction is apparent. Hydronephrosis may occasion dystocia and is rather commonly associated with other congenital malformations; 65 per cent. of congenital cases are bilateral (Roberts). (*b*) The *acquired form* may be bilateral or unilateral, depending on its cause. This may, in rare cases, be in the kidney itself (Rayer, Chopart, Christian Fenger), producing partial hydronephrosis. Enumeration of all possible causes of obstruction is unnecessary; one or both ureters may be compressed by malignant pelvic growths, as uterine cancer, and this in part explains the large proportion (67 per cent., Newmann) of bilateral hydronephrosis which occurs in autopsy figures; in clinical cases 62 per cent. are unilateral (Roberts). Ureteral obstruction may arise from calculus, tumors, tuberculous detritus, ulceration, cicatrization, floating kidney, or rarely clots from trauma or parasites. Large sacs are usually caused by affections of the ureter. Other possible types are obstruction from urethral stricture, enlarged prostate or more rarely phimosis and atresia of the hymen.

As Cohnheim contended, a slow development with intermissions is more potent in the production of large sacs than is acute obstruction, for slow compression permits more continued renal secretion.

The *results* of *urinary accumulation* are (*a*) distention of the pelvis, (*b*) flattening of the papillæ, (*c*) atrophy of the renal tissue, though some normal tissue usually remains, and (*d*) the formation of a fibrous sac.

Symptoms.—The chief finding is a *fluctuating renal tumor.* (*a*) It is located at first in the *renal region* but later assumes an abdominal importance. (*b*) Its *size varies* in different cases, being largest when the ureter is obstructed and in slowly developing cases it may be as large as the head, and even simulate ascites; Peter Frank mentions a case in which it contained 60 quarts. (*c*) It may in the same case vary from time to time; this is known as *intermittent hydronephrosis*, and its chief causes are valve formation, stenosis or abnormal insertion of the ureter into the pelvis. The amount of urine is (i) normal from hypertrophy of the sound kidney, (ii) decreased in bilateral disease or from increased pressure in the diseased kidney, lessening the secretion from the sound kidney (Israel); or (iii), when the sac evacuates, suddenly very abundant, and then the tumor disappears; this point is often volunteered in the patient's history. The author has seen hæmaturia and renal colic occur in hydronephrosis due to an insertion of the ureter high up in the renal pelvis. (*d*) Its *form*, on bimanual palpation, is *irregularly oval;* in moderately severe cases the kidney itself may be palpated as well as its irregularly protuberant calices. (*e*) Sometimes the dilated ureter can also be felt. (*f*) The *ballottement rénal* is a repercussion shock obtained by tapping the tumor with fingers of one hand, while the other is spread broadly over it. (*g*) Small tumors are *covered by the colon*, which imparts a modified tympanitic note on percussion. Distention of the colon by air or gas brings out tympany over the tumor. Very large sacs push the colon aside. The *ren saccatus* may develop in a floating or horseshoe kidney. (*h*) Its *contents* are albumin, paralbumin, mucus, urea, uric acid and other urinary constituents, which are usually absorbed only after a long time. Diagnostic puncture is not always safe. (*i*) Sometimes it is *tender* from tension of the capsule; pain may also arise from torsion of the ureter. (*j*) *Complications* include rupture, infection (pyonephrosis), left ventricle hypertrophy, and anuria or uræmia if both kidneys are implicated.

Diagnosis.—Diagnosis is quite easy (*a*) by recognizing the cause, if possible, as stone or tumor; (*b*) by careful bimanual palpation and (*c*) by cystoscopic proof that urine does not flow from one ureter. *Differentiation* concerns especially—

1. The Exclusion of Other Renal Tumors.—(*a*) *Pyonephrosis*, a dilatation of the pelvis and calices by purulent fluid (instead of water-like fluid as in hydronephrosis), is caused by obstruction plus infection (especially from pyelitis with obstruction). It occasions the same kind of tumor as to location and physical examination, but presents some other features; it is generally smaller and more irregular than hydronephrosis; its walls are thicker and more tender, for infection is superimposed on obstruction; there is more renal necrosis; systemic septic symptoms prevail; its contents are pus, pulpy detritus, triple phosphates, bacteria, disorganized blood, fat, cholestearin, fœtid ammonium compounds and even gas. Puncture is always dangerous, in doubtful cases more so than operation. Its prognosis also is clearly more unfavorable. (*b*) *Cystic kidneys* (*q.v.*) are very difficult (or impossible) to differentiate. (*c*) Malignant *renal growths*, and (*d*) echinococcus, will receive consideration below.

2. Other Abdominal Tumors.—(*a*) *Ovarian tumor* is connected with the uterus, grows upward, and may be associated with changes in the mammary glands and genital functions. Ovarian tumor with a long pedicle may deceive, but it pushes the cæcum backward. The dulness is greater, though in large renal sacs the colon is pushed aside. The slight respiratory excursion and *ballottement* in hydronephrosis is of quite secondary diagnostic value. Confusion is very likely to occur when hydronephrosis develops in a floating kidney. Exploratory puncture is an unsafe procedure; when made, the fluid from ovarian cysts shows a high specific gravity (1,020); much paralbumin is present, also cylindrical epithelium, colloid or chocolate-colored material and of course no urea. Under narcosis the entire hand may be introduced into the rectum or colon to determine the point of origin of the tumor, though this method is severe, and is seldom necessary. (*b*) *Ascites and pregnancy* are readily differentiated. (*c*) *Encapsulated peritonitis* may be harder, but the urinary conditions and sac-like distention are usually decisive. (*d*) *Splenic* and *hepatic* tumors are more flat on percussion, encroach more upon the lungs and are above or anterior to the gas-distended colon. In a renal growth a furrow and tympanitic loop of bowel may lie between it and the liver or spleen.

Prognosis.—The prognosis is generally unfavorable, as the cause is often inaccessible or the process well advanced. The prognosis is better in cases of renal stone than in cancer or in the generally inviable congenital forms. The course is often protracted and intermittent.

Treatment.—Treatment is surgical. Nephrotomy is indicated, while nephrectomy is generally considered bad surgery since Ayer and others have shown that some sound renal tissue nearly always remains. For the same reason ligature of the renal vessels should be avoided (Nicolai). The mortality resulting from nephrectomy for hydronephrosis is 5.7 per cent., for pyonephrosis 15.3 per cent. (Schmieden); Rosving reports 0 per cent. and 19 per cent. mortality in his cases of nephrotomy for hydro- and pyonephrosis, respectively; 34 per cent. of his cases recovered completely, 27 per cent. recovered though with fistula, and secondary operation was necessary in 20 per cent. Fenger advised conservative operations, especially in partial hydronephrosis. Reference should be made to surgical treatises for details of operations on the ureter, excision of the pelvic sac, etc. Caspar and Kümmel maintain that the phloridzin and cryoscopic tests are of great value in determining which side is involved, by testing the urine obtained from each ureter by ureteral catheterization; Israel has a very different opinion, as has Rosving, who prefers to cut down on both kidneys before extirpating either one.

(J). RENAL CALCULUS (NEPHROLITHIASIS).

Etiology.—Stone in the kidney or its pelvis results from deposition :rein of the solid constituents of the urine. It is probable that stone mation is due to *bacteria*. The kidneys usually eliminate bacteria thout changes to themselves, but sometimes bacteria, especially the lon bacillus (Schmorl), form the nucleus of stones. Ebstein, Moritz, endelsohn and others have shown that an albuminous cement sub-ınce is found even in very slight urinary deposits or renal sand. :nal stone has been known since the time of Hippocrates.

The *predisposing factors* are obscure. (*a*) A *sedentary life* and over-lulgence in food and alcohol are apparently predisposing factors. den noted the coincidence of gout and renal calculus. Stones are d to be uncommon in very active persons. (*b*) Most cases occur persons between *thirty and sixty years* of age, though they may velop in early youth or old age, in which extremes of life vesical culus is more common. (*c*) Seventy-five per cent. of cases are in *les*. Stones are passed more easily, and, therefore are possibly less en detected, in women. (*d*) Renal calculus occurs most frequently Asia and there are certain foci, more of them in Europe than in ıerica, where it is particularly frequent, as central Russia, Holland, ly, Altenberg, Munich, Hungary, Scotland, west France and south-t England. (*e*) Other possible factors are heredity, which is espe-lly a factor in formation of uric acid and cystin calculi; paraplegia, üller reported 10 cases of spinal fracture with calculus formation); l foreign bodies in the renal pelvis, as blood clots from trauma, l such parasites as the distoma. The older classification of primary l secondary calculus has been abandoned.

hemistry and Pathology.—According to their *size*, these urinary osits are spoken of as sand, gravel or stones; sand and gravel ırly always consist of uric acid or its salts. They may range from size of a pea to that of a hazel-nut. The heaviest recorded stones ighed nineteen and thirty-six ounces. Though they are most often ınd, oval or cylindrical, they may be dendritic or coral-shaped. metimes they are facetted. A number of them may exist, perhaps or 20; in rare cases 1,000 to 2,000 may be found. (*a*) The stone used by *uric acid* and urates (described first by Scheele, 1776) is e most common form, constituting 66 per cent. of renal stones. It hard, smooth or slightly uneven, sometimes facetted or lamellated, ıd consists of uric acid, ammonium and sodium urates, urinary gment, and a small amount of xanthin. Its broken surface is amor-ıous. Its color is yellow, red, or brown, depending on the amount urinary pigment. It may contain some oxalates and phosphates. nder the microscope needles are seen. Uratic calculi turn carmine d on adding nitric acid and ammonia. They dissolve in caustic tash. They are deposited in acid urine, but the exact reason for is is not known; the normal sodium urate may lose part or all of its sodium by action of the alkaline phosphates, so that free uric acid, and the less soluble bi- and quadriurates are formed; Roberts and Klemperer consider a decrease of the urinary pigments of great importance; disturbed metabolism or indigestion may occasion uratic deposits, though little is known of the "uric diathesis." (*b*) *Oxalate stones* (Fouccoy and Vauquelin) are next in frequency; Klemperer holds that oxalates occur in even 50 per cent. of cases. They are very hard, rough, warty or prickly (the mulberry calculus); for this reason they cause more pain and hæmaturia than the uratic stones and are more often removed surgically. They are usually single and gray, but may be dark-colored from the bleeding they induce. Lamellation is never observed. Uric acid, calcium carbonate or xanthin may also enter into their composition. No effervescence occurs on adding acetic acid, unless carbonates are also present. They dissolve in mineral acids, and oxalate of lime crystals gradually precipitate on the addition of ammonia. Oxalates precipitate from acid urine. (*c*) *Phosphatic* calculi (Bergmann) are formed in alkaline or neutral urine. They are usually calcium salts, but if the urine is ammoniacal, triple phosphates may be formed. Admixture of bacteria, calcium carbonate and xanthin may be noted; phosphatic stones are small, soft and clay-colored. They are infrequent and usually only form a coating to calculi of other composition. (*d*) *Other forms* are rare. Calculi of (i) *calcium carbonate*, and more rarely ammonium or magnesium carbonate, may occur in the aged, though rarely in man compared with their frequency in cattle; they are white and dissolve with effervescence in mineral acids. (ii) *Cystin* calculi (Wollaston) are smooth, yellow, and waxy; they never exceed the size of a pea, are soluble in acids and alkalies and deposit hexagonal crystals when dissolved in acetic acid and ammonia. Only 60 cases are reported (Riegler, 1904). (iii) *Xanthin* stones (Marcet) occur almost exclusively in children; only 10 cases are recorded; they are smooth, yellow or brown, dissolve in nitric acid, and on evaporation leave a residue which turns red on adding caustic potash. (iv) Only 3 cases of *indigo* stones are on record. (v) The fatty stones (*urostealiths*) described by Heller probably do not originate in the kidneys, but result from fatty substances deposited in the bladder; they are 85 per cent. fat, and are soft, rubber-like, and combustible.

Infarcts may be considered here. (*a*) *Uric acid* infarcts occur in gouty subjects and in 50 per cent. of the new-born, chiefly in the medullary collecting tubules, but sometimes in the cortex; they are most common in the still-born or in weakly infants, and are thought to result from destruction of the tissues or cells (nuclein). (*b*) *Lime infarcts* are chiefly phosphates and carbonates but sometimes oxalates; they are deposited principally in the pyramids and in the membrana propria when the renal cells are destroyed; they occur in the aged, and also in poisoning by mercury, arsenic, phosphorus, or bismuth. (*c*) *Pigmentary infarcts* of hæmatoidin, bilirubin, hæmoglobin or methæmoglobin are uncommon.

Symptoms.—Stones may be first discovered accidentally at autopsy, or may pass without symptoms, though very exceptionally. Gravel or renal sand is more often latent than is stone.

1. *Pain* is the most common symptom. It is of two kinds: (*a*) The *dull pain* in the loin, often associated with local tenderness over the kidney; Fenwick describes localized pain without any urinary changes, indicating that the stone lies in the medullary substance. Pain may be felt over the opposite kidney. (*b*) *The renal colic,* which arises from incarceration of the stone as it attempts to leave the pelvis, where it nearly always originates; it is often preceded by pain in the loin and may be initiated by exercise, as riding, or by indigestion or alcoholic indulgence. Eichhorst mentions a surgeon who had renal colic every time he drank white wine. The pain in renal colic begins over the kidney and *radiates down the ureter* to the testis, labia, bladder or urethra; the testicle is retracted. It may rarely radiate to the chest, heart, shoulder, epigastrium or to the opposite side. During the colic, the *kidney* is so frequently *sensitive,* that when it is not so, Israel hesitates to diagnosticate stone. The *pain* is *cutting* and is often very *severe;* it often occasions reflex nausea and vomiting, sometimes chills, sweats and fever, or in children convulsions, and rarely collapse, or even death in most exceptional cases. Vesical tenesmus is frequent. If the stone passes, the pain is greatest as it enters the bladder. Constipation of a spastic order is much more frequent than diarrhœa or involuntary movements. Some observers hold that renal colic occurs oftenest at night. It lasts, with intermissions, a few hours, rarely days; movement may excite recurrence. The patient lies on his back with the leg drawn up and the abdominal wall rigid. After the attacks there may be dull pain (*v.s.*), which may be caused by large, numerous, or sharp stones. Eichhorst records a case in which the sole symptom was the patient's attitude; he leaned far forward and depressed one shoulder to avoid the pain induced by the erect attitude.

2. *The urine* is almost invariably altered. (*a*) *Hæmaturia* occurs ndependently of pain and colic, as well as after them. Absence of æmaturia after a renal colic, speaks strongly against a diagnosis of one (Israel). It is often macroscopic, and red disks are always found ith the microscope (Musser), except in the rare cases when stone is bedded in the renal tissue, or there is permanent occlusion of the eter. At the time of the colic the scanty, bloody urine is frequently ided with considerable tenesmus. In exceptional cases, early pro- e hæmaturia may result from vascular erosion. Musser found line casts in nearly every case. (*b*) *Anuria* (suppression of urine) ot common. It may occur (i) with stone in one ureter, and disease trophy of the other kidney, (ii) with bilateral calculi (Albarran), with stone in one ureter, and reflex suppression (Morgagni) e other (sound) kidney through the splanchnic nerve, or (iv) by ral stoppage by a stone. Legeau (1891), quoted by Dieulafoy, ed 30 autopsied cases of calculous anuria; there was stone in

one kidney, and the opposite kidney (α) contained a calculus in 14 cases, (β) was hypoplastic in 3, (γ) sclerotic in 6, (δ) its ureter was stenosed in 6, and (ε) in one case the opposite kidney was absolutely normal. The resulting symptoms resemble those of uræmia. However, in 41 cases collected by Herter, convulsions were present in but 12 per cent., headache in 14 per cent., and vomiting in 29 per cent. Consciousness is usually preserved, the pupils are usually narrowed and the temperature is low. Roberts speaks of this condition as "latent uræmia"; 11 cases of anuria persisted over four days, 18 cases seven to fourteen days, and seven over fourteen days. Russel records recovery after twenty-eight days of anuria. With its relief large quantities of urine are voided. (*c*) *Pyuria* results in protracted cases from pyelitis, which is much oftener secondary to the calculus than primary. Rather infrequently a renal intermittent *fever* is observed, resembling the hepatic intermittent fever of gall-stones. Pyelitis may be catarrhal, suppurative, ulcerative or perforative. (*d*) *Crystals* of uric acid, and oxalates are considered especially important in the diagnosis by some writers, as Leube; Ulzmann regarded the pointed uric crystals as particularly suggestive of renal calculus. (*e*) *Stones in the urine* determine the diagnosis, though they are not frequently found.

3. *The x-rays* may disclose renal calculi. It is certain that oxalate stones throw a darker shadow than uric acid stones. Opinions differ regarding the value of the *x*-rays. The difference may be wholly technical, as Leonard reports the unusual figures of but 3 per cent. of errors; he found calculi in the ureter twice as often as in the renal pelvis. Kümmel insists that every stone casts a shadow.

4. *The physical findings* are generally less important than the urinary findings and the colic. Local tenderness has been mentioned (*v.s.*, PAIN). Enlargement of the kidney (hydro- or pyonephrosis) is not common. Palpation of the stone in the ureter is rarely possible, either from without or by the rectum or vagina, though once the writer felt a calculus at McBurney's point. Stones have rarely been felt to crepitate in the renal pelvis (Piorry). Cystoscopic examination and catheterization of the ureters are methods which can be employed only by the most expert, and have their disadvantages as well as advantages. M. L. Harris' segregator may determine which kidney is affected.

Complications.—Ulcerative *pyelitis* may lead to perforation and paranephric abscess. Pyelonephritis may develop. Hydronephrosis results from mechanical occlusion of the ureter, and pyonephrosis from obstruction with infection. Injury to one kidney may be compensated by hypertrophy of its fellow. Amyloid degeneration, renal tuberculosis, or even cancer, from irritation, may develop. The ureter may rupture from ulceration or tension. Pregnant women may abort, and Troja instances a case in which 14 abortions occurred after 14 attacks of colic.

Diagnosis.—The diagnosis is based on (*a*) the association of the pain and colic, (*b*) hæmaturia, (*c*) pyelitic pyuria, (*d*) *x*-ray findings,

l (*e*) less often, the anuria and acute swelling of the kidney. The
ults of the *x*-rays, if positive, are definitive; if negative, stone is
excluded. It is said that a diagnosis of stone is found correct in
y 60 per cent. of the cases operated on.

. *Pain and colic* may occur, though rarely, in other renal conditions,
in renal embolism or passage of blood clots, parasites, echinococcus
icles, malignant tissue or tuberculous detritus, renal aneurysm,
sular thickening and floating kidney. In one case previously
rated on for appendicitis, the author made a diagnosis of calculus
m pain, colic, and hæmaturia; the operation by Murphy revealed
artial hydronephrosis from high insertion of the ureter. Confusion
y result from vertebral disease, the crises of tabes or angioneurotic
ema, intestinal colic, gall-stones, appendicitis, diaphragmatic pleu-
r or hysteria.

. *Hæmaturia* (*q.v.*), associated with pain, may cause an error in
gnosis, which is usually corrected only at operation; thus acute
gestion and particularly capsular fibrosis may be found. Israel
tes that there is a unilateral nephritis (*a*) with colic, (*b*) with albumin
d without casts; (*c*) with casts and without albumin; (*d*) with hemor-
age, simulating tumor; but (*e*) the bleeding never causes the colic.
these doubtful cases exploratory incision is also indicated. The
maturia resulting from cancer and tuberculosis does not improve
th rest in bed, while calculous hæmaturia usually does. Hæmaturia
d renal colic may develop in chronic nephritis from acute renal con-
stion (Israel).

Prognosis.—The prognosis must be conservatively made, for the
urse is usually chronic and marked by relapses. The author knows
four physicians who passed calculi without symptoms afterward,
t this is not the rule. Rupture of the ureter, collapse during the
ic, pyelitis becoming pyelonephritis, anuria and uræmia must be
nsidered in making the prognosis. Pyuria is a more ominous
velopment than hæmaturia.

Treatment.—1. *Prophylaxis* of renal calculus must be indefinite,
ause so little is known regarding its pathogenesis. It is usually
ted that regulation of the diet concerns moderation in both proteids
l carbohydrates; as in nephritis (*q.v.*) foods rich in nuclein are to
avoided; vegetables may be allowed. Acid foods and alcohol
uld be interdicted, water is given freely, and exercise must be
isted upon. In uric acid calculi, the alkaline waters are valuable,
those of Karlsbad, Vichy, Marienbad, Saratoga, Bedford, Poland
l Mountain Valley. Sodium phosphate gr. x or bicarbonate ʒss
d. are indicated. It is questionable whether lithium carbonate is
ric-acid solvent; it is given in doses of grs. iij to v t.i.d. in effervescing
ters. Piperazin and glycerin possess no proven solvent powers.
localities where calculi develop very frequently, the drinking water
st be used with caution. Of course stones once formed cannot be
solved by drugs.

If the oxalates are deposited, treatment is the same as in oxaluria (*q.v.*). In alkaline urines (from which phosphates are precipitated) the urine must be made acid by salol, gr. x t.i.d.; acid waters, as Seltzer or Apollinaris may be employed; meats should be restricted; urotropin gr. x t.i.d. is excellent, especially in bacteriuria. Altogether, diatetic treatment, though important, has in the author's opinion, no great bearing on prophylaxis or treatment, and he cannot agree with those who declare that these measures are generally efficacious.

2. The *colic* should be medically treated like that of gall-stones (*q.v.*).

3. *Surgical intervention* is indicated by (*a*) anuria; (*b*) severe pyelitis; (*c*) septic or aseptic obstruction of the ureter; (*d*) exhausting hæmaturia, and (*e*) repeated attacks of colic. As one or more of these conditions is present in most cases, successful treatment must be surgical.

The ideal operation, advocated by Morris (1880) is nephrolithotomy (nephrotomy), closing the renal wound if there is no infection; operation on the aseptic kidney entails a mortality of 3 per cent. and operation in the presence of infection 10 per cent. (Albarran); in these cases the pelvis should be drained (nephrostomy). Tenny (1904) collected 134 cases of ureteral calculus; 122 were operated on, with a mortality of 19 per cent., or of 47 per cent. if pyelitis was present; anuria is a serious complication, for 52 per cent. of the operated cases die; without pyelitis or anuria, the fatalities were 6 per cent. Rosving has operated without a death. Medical treatment of anuria, with full doses of nitroglycerine, hot packs, cups to the lumbar region and subcutaneous saline transfusions should not last more than a day or two, for delay raises the mortality from operation; nephrostomy is indicated.

(K). TUMORS OF THE KIDNEYS.

I. Cancer.

Etiology.—The etiology is obscure. It constitutes but 2 per cent. of all cancers and but 1 per cent. of primary cancers in adults (Rubinstein); 62 per cent. occurs in males, and 32 per cent. in the first ten years of life. Its frequency in children was noted by Bright (though some reported cases were sarcomata), and it constitutes 52 per cent. of renal tumors in children (Hirschsprung). Weigert observed one case in a new-born child. Cancer in the pelvis may result from stone. In the Albarran and Imbert series of 380 renal tumors, cancer constituted 50 per cent.

Pathology.—The primary form is usually infiltrating, and the secondary form consists of nodules. Medullary cancer is more common than scirrhus, colloid, and other forms. It usually begins in the convoluted tubules of the cortex. Its growth is usually limited by the kidney's capsule. The kidney is nearly always enlarged, its average it in children being 8½ pounds (Guillet) and in adults 10 to 15 ds (or even 30 to 50). The opposite kidney and the sound parts e affected viscus may show compensatory hypertrophy. Hemor- s into the neoplastic tissue may occur. Local invasion of the veins, ureter, inferior cava, pancreas, adrenals or intestine and stases (58 per cent.) in distant organs sometimes develop.

mptoms.—Some cancers are clinically latent, particularly secon- growths.

Renal tumor is the most common finding, and occurs in 97 per of cases (Guillet). It begins in the region of the kidney, but, resisted by the firm lumbar tissues, it grows forward. Bimanual tion clearly demonstrates its origin in most cases. If large, it fall forward over the spine. It is tender and usually *firm*, though egeneration may occasion slight fluctuation. The form of the y is usually preserved, though nodules may be palpated. It lly attains a large size (*v.s.*), compressing and dislocating the and spleen upward, and crowding the abdominal viscera. Für- er reports a case in which the heart was pushed up to the clavicle. apping it a repercussion wave is sometimes noted (*ballottement*). Percussion gives a dull note, except so far as the intestines er it, when mixed flatness and tympany are elicited. The *colon es the kidney*, running from left to right, from above downward; e left side this relation is more clearly distinguished; on inflation e colon with air or gas, its tympanitic note in some degree obliter- he renal dulness; in a recent study by Kofmann, this condition ound in less than 50 per cent. of large renal tumors; in tumors e right side the colon is often dislocated laterally or even down- . There is but little if any respiratory excursion. In rare in- es the tumor may pulsate and produce by pressure on the aorta tolic murmur, which, as in Richard Bright's case, may simulate ysm. If the tumor grows into the ureter, hydronephrosis may ent the swelling.

Pain is an early and frequent symptom, due to pressure exerted e lumbar plexus. For this reason also there may be slight sensory rbance in the legs. Pain is usually dull and only rarely is it lan- ing or colic-like, due to passage of clots or bits of tumor (*v.i.*).

Hæmaturia is an early symptom. According to Guillet it occurs per cent. and is the first symptom in 25 per cent. of cases; Israel that it occurs in 92 per cent. and is the first manifestation in 70 ent. It is less frequent in children (16 per cent.) than in adults. times worm-like blood clots are passed, with great colic; they times measure one-half to one inch; clots in a clear urine are st pathognomonic (Israel). The cystoscope and ureteral cathe- tion prove that the hæmaturia is unilateral. The hemorrhage lly originates in the diseased kidney but may come from the sound gh hyperæmic) kidney. In other repects the urine is generally al. It is usually held that albuminuria corresponds to the amount od, but Albarran asserts there is more albumin than the blood accounts for. Very rarely bits of cancer tissue pass. The urea is increased as in all cancers, and the chlorides are sometimes decreased. Pyuria is no part of renal cancer.

4. *Cachexia* develops in most cases, but not in all.

Complications.—*Compression* symptoms include paraplegia dolorosa, constipation, intestinal obstruction, œdema from compression of the iliac veins and ascites; varicocele, which was first noted by Guyon, is said to seldom occur in other renal tumors, and is due to late compression of the spermatic vein by the tumor or contiguous lymph nodes. Metastases, enlarged inguinal glands, rarely growth into the lumbar muscles or skin, and perforation into the peritoneum or intestine, with fatal hemorrhage, uræmia, fever from cachexia or terminal infection, may be observed. According to Israel, tumor of the renal pelvis causes greater hæmaturia, and more hydronephrosis (therefore greater variations in the size of the tumor); it sometimes grows down the ureter and is seen with the cystoscope to project into the bladder; particles of tumor tissue and cells are more often obtained. Albarran and Imbert collected 65 cases of tumors in the pelvis and ureter; 27 were ureteral. Savory and Nash collected 60 cases of painful villous tumors in the pelvis.

Course and Prognosis.—Death is invariable without operation; it usually results from cachexia, in two years in adults and in eight months in children (Rohrer and Roberts). Guillet states that 6 cases have lived longer than ten years and 1 lived seventeen years.

Diagnosis.—The following are of clinical importance: (*a*) renal tumor, (*b*) pain, (*c*) hæmaturia, and (*d*) cachexia. Imbert speaks of the complete type (tumor and hæmaturia), the hæmaturic type (without tumor, 6 per cent.) and the tumor type (without hæmaturia, 23 per cent.).

Differentiation is necessary, from (*a*) *liver tumor*, which encroaches much more upon the diaphragm and is characterized by its respiratory excursion, by the location of the tumor within the edge of the liver, and by such complications as icterus and ascites. The colon lies below the liver and there are no urinary findings. Bimanual palpation clearly differentiates uncomplicated cases. Between a kidney tumor and the liver a furrow and loop of resonant intestine are frequently found. (*b*) A *splenic tumor* maintains the form of the spleen, lies above the colon, (whose distention does not modify the splenic dulness), moves on inspiration, shows the characteristic notches and seldom causes confusion except when it so rotates that only its convexity can be felt. (*c*) An *ovarian* tumor projects upward from the pelvis, with which its connection can be demonstrated, save when it has a long pedicle. It is more central and sustains no characteristic relation with the colon. (*d*) *Retroperitoneal* growths are much more difficult to differentiate, especially enlarged lymph glands and lymphosarcoma. They are generally more central and less movable. The author recalls an enormous tumor which was considered renal, but which proved at autopsy to be a retroperitoneal lipoma. (*e*) Other renal ons must be differentiated. (i) *Cystic degeneration* (*v.i.*) is ' bilateral and runs a most chronic course. (ii) Sarcoma cannot 'erentiated. (iii) Hydro- or pyonephrosis (*q.v.*), in which the varies exceedingly and has characteristic urinary changes. (iv) :ulosis is distinguished by hectic fever, tuberculosis elsewhere, , and finding of the tubercle bacilli.

II. Sarcoma.

r-six per cent. occurs under ten years of age; G. Walker in ses, found that most cases occurred in the first two years of life. an and Imbert state that sarcoma constitutes 20 per cent. of rowths. It is slightly more common in girls than in boys, and left than in the right kidney. Cohnheim's theory of embryonal on is supported by the occasional presence of cartilage, bone, riated and unstriated muscle. The sarcoma is of the round, : or melanoid cell type and may contain furrows, fatty, vascular, en carcinomatous tissue. It may weigh 6,000 gm. Secondary ata may develop by metastasis from other organs, or by invasion tiguity.

ymptoms are essentially those of carcinoma, from which an tam diagnosis is very rarely possible, except when sarcomatous s passed in the urine or excised at operation. Hæmaturia and ia are infrequent; age is of no diagnostic value, and glandular ment may occur in either. In some cases a remarkable over- of the pubic and axillary hair, and pigmentation of the skin : observed. Operated cases live on an average sixteen months n-operated ones, half as long (G. Walker) but death is frequently t result of the operation.

III. Hypernephroma.

rawitz named this form "struma lipomatodes aberrans renis"; nor develops from aberrant (included) particles of adrenal Small tumors are not a rare autopsy finding. They constitute cent. of renal tumors and consist of a scanty but vascular tive tissue stroma, containing fatty cells like those of the adrenal Hemorrhage, cystic degeneration, a tendency to invade the ind a large proportion of glycogen and perhaps cholestearin or are considered characteristic. Fatty cells may be found in ne. Hæmaturia occurs in 80 per cent. of cases. Metastases evelop. Bone metastasis is the first symptom in 16 per cent. y develop as a late symptom only. Fever is noted in 57 per hough it occurs in but 1 or 2 per cent. of other tumors of the (Israel).

IV. Other Tumors.

Other tumors as fibroma, lipoma, myxoma, angioma, and rhabdomyoma are of little importance. Hæmaturia occurred in only one case, an angioma. Thirty-three cases of tumor of the capsule are on record.

Treatment of Renal Tumors.—This is symptomatic and surgical. The former concerns chiefly the management of pain, by means of anodynes, chiefly opiates. Surgical treatment alone is of value. The mortality resulting from total nephrectomy in Gross' figures was 61 per cent.; in Küster's, 24 per cent. Schmieden collected 329 cases; 33 per cent. (average) have died from the operation; the mortality in the first decade of renal surgery (dating from Gustav Simon, 1869) was 64 per cent., in the next 43 per cent., in the third 22 per cent. The mortality of nephrectomy for all causes is at present 17 per cent. and for tumor 22 per cent. (in children, 28 per cent.). Thirty-three recoveries after radical operation are on record, one patient being alive after eighteen years. Israel in 43 cases reported a mortality of 18.6 per cent., of which two-thirds died of heart paralysis; 8 cases (19 per cent.) enjoyed permanent recovery, after three and a half to fourteen years. Schede in 18 operations obtained 4 permanent recoveries (22 per cent.) after four to nine years. Recurrence after two years is very exceptional. The extraperitoneal operation is three times as safe as the transperitoneal. Operations are usually performed too late; advanced cachexia, very large tumors, metastases and growth into the cava are contra-indications. The other kidney must be in good condition to admit of operation.

(L). CYSTIC DEGENERATION.

Various renal cysts are observed. (*a*) Those which occur in chronic interstitial nephritis are the most frequent; they result from snaring off, by fibrous tissue, of the tubules and glomeruli. (*b*) Parasitic cysts (*v.i.*) are next in frequency. (*c*) Dermoid cysts are very rare; but two are on record. (*d*) Cysts of the fatty capsule, retroperitoneal cysts, and degenerated lymph glands, are also very rare. (*e*) Complete cystic degeneration (*hydrops renum cysticus, degeneratio renum polycystica*) was first separated by Rayer from the above mentioned varieties and constitutes cystic degeneration, as it is generally understood.

Etiology.—Over 50 per cent. is observed in *men* between *forty and fifty years* of age. Luzzatto found but two cases (in 187) in persons under twenty years; Sieber (1906) collected from the literature 32 cases of cystic kidneys in persons under and 211 in those over twenty years of age. Cystic kidneys are observed in later fœtal life, at birth, and in the adult. It is often difficult to determine whether e acquired or congenital, though the latter conception is most ; the cysts are thought to remain latent until middle life.

ogenesis.—Three main theories obtain; (*a*) that they are n cysts, (*b*) that they are new formations, *i.e.*, neoplastic enomata), and (*c*) that they are malformations. The neoplastic ion is probably the most acceptable hypothesis.

ology.—Congenital cases are almost always bilateral; Lejars ne unilateral case in 67, and Luzzatto 41 in 226 cases. The may contain innumerable small cysts, when the shape of the is preserved, or it contains larger cysts, when its contour is regular. According to Engländer, only 23 cases of unilocular e on record. The size of the kidneys may be that of a child's they may even weigh twenty-one pounds. In congenital cases times causes dystocia. On section the kidney is honey-combed. st walls consist of thin or thick fibrous tissue. The cysts usually thin, yellow, transparent fluid, less often milky, gelatinous or sh contents (altered blood). They are uni- or multilocular. re lined with cells like the urinary tubules, or with flat polyg- lls. The cysts contain albumin, oxalate of lime, leucin-like , red disks, epithelial detritus, fat granules, and rarely urea or d. Between the cysts vestiges of renal tubules or glomeruli are which here and there may be vicariously hypertrophied. nay occur in the pelvis, ureter and liver (*v.i.*). In congenital her deformities are sometimes found, particularly in the genito- tract, brain and fingers.

ptoms.—In congenital forms the cystic kidneys may cause an tion to delivery; if the infant is born alive, death from asphyxia l, for the diaphragm is crowded upward. In adult forms, 90 t. of cases come to autopsy, undiagnosticated, or with the is of some other renal disease as nephritis. The symptoms classified as follows: (*a*) *Renal cysts* are found clinically in cent. of cases only (Luzzatto) or in 12.4 per cent. (Sieber). eneral characteristics are those of solid kidney tumors, but they nost always *bilateral* (*v.s.*). Fluctuation seldom occurs. In w cases the individual cysts can be palpated; in a case which hor demonstrated in the Chicago Medical Society they could rly palpated. Echinococcus cysts, hydro- and pyonephrosis, a and cancer of the kidney are only distinguished by their al occurrence; if bilateral, differentiation is almost impossible. e *urine* is variable; it may be normal, increased or decreased. inary findings are typically like those of interstitial nephritis, urine is abundant, with few solids, low specific gravity, and f albumin. Red disks are often found (20 per cent.), some- great numbers. In two of the author's cases red cells appeared bers each time the kidneys were palpated. Leucin-like bodies pear in the urine, or in exploratory punctures; Beckmann first ed these small colloid bodies ("rosettes") with structureless and one to five concentric rings and clearly radiating striations.

(*c*) *Cardiovascular* changes (left ventricular hypertrophy and sclerosed arteries) are common and heighten the resemblance to contracted kidney. The changes were well marked in the three cases which the author was able to diagnosticate. (*d*) *Uræmia* is the common cause of death. The clinical course is very chronic and is broken by attacks of "periodic uræmia." The digestive disorders of uræmia (see CHRONIC INTERSTITIAL NEPHRITIS) are very frequent. Lejars observed a case living fifteen years, and Senator one living twenty years after the cysts were found.

Diagnosis.—The diagnosis is difficult in many cases, but can be made when there are (*a*) bilateral (perhaps palpably cystic) renal swellings; (*b*) the cardiovascular and urinary findings of contracted kidney, with hæmaturia and colloid "rosettes," which are obtained in the urine, or by puncture; (*c*) uræmia and (*d*) perhaps renal pain, and tenderness or congenital cysts in the liver (in 28 per cent., according to Lejars); in a case of Dr. W. F. Dickson of Chicago, renal cysts were found with cystic liver, atrophic heart, and pain resembling appendicitis (see ECHINOCOCCUS OF THE LIVER, DIFFERENTIATION). Hanot and others describe cases resembling calculous colic.

Treatment.—Operation is permissible only in the exceptional, unilateral cases, or when suppuration develops. About one-third of the operated cases die immediately, and in few are any lasting results obtained. The treatment is that of chronic nephritis.

(M). RENAL PARASITES.

Echinococcus cysts (see section on ANIMAL PARASITES) of the kidneys, occur in 9 per cent. of echinococcus disease (Neisser's 900 cases). In 970 cases (Vegas and Cranwell), the liver was diseased in 64 per cent., the lungs in 7 per cent., the spleen in 3 per cent., and the kidneys in 2 per cent. Baradulin in 1906, collected 142 cases of renal echinococcus.

Symptoms.—(*a*) A *renal tumor* is found in 50 per cent., when the cyst attains large dimensions. It is easily confused with other cysts, hydronephrosis, cancer, or tuberculosis without the exploratory puncture, and the urinary findings. *Fluctuation* and the *hydatid thrill* (see LIVER ECHINOCOCCUS) are not often found. The cyst begins in the cortex, and usually in either pole of the kidney; it is unilateral in 96 per cent. of cases, and is left-sided in 62 per cent. If the cyst calcifies it may be seen by the *x*-rays. The opposite kidney may hypertrophy. (*b*) Exploratory *aspiration* may show (i) fluid or pus, containing urinary constituents, cholestearin, succinic acid, much sodium chloride and a reducing substance; (ii) vesicles, hooklets, or particles of lamellated membrane. (*c*) The *urine* may contain vesicles, hooklets, membrane, pus from pyelitis, and blood from rupture. As many as 100 vesicles have been passed. (*d*) Passage of vesicles is attended by *colic*, as severe as that of nephrolithiasis, with fever, chill, and vomiting.

Complications.—Seventy-five per cent. *rupture*, chiefly into the renal pelvis; perforation may occur into the intestines, lumbar muscles, lung or pleura; the patient may feel the rupture. The toxic substances, liberated by rupture or puncture, may cause urticaria, severe heart symptoms or even fatal syncope. Adhesions to the liver, spleen, and intestine may form. Suppuration or gangrene may develop (pyonephrosis or paranephritis). Obstruction of the ureter causes hydronephrosis; vesicles which plugged the lower end of the ureter have been seen with the cystoscope.

Prognosis and Treatment.—The course is very chronic, covering years (even twenty to thirty). Suppuration may cause pyæmia. The cyst may shrink or calcify. Bilateral involvement makes the prognosis unfavorable. Spontaneous recovery by rupture into the urinary tract, occurs in 32 per cent. (Béraud). Operation should be extraperitoneal. Nephrotomy is indicated, as there is seldom more than a single cyst. Nephrectomy produces a higher mortality (8 per cent.). In 30 operations (nephrotomy), by Schede, there were no fatalities; 87 per cent. completely recovered, and 10 per cent. recovered but had fistulæ.

The **Enstrongylus gigas**, which occurs frequently in dogs or wolves, is very rare in man. It is a nematode whose ova appear in the urine. It causes pyelitis, hæmaturia, colic, tenesmus and sometimes chyluria.

The **Distoma hæmatobium**, described by Bilharz (1851) in Egypt, causes endemic hæmaturia. It is a trematode and enters the alimentary tract, by the drinking water. Reaching the veins, its ova obstruct them and cause pyelitis, hæmaturia, tenesmus, and sometimes form the nucleus of renal stones. Salol is said to be beneficial.

The **filaria** has been mentioned under CHYLURIA.

Tuberculosis and **syphilis** of the kidney are considered under INFECTIONS.

SECTION VI.

DISEASES OF THE BLOOD.

ANÆMIA is a reduction in the blood as a whole, or more often a reduction of its important constituents, as the red corpuscles, their number, hæmoglobin or albumin. Every anæmia has a cause, yet in our present ignorance, we adhere to the older division of anæmia into primary and secondary forms. *Primary anæmia* includes chlorosis and pernicious (progressive) anæmia.

(A). CHLOROSIS.

Definition.—Chlorosis is a disease of hæmogenesis, occurring almost invariably in women, chiefly in the developmental years and in the decennium following, in which the cardinal symptom is anæmia; the red cells are reduced but the hæmoglobin is disproportionately reduced; chlorosis develops spontaneously, usually runs an uncomplicated course, tends to recur, and responds to therapy by iron. Chlorosis is a disease, while anæmia is but a symptom.

Etiology.—Its etiology is uncertain. (*a*) Nearly *all* cases occur *in women;* a few alleged instances in delicate men are recorded. It develops especially in girls who menstruate before the pubes and mammæ develop (Niemeyer). It is more common in blondes than brunettes. (*b*) Chlorosis occurs largely between the *fourteenth* and *seventeenth years.* Great care must be exercised in diagnosticating the disease, after the twenty-fourth year (chlorosis tarda), unless previous attacks have occurred. (*c*) *Heredity* is undoubtedly a marked factor (Bramwell). Chlorosis, tuberculosis or hysteria is not infrequently associated in the family history. (*d*) *Malnutrition,* lack of hygiene, insufficient or excessive exercise, working in dark quarters, nostalgia, improper clothing, wearing of corsets, and emotional or sexual factors (unsatisfied desire, masturbation), are indirect causes. Various theories have been advanced as to the nature of the disease, as deficient development of the genitalia, hypoplasia of the vascular apparatus, adynamia of the blood-making organs, reflex action of the nutritive organs from genital maturity, inability of the blood-making organs to respond to the bodily needs at puberty, insufficient absorption of iron, dilatation of the stomach, constipation, enteroptosis, auto-intoxication from the internal genitalia at the time of puberty, and intestinal auto-intoxication.

General Symptoms.—Among the early symptoms are *rapid tiring*, *dyspnœa* and *palpitation*, which are rarely absent and are most noticeable in the morning. *Pallor* is the earliest and most constant sign and affects the skin and mucosæ, the skin being of a *yellowish-green hue* (chlorosis, as named by Varandel, in 1670). Sometimes the color is maintained, the face being flushed, probably from peripheral irritability of the vasomotor system (*chlorosis rubra*); hence the mucous membranes should be examined, even more than the skin. The eyes are often brilliant. Psychical changes are noted (Sydenham and Morton), as stupor, and *lack* of *mental and muscular energy*. Associated symptoms are *headache*, which may be dull or sharp, sometimes migraine-like, or increased at the time of menstruation, spots before the eyes, tinnitus, vertigo, and syncope which occurs especially on standing and results from travel and exposure to the sun, but less from movement. *Menstrual disturbances* are frequent, the flow being often decreased, most markedly in younger subjects. Digestion is very frequently disturbed, and there may be *anorexia*, perversity of appetite craving of sour articles, hunger between meals, pain in the stomach eructations and cardialgia. *Constipation* is frequent. The hands are cold, the skin dry and the peripheral circulation sluggish. Circulatory symptoms appear, as the *bruit de diable* in the neck veins, *anæmic heart murmurs* and rapid pulse.

Special Symptoms.—1. *The blood* is fluid and its coagulability is increased, thereby contrasting with pernicious anæmia and leukæmia. The red blood cells are decreased (*oligocythæmia*) and average *three to four millions* at the first examination; as few as one million red cells have been observed. In some rare cases they are not decreased (Duncan, Hayem, Laache). Formation of rouleaux is poorly marked. The red cells appear *pale* and their average *size is reduced*. Irregularity in contour (*poikilocytosis*) is not frequent; megalocytosis and microcytosis are rare; normo- and megaloblasts are found only in the severest cases; normoblasts are sometimes present in "blood crises." More suggestive than the red cell reduction (oliogocythæmia) is the reduction of hæmoglobin (*oligochromæmia*), which is reduced more than the red cells (Duncan, 1867); the hæmoglobin averages 30 to 40 per cent. This *reduction in the color index* always suggests chlorosis, is most marked in the first seizure in young individuals, and is less conspicuous in relapses or in older individuals. Some writers claim that the reduction in hæmoglobin is more apparent than real, because there is more rapid regeneration of the cells than of the hæmoglobin. The *white cells* are essentially *normal*, or at least but little increased, therein differing from many other forms of anæmia; occasionally lymphocytosis and eosinophilia occur. The iron and nitrogen of the blood and its specific gravity are reduced proportionately to the decrease in hæmoglobin.

2. *The vascular system* presents various symptoms. (*a*) Rokitansky and Virchow observed hypoplasia of the aorta and vessels, which may co-exist with hypoplasia of the genitalia, muscles and bones, but cannot be regarded as causative. (*b*) *Palpitation* is almost constant on exertion; the apex-beat is strong to compensate for the poverty in hæmoglobin. (*c*) *Heart murmurs* occur in two-thirds of the cases; they are largely functional and may be caused by insufficient tension of the mitral valve, dilatation of the left ventricle causing relative mitral insufficiency, weakened heart muscle (producing a muscular murmur in place of the tone), tricuspid insufficiency or vibration of the pulmonary artery. The murmurs are largely systolic; undoubted diastolic venous bruits have been heard. (*d*) *Dilatation of the heart* occurs from anæmia or myocardial degeneration, though according to Leube, Grunmach and von Noorden, the dilatation is not real but apparent, being due to retraction of the lungs away from the heart, because of insufficient lung expansion; this has been confirmed by the *x*-rays. (*e*) The *pulse* findings are not constant; the pulse may be rapid, throbbing, or excitable. The arterial tonus is reduced, dicrotism and capillary pulsation are frequent, and double tones have been noted in the crural vessels from decreased tension and diastolic recoil. (*f*) A venous bruit (*bruit de diable*) in the neck occurs in over 50 per cent. of cases, and more frequently than in other anæmias; it may be palpable and is heard more frequently over the right jugular vein, because it is more vertical than the left; it also is heard in other veins, and in sound individuals. It is increased in the sitting posture, by turning the head to one side, and by deep inspiration. Its explanation is difficult; it is referred to change in the tonus of the vessel, waves in the veins above their valves, and a change of the blood plasma. (*g*) Arterial murmurs may be heard, as over the subclavian artery; pulsation in the peripheral veins, angiospasm, and erythromelalgia, have been observed. (*h*) *Œdema* is seen in 12 per cent. of cases, and is usually slight, occurring chiefly about the ankles and eyes, and rarely in the serous cavities.

3. In the *respiratory tract*, dyspnœa is a common complaint, and is described by Eichhorst as an hæmatogenous respiratory insufficiency. The respiration rate is 26 to 30. Sometimes a paroxysmal cough develops.

4. The *digestive tract* is involved in 31 per cent. of cases. There is sometimes *fœtor ex ore*. (*a*) *Pain in the stomach* is frequent; it may occur near the ensiform with tenderness, especially in nervous

individuals. It sometimes results in vomiting, and may be associated with pyloric cramp; similar pains occur in the transverse colon. Sensitiveness at the tip of the twelfth costal cartilage is due to pressure from corsets. (*b*) *Dyspepsia* may be purely nervous. The stomach is exceptionally dilated and atonic, but as a rule no motor insufficiency occurs. Corsets and chlorosis may cause gastroptosis. (*c*) The hydrochloric acid, formerly thought to be decreased, is increased in 80 to 95 per cent. of cases; nervous irritability is its most probable cause. (*d*) It is said that *constipation* is usually present, but it is probably no more frequent than in the average case; intestinal fermentation may co-exist but it is not the cause of chlorosis.

5. In the *sexual sphere* there may be symptoms. The uterus infantilis and small ovaries are not causal, but are coördinate with chlorosis, being evidence of degeneration or hypoplasia. Menstruation is decreased or absent in 80 per cent.; menstrual disturbance is neither the cause nor result of chlorosis; both result from a common factor. Conception is unlikely to occur during a chlorotic attack. Chlorotics are often first treated by the gynæcologist, for leucorrhœa (in 22 per cent. of cases).

6. *Special senses*. The usual symptoms of anæmia, as spots before the eyes, and weakness of accommodation, are observed. The retinal vessels may be transparent or pulsating; retinitis and neuroretinitis with degeneration and exudation, or "choked disk," occur in exceptional cases and simulate brain tumor. Retinal hemorrhage is rare.

7. The *skin* is usually anæmic; angiospasm, coldness, and paræsthesia occur. In chlorosis rubra the cheeks are flushed. Seborrhœa and acne are very common. Sweats, urticaria, eczema, purpura, chloasma and trophic disturbance of the hair and nails may occur.

8. The *nervous system* may be affected. Chlorosis often brings out a latent *neurosis*,—especially hysteria,—and psychosis, the latter of which is frequently curable. Neuralgia is infrequent.

9. *Metabolism* is not essentially disturbed. The body-weight is maintained, less from decreased oxygenation than from inactivity or water retention. Some cases are emaciated, and occasionally an actual gain in weight is noted. The *urine* is clear, of low specific gravity, often alkaline with decrease of urea and uric acid and sometimes albuminuria. Fever is usually held to indicate a severe anæmia, though Leube has found it frequently in mild chlorosis. When present it is always well to differentiate "tuberculous pseudochlorosis."

Complications.—Chlorosis may induce *acute infections*, as typhoid, pneumonia, influenza and scarlatina, and may render their clinical course severe. Venous *thrombosis* complicates 2 per cent. of cases, occurring largely in the femoral veins, sometimes in the cerebral sinuses, and axillary and other vessels. It is probably inflammatory. In Leichtenstern's series of 86 cases with thrombosis, 56 per cent. involved the veins of the leg and 34 per cent. the cerebral sinuses. Pulmonary embolism occurred in 25 per cent. of 52 cases of thrombosis of the lower extremities (Welch). Virchow emphasized the frequency of septic endocarditis. Rheumatic endocarditis with organic valvular lesion is frequent, as also are ulcer of the stomach, tuberculosis and Basedow's disease. The *spleen* is enlarged (in 37 per cent. according to Chvostek, or in 16 per cent., according to Leube). The thyroid gland is often moderately enlarged. *Hemorrhages* are not infrequent from the nose, stomach or intestine, although it is usually stated that they occur mostly in severe or pernicious anæmias.

Clinical Course and Prognosis.—Simple chlorosis develops rather suddenly in a few days or weeks. It runs a relatively *short course* of two to three or four months. The prognosis is generally good. The tendency to *recurrence* is great, especially in unmarried or childless women; the blood may not return absolutely to normal; this is shown by persistent pallor or irregular menstruation. *Acute chlorosis* is observed especially in young girls after their first menstruation; it is attended by severe headache, nervous symptoms, great weakness and optic neuritis or "choked disk," and may be confused with organic disease, as brain tumor. The *periodic type*, according to Von Noorden, occurs in the higher classes and in those who acquire the disease in their seventeenth to nineteenth year. The course may be chronic; the disease may last for years with chlorotic blood, menstrual, dyspeptic, muscular and nervous symptoms, with alternate exacerbation and amelioration, indicating an essential inactivity or hypoplasia of the blood-making organs; this form begins early in life in individuals with poor development of the bones, muscles, genitalia, mammæ, and adipose tissue; marriage, pregnancy, and iron often benefit these cases but they may last until the menopause.

Diagnosis.—It must be remembered that the diagnosis cannot be made by any single symptom but by the entire group of symptoms, both positive and negative, for no single symptom is pathognomonic. The diagnosis is based upon the *anæmia*, especially when there is disproportionate *reduction in the hæmoglobin*, when the color index is less than one, and the white cells are normal; a diagnosis, however, cannot be made from the blood findings alone. (Rhomberg has described exceptional cases in which the blood was normal.) Chlorosis is *infrequent before the twelfth* and *after the twenty-fifth year*, unless the attack is a relapse. Its *rapid development* and *periodic manifestation* are important features. Successful *treatment* with iron is practically diagnostic. *Secondary anæmia* is most difficult to differentiate (see DIFFERENTIATION under PERNICIOUS ANÆMIA); it is usually attended by some leukocytosis. Anæmia from intestinal parasites, hemorrhage from latent ulceration of the stomach or bowel, renal disease, early pregnancy in unmarried girls, the anæmia of heart disease, incipient brain disease, rheumatism, and lead and arsenic anæmias, must be considered. *Splenic anæmia*, especially in children, requires differentiation; cases of chlorosis with enlarged spleen are regarded as a transitional form between chlorosis and splenic anæmia proper. *Malignant disease* usually causes little diagnostic difficulty after careful examination. There is sometimes great difficulty in differentiation from incipient *tuberculosis*. Frequent physical examination and watching of the temperature usually establish the diagnosis.

Treatment.—*Prophylaxis* includes general hygiene and proper nourishment, in that development of adipose tissue to a reasonable extent is prophylactic. More albumin should be given to growing children than to adults, although fruit and vegetables, for their iron, and butter, cream and oils, rather than animal fats, should also be administered. Physical culture and trips to the country are valuable, and constipation should be prevented. Education should not be hurried; all children at this age are confined too much to the school-room; they should be kept children as long as possible. In society, and in the struggle for existence, young girls too often overtax their strength.

Specific Therapy.—Iron exists in the hæmoglobin of the blood cells, as an oxygen carrier, and is a food as well as a medicine; it is found in blood, meat, oatmeal, peas, spinach, beans, and the yolk of eggs. Milk is poor in iron.

Physiological Action of Iron.—(*a*) The tonus of the *nervous system* is increased, though an excess produces frontal headache from constipation; some persons show a peculiar intolerance of iron, as shown by the headache, the vertigo and the palpitation which it produces. (*b*) It affects the *circulation;* locally astringent, it coagulates albumin and contracts the bloodvessels. It increases the number of red disks and the amount of hæmoglobin. (*c*) Its action on the *gastro-intestinal tract* is as follows: iron stains the teeth because of the acid which it contains. Small doses stimulate digestion, improving, by their astringent action, the tone of the tissues. Hydrogen is generated and from it sulphureted hydrogen, which causes eructations. Small doses cause constipation, but large doses relax the bowels; diarrhœa also sometimes follows small doses. Sulphide of iron is found in the dark stools. (*d*) The amount of urine is increased. Iron is found in the urine in very small quantities. The kidneys may be irritated by very large doses. (*e*) In regard to its absorption, there is great conflict of opinion, and experiments are incomplete. There are four theories: (i) That iron is absorbed from the stomach and intestines as an albuminate; it reaches the blood and is deposited first in the spleen and then in the liver, whence it is then carried by the bloodvessels to the intestines for elimination. Although Schmiedeberg and Kobert insist that iron is not absorbed, there is strong clinical evidence that it is. (ii) That, being an astringent, it calls forth tonicity of intestinal tissues and stimulates absorption of other foods, which is probably also true. (iii) Bunge's theory is that in anæmia, hydrochloric acid is deficient and allows fermentation; that iron acts by neutralizing the alkaline sulphides (products of fermentation) and allows iron in albuminous food to be absorbed, as it thus escapes union with sulphides. This theory is now abandoned. (iv) On reaching the blood, it acts as a stimulant to the blood-making centres. (*f*) Elimination occurs by the intestines, the kidneys and liver.

Administration.—It may be advantageously combined with quinine, particularly when the spleen is enlarged; it may also be given with oxygen, normal salt transfusions, and massage; the patient should have rest and change of environment, and climate. Soluble preparations would seem to be better than insoluble ones, but Quevenne has shown that they are precipitated by the gastric juice. Headache may occur; it is obviated by using laxatives, bromides or ferrous salts, which derange digestion less than ferric salts. Iron is said to be best absorbed when some gastric catarrh exists (Brunton), hence some practitioners give large doses at first, to improve absorption. There are only 38 to 45 grains of iron in the body and the ordinary daily diet contains but one-sixth of a grain of iron, but the nucleo-albumin preparations, which are sufficient as food, are not sufficient as medication, because they do not stimulate the blood-making centres. The *rules for administration*, according to von Noorden are: (*a*) The doses should not be too small (Niemeyer and Immermann). (*b*) Every physician has his own method, therefore no one form especially excels any other. (*c*) The administration should be regular, for irregularity is injurious. Statements of the patient that she has previously taken iron should not be regarded, for its administration may have been irregular. (*d*) Administration should continue for six weeks to three months. In the first week the dose should be gradually increased; for three weeks the maximum dose should be given and for the last two weeks it should be gradually decreased. (*e*) If iron is not beneficial, it should not be repeated but other methods tried. (*f*) Relapses are less easy to treat than the first attack. (*g*) Idiosyncrasy, as gastro-intestinal disturbance, is only a relative contra-indication. The administration should then be subcutaneous. (*h*) At mineral springs, hygiene is more important than iron; the water should be taken on an empty stomach, for the gas promotes absorption of iron. When bottled it is precipitated as a carbonate.

Iron may be given in the *following forms: Ferrum reductum* (reduced iron) which is iron gray (if it is black, it is impure), and tasteless; it is least astringent, but may cause eructation; gr. j. (See formula for reduced iron given under THERAPY OF VALVULAR DISEASE, page 428). *Pilula ferri carbonatis*, a ferrous salt nearly free from astringency; grs. ij to v. *Ferri sulphas* (green vitriol), a ferrous salt which is very astringent, whence its use in chronic diarrhœa. It stimulates digestion. In large quantities it is toxic, causing vomiting or diarrhœa. Smart considers it the best form: gr. ij should be given with one dram of magnesium sulphate p.c. Ferri sulphas exsiccatus gr. ss to ij (gm. 0.03 to 0.12) may also be given. Liquor ferri et ammonii acetatis (*Basham's mixture*) is a vascular tonic and diuretic; it is excellent for chlorosis in the obese; ʒj to iv should be given after meals, well diluted. *Tinctura ferri chloridi* is the chloride of iron plus hydrochloric acid and alcohol, which develop hydrochloric ether. It is yellow-red, astringent, somewhat corrosive and diuretic, increasing the amount and frequency of micturition. It is used to best advantage when the tongue is white, flabby, and indented. In weak, anæmic girls, with vomiting, neuralgia and epigastric pains, full doses should be given; when poorly tolerated, one drop in a full glass of water should be given; this is more beneficial than most chalybeate waters. *Syrupus ferri iodidi* is transparent and has a greenish color; iron and iodine given separately are usually more beneficial, but the syrup may be given in doses of 30 to 40 drops in adults and of 5 to 10 in children. *Ferri citras* in doses of gr. v, is efficacious in children and in persons having delicate stomachs. Ferri phosphas solubilis, gr. v, is almost free from astringency and taste. The *organic preparations* are generally useless. *Ovoferrin*, however, is excellent. The author thoroughly agrees with the conclusions of R. W. Wilcox: (*a*) It is the exact chemical form for assimilation. (*b*) It is not dissociated by the gastric juice into the astringent iron chloride. (*c*) It does not interfere with digestion, but has a noticeable effect in increasing the appetite. (*d*) It does not irritate the alimentary canal. (*e*) Its effect in increasing the red corpuscles and the hæmoglobin is constant and progressive. (*f*) It has, aside from its blood-forming effect, a demonstrable tonic and reconstructive action, which can be explained only by a stimulating influence upon metabolism.

Arsenic may be given with the iron or in its place; it acts on the blood-making centres; it should be given as in pernicious anæmia, uninterruptedly, with gradually increasing doses, sustained doses, and then decreasing doses, for a period of four to six weeks.

High altitude increases the number of red cells and the amount of hæmoglobin. Phlebotomy, sweating and organotherapy (bone marrow, splenic and ovarian extracts), have been suggested.

. **Dietetic Treatment.**—Regular meals should be given at three-hour intervals. The amount of *albumin* should be increased, even in the obese. The heavy English breakfast with meat is the best one, because the albumins are absorbed and taken up by the tissues more rapidly than are fats and carbohydrates; according to von Noorden two to four ounces of meat should be given with some bread and a cup of coffee. In administering *milk*, care should be observed lest the appetite be spoiled, lest too much fat be deposited, and the stomach suffer atony. Equal parts of milk and cream should be given. Milk should be avoided in the obese. *Alcohol* is indicated in thin, rather than in obese individuals. Patients should always rest before and after eating. The patient should retire early. In thin subjects the diet should be concentrated, consisting of albumin, vegetables, fats, alcohol and milk at breakfast and at bed time, but without increase of the carbohydrates. Iron produces better results in such cases after limiting the amount of water to about one quart; the latter procedure of itself is said to be beneficial in chlorosis. Sweats may produce the same result. When there is much gastric disturbance, eight meals may be given at two-hour intervals, and fluid and solid meals should be alternated. Gastric disturbance disappears after administration of iron and increase in weight. Hydrochloric acid, strychnia and belladonna are helpful.

Exercise may be prescribed in convalescence, but in moderate and severe cases the patient should rest in bed. *Hydrotherapy,* especially by cool rubbings early in the morning, is advantageous. Cold baths are to be avoided, because they produce chills, insomnia, and digestive disturbances. Marriage may help mild cases, but produces injurious results in the severe type.

(B). PERNICIOUS ANÆMIA.

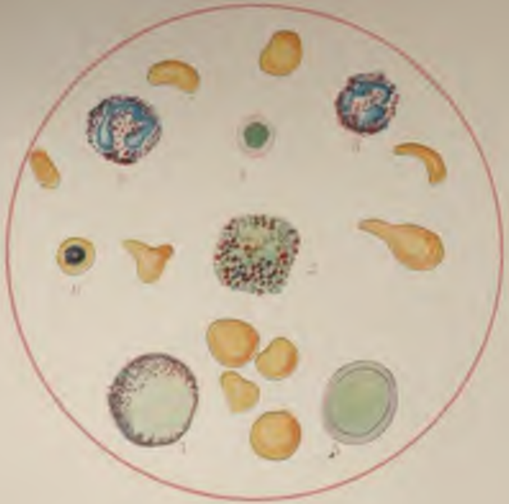
Definition.—A primary anæmia, in which are observed progressive diminution of the red cells, with changes in their form and color, and reduction in the hæmoglobin, without changes in the white cells; this form of anæmia is due to hæmolysis and to defective hæmogenesis; it is characterized by pallor, adynamia, œdema and cardiac symptoms, and usually results in death from vomiting, diarrhœa, hemorrhages, fever and exhaustion. It was described by Andral (1826) and Channing of Boston (1832); Addison gave the best early description, and Biermer later drew attention to the subject.

Etiology.—The disease occurs in 0.2 per cent. of cases, especially in the *third and fourth decades* of life, although also observed in other periods, as in nurslings, and very late in life. Baginsky could collect but 16 cases in children. Sixty per cent. occurs in males. Although called a primary or essential anæmia, some cases are undoubtedly secondary which may properly be included under this head, because the symptom complex is the same. Bothriocephalus, and anchylostoma anæmia present identical blood findings, symptoms, and pathology; not merely the parasite but also its toxins are causative. Pregnancy and the puerperium, in certain localities at least, are causative. Syphilis, poor hygiene, infectious diseases, as typhoid or malaria, and diarrhœa, vomiting, repeated loss of blood, and oral or gastro-intestinal sepsis have been considered factors. The precise cause of the blood destruction (hæmolysis) is not known.

Symptoms.—1. The Blood Changes (Sorenson, 1874).—The amount is decreased, several punctures often being necessary to obtain enough for the blood-count; it is often watery, or dark, or amber-colored in some instances. The specific gravity is low, even 1,027; the dry substance is reduced, even to 9 or 10 per cent. of the normal. There is an increase in the albumin in the individual red cell (hyperalbuminæmia rubra, von Jaksch). The serum does not contain free hæmoglobin, as has been asserted by Limbeck. If injected into normal individuals, the serum has a globulicidal action. In contrast to chlorosis and secondary anæmias, the *coagulability* of the blood is *decreased,* and the blood may remain fluid for some time after death; Hayem holds that the rarity of blood plates and lack of retractility of the clot are the most characteristic features of pernicious anæmia. The *hæmoglobin* is decreased; it is rarely more than 50 per cent. of the normal, and may be reduced to 10 per cent. In some instances severe symptoms may be present, with 65 to 70 per cent. hæmoglobin. In contrast to other anæmias, the *hæmoglobin per corpuscle* is proportionately *increased, i.e.,* the color-index is more than one (even 1.75). Laache explains the phenomenon in that certain red cells, especially giant forms, contain a *large compensatory individual amount* of hæmoglobin, which is also true of the microcytes, but less so of normal-sized red cells. The relative increase of the hæmoglobin per corpuscle is thus best explained, and Limbeck's assumption of a hæmoglobinæmia is not generally admitted. Schmidt, Ponfick and others of the Dorpat school, maintain that the free hæmoglobin, acting on the white cells, produces a fibrin ferment, which causes fever, hemorrhage and capillary embolism. The *red blood corpuscles* are greatly reduced in number, the average being 1,200,000. Plate XIII. Patients often die when this figure is reached. Lower counts have been noted, as 143,000 (Quincke). *Poikilocytosis* is marked, the irregularity in form and size of the corpuscles being great; great deformities, as Litten's horse-shoe or pessary-shaped corpuscles are noted. The individual *red corpuscle* is *above the average size, i.e.,* 11 to 13, even 16 to 18μ instead of 5 to 8μ, the highest normal measurement. *Megalocytes* may be observed in number, with less marked or absent umbilication. Polychromatism is frequent. Megalocytes in pernicious anæmia are evidence of megaloblastic degeneration of the bone-marrow (but have been held as evidence of regeneration). The varieties of nucleated and non-nucleated erythrocytes are depicted in Fig. VIII, Plate XIV. *Megaloblasts* are found but rarely in large numbers, seldom more than six or eight being seen in a single preparation (Ehrlich); in rare cases they may be abundant, as just before death, whence their generally ominous significance; they may be absent for months or weeks at a time. *Normoblasts* occur in relatively light cases, especially in the "blood crises" of von Noorden, but are not numerous. Microblasts are frequently absent and are not umbilicated; their teleologic significance lies in the increase of the respiratory surface of cells thus afforded. Cell divisions are found. (Plate XIII, Fig. 3.) Granular deposits (staining with basophilic dyes), in the protoplasm are noted, as in malaria, cachexia, leukæmia and suppuration; according to Ehrlich, they are not due to polychromatophile degeneration but to nuclear degeneration. The cells may stain only at their margins. Decreased formation of rouleaux is observed as well as amœboid movement of the red cells. The *white corpuscles* are decreased absolutely, the lowest record being 1,500; the polymorphonuclears are decreased. Leukocytosis is a distinct complication, is often ominous though not necessarily fatal, and indicates inflammation, suppuration, or pneumonia. Apart from complications, increase in the leukocytes may take the form of a lymphocytosis, the lymphocytes being relatively increased, even to 62 per cent. of the total white cells. The leukocytes are readily increased in children in any variety of anæmia. Marked decrease of the eosinophiles is an unfavorable sign according to Neusser. Pernicious anæmia due to parasites shows increase in the eosinophiles from

PLATE XIII.

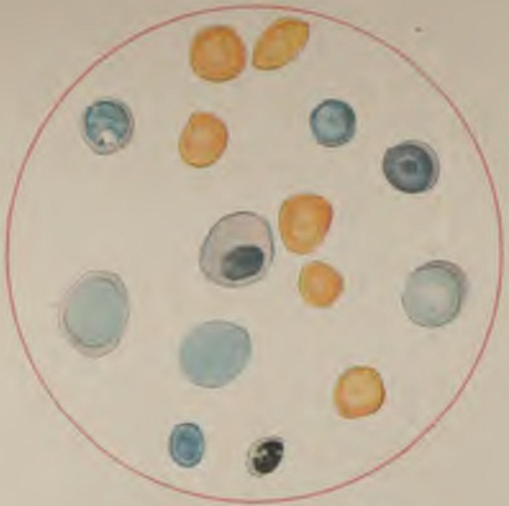
FIG. 1.



Myelogenous Leukæmia.

1. myelocyte; 2, eosinophile myelocyte; 3, 3, eosinophile polymorphonuclears; 4, small and 5, large lymphocyte; 6, nucleated red cell.

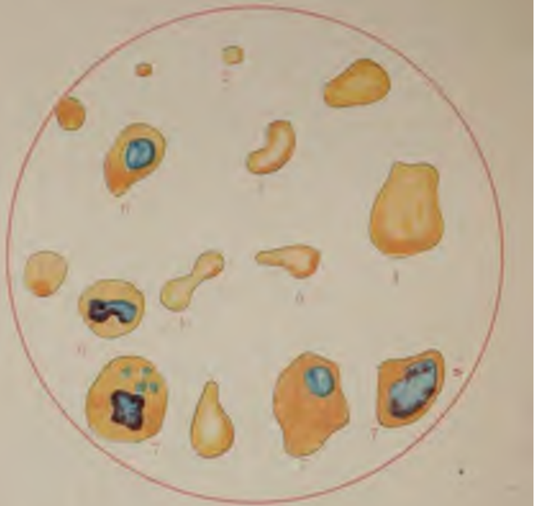
FIG. 2.



Lymphæmia.

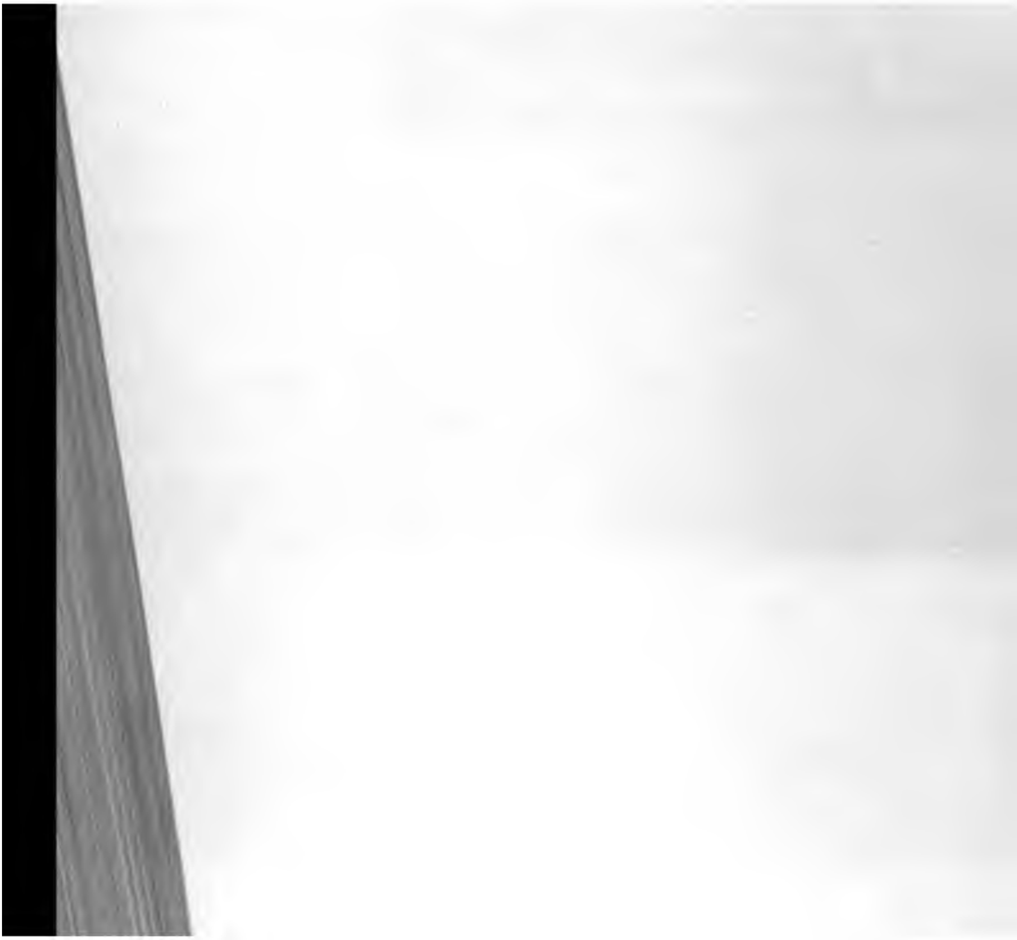
Large and small lymphocytes with various intensity in stain of nuclear chromatin.

FIG. 3.



Pernicious Anæmia.

1. megalocyte; 2, microcyte; 3, 4, 5, malformed erythrocytes; 6, 6, normoblasts; 7, 7, 7, megaloblasts.



1 to 4 per cent. (normal) to 6, 36 and even 50 per cent. (Walker). (See Plate XIV, Fig V).

2. **Subjective Symptoms.**—The most marked are general weakness, muscular adynamia on exertion, vertigo, palpitation, dyspnœa and the ordinary signs of anæmia.

3. **Skin.**—The skin presents a peculiar pallor, often of a lemon color, which is somewhat characteristic. Slight icterus is not infrequent. The skin, usually dry, may become drenched upon exertion. Œdema is invariable, occurring especially about the ankles and eyelids. It is usually not marked, but is obstinate and may produce pronounced general anasarca; it is due to vascular degeneration and permeability. Hemorrhages, usually punctate, are frequent in the skin and mucosæ. Large hemorrhages are infrequent. Pigmentation is sometimes as marked as in Addison's disease. The author once saw herpes zoster, and once herpes along the crural nerve. Sometimes before death the skin emits a foul, cadaverous odor (Eichhorst).

4. **Metabolism.**—The nutrition is generally good and the panniculus adiposus is maintained, because tissue waste is lessened and water is retained in the tissues. The weight and strength may finally decline with persistent vomiting and diarrhœa. Opinions differ as to the destruction of the albumins. In the *urine*, indican, putrescin and cadaverin are found. Hæmaturia and glycosuria are rare. Albuminuria, due to tubular degeneration, is frequent. Albumosuria is sometimes noted; urobilin is frequently increased, and this sometimes imparts a dark color to the urine and indicates hæmolysis. The uric acid is sometimes increased. Leucin is almost constant and tyrosin not infrequent. The chlorides are decreased. Acetone and diacetone are sometimes found. The iron in the urine is sometimes increased, and the phosphates are greatly increased. *Fever*, present in 80 per cent. of cases, bears no constant relation to the severity of the disease. It may rise considerably; the temperature may fall below normal in the ante-mortem coma. The type is irregular. Its cause is ill-understood; it is probably due to the production of a ferment from the destroyed red cells, or according to Schmidt, Ponfick and others, from a fibrin ferment formed from the leukocytes (*v.s.*).

5. **The Circulation.**—Palpitation, precordial anxiety and dyspnœa are observed, but sometimes only on exertion or excitement. The heart impulse is irregular, and fallaciously strong to the palpating hand. Its outlines are usually normal, save that some dilatation of he right heart may occur or hydrops pericardii develop. Frequently blowing systolic murmur is heard, usually with a clear diastolic ne; the murmur is rarely diastolic. The second pulmonic sound is riable, sometimes accentuated. No parallelism exists between the gree of anæmia and the loudness of the murmur. Leube reports umber of cases of chlorosis and pernicious anæmia in which there as relative tricuspid insufficiency; the positive venous pulse was esent but no cyanosis. A venous hum in the neck, carotid pulsation d thrills, a loud, clear systolic tone and murmurs and double tones

over the peripheral arteries are often present. The *pulse* tension is decreased, and its *rate increased*, being rarely below 80 and usually ranging between 90 and 120. *Anatomically* the heart shows vestiges of hemorrhage, degeneration and atrophy. The *fatty*, "tabby-cat heart" is almost constant, though absent in some cases (Litten). Degeneration in the smaller vessels has been noted, especially in those of the brain.

6. Digestive Tract.—Anorexia, distaste especially for meats, gastric achylia, pain, and sensitiveness throughout the alimentary tract ar noted. The tongue and gums are sensitive, and circumscribed o punctate hemorrhages are often noted in them. Pain in the mout and œsophagus is sometimes due to the administration of arsenic The gums may ulcerate, erythema bullosum and other eruptions ma occur. *Vomiting* is frequent in various stages of the disease, and i often continuous or incoercible; the vomitus consists of food, greenis mucus, and sometimes traces of blood. Epigastric pulsation, modera abdominal distention and decreased or suppressed secretion of hydr chloric acid are observed. The author once saw expansile abdomin pulsation which was wrongly diagnosticated as aneurysm. The glan in the gastro-intestinal tract may atrophy, which probably toxemic co dition is coördinate with and not causal of, pernicious anæmia. T mesenteric glands are not infrequently enlarged. Constipation or oft *paroxysmal diarrhœa* may occur. The stools are not characteristi although sometimes acholic from poor absorption of fat. Parasit or their eggs may be noted. Leucin and tyrosin crystals are frequent an are caused by diarrhœa, but are not in any sense characteristic of th disease. Fermentation in the bowel (with indican in the urine some œdema, hemorrhage and follicular swelling in the bowel ar frequently observed. Some ascites is found in the terminal stage o the disease. A deposit of iron (siderosis) in the liver, in the outer and middle zones of its lobules, also in the spleen, marrow, kidneys and lymphatic glands, is usual, and results from hæmolysis; it is very unusual in other anæmias. There is most hepatic siderosis in acute cases. The liver is often slightly swollen and tender.

7. Blood-making Organs.—The *lymph glands* are not usually enlarged. The prevertebral hæmolymph glands have been carefully studied by Warthin, who found their sinuses dilated and evidences of hæmolysis in their tissue which was shown by increased phagocytosis and sometimes hyperplasia of the glands; Warthin holds that pernicious anæmia is a hæmolytic disease caused by an unknown poison which stimulates the phagocytes of the spleen, lymph nodes and hæmolymph glands to increased hæmolysis. The *bones are tender*, especially over the sternum and tibia and sometimes painful, and their fatty marrow may become lymphoid; this is not characteristic of the disease, for lymphoid transformation may occur in other forms of anæmia in cachexia or in fevers; in pernicious anæmia the red cells of the marrow are abnormally large, abnormally dark from increase of the moglobin, and are oval rather than round. The bone changes were first described by Wood, Pepper and Tyson (1875). The *spleen* i not enlarged, save from accidental causes.

8. **Sexual Sphere.**—The menses cease, otherwise no characteristic changes are observed.

9. **The Nervous System.**—The psychical functions are decreased, the memory is weak, and psychoses, delirium, hallucinations, mania and intractable insomnia may develop, either temporarily or antecedent to the terminal coma. Sometimes improvement is noted after severe mental symptoms or coma, compared by Laache and von Noorden to a crisis. The pupils are rather wide, and sometimes their reflexes disappear. Paresis, apoplectiform attacks, cramps, convulsions, paræsthesia, aphasia, vertigo, or in women and children the hydrencephaloid state may develop. The *cord* is more frequently involved than the brain. The weakness may amount to paresis, not due simply to muscular adynamia but to spinal changes. The picture may resemble tabes as concerns the eyes, patellar reflexes, ataxia, bladder and rectal changes, pains, anæsthesia and paræsthesia; or the symptoms of spastic spinal paralysis prevail, as increased reflexes and spastic paresis. Frank Billings in 36 personal cases finds two groups, one in which the nervous symptoms are slight, and a second in which they are so conspicuous as to even overshadow the anæmia itself. He found early paræsthesia in all his cases, usually in the hands and feet, sometimes in the mouth; sometimes hyperæsthesia or disturbance of the temperature sense; 10 cases were spastic and atactic; three had paraplegia with abolished patellar reflexes, involuntaries, œdema and bed-sores; the sexual function was decreased in males, and amenorrhœa was regular in women; histologically he found a sclerosis of the posterior and lateral columns, which was most marked in the lower cervical and upper dorsal segments, was not always diffuse but sometimes focal, and involved chiefly the white substance, but did not invade the entire neuron, and was probably due to a toxin.

The peripheral nerves present few symptoms. The discrepancy between the anatomical findings and the clinical symptoms is remarkable, *i.e.*, there may be many symptoms with few autopsy findings, and the converse is also true; consequently it is probable that the spinal and nervous symptoms are less sequences of anæmia than coördinate toxic manifestations. Anatomically, capillary hemorrhages and disseminated foci of softening may be observed in the brain and meninges. Degeneration is found in the upper part of the cord, r possibly throughout its length. The white substance suffers more xtensively than the gray. Small foci of degeneration with some condary connective tissue growth, occur about the bloodvessels, hich foci may fuse into plaques of small size with secondary degenation, hence the symptom complex of a systemic disease; the changes ually occur in the posterior column, which rarely wholly escapes. e bloodvessels may suffer degeneration, widening, thickening or leed obliteration.

In *the eyes,* the general symptoms of anæmia occur; the patie thinks he sees floating spots; hemorrhages in the retina are almo constant; occurring on a pale background, with tortuous veins, an a strong light reflex from the vessels, the hemorrhages form a strikin picture (see Plate VI, Fig. 9). They are punctate or larger and the resorption is infrequent. The other senses are obtunded; labyrinthi hemorrhage with vertigo is recorded.

Clinical Course and Prognosis.—The pallor and weakness whic are first observed increase insidiously, and later dyspnœa, weak hear action, œdema, complete anorexia, langor and violent gastro-intestin disturbance and finally capillary hemorrhages and fever develop this vomiting and diarrhœa may at times resemble acute poisoning In other instances remissions may occur with treatment or independently of it, perhaps after violent gastro-enteric manifestations. The symptoms may partially or wholly disappear, as in Ehrlich's case, where the red cells in seventeen days rose from 1,340,000 to 4,115,000. According to Laache, in simple anæmia the red corpuscles gain more rapidly than the hæmoglobin, but in pernicious anæmia, improvement increases both equally. The disease with remissions may cover a period of years, the longest duration being ten years. Death usually occurs within half a year.

The *prognosis* is usually poor when megaloblasts are present in numbers or when the color index is over one. In some cases the blood is rapidly regenerated; and 200,000 new red cells may appear in the blood within a day.

Cabot's description of the disease recognizes *two forms.*

THE SEVERE, RAPIDLY FATAL TYPE	—AND THE LESS SEVERE, SLOW TYPE.
(*a*) Extreme progressive anæmia.	Remissions.
(*b*) Color index high.	Normal or low.
(*c*) Red cells increased in size.	Normal.
(*d*) Degenerative changes.	None.
(*e*) Many megaloblasts.	Many normoblasts, few megaloblasts.
(*f*) Lymphocytosis.	Normal ratio of the various white cells.

Death usually occurs in coma or possibly from cerebral hemorrhage, or other rare complications, as valvular disease, gastric carcinoma, tuberculosis, nephritis, sepsis, sarcoma, or leukæmia. Pye-Smith collected 20 cases of recovery. According to Schauman, the mortality in the parasitic form is 17 per cent.

Diagnosis.—No single finding is absolutely characteristic, though the pallor, apathy, adynamia, œdema, weak heart, retinal hemorrhages, maintained nutrition, and the blood findings are highly suggestive. The yellow color is very often described as icterus and mistaken for it. The increased diameter of the red cells may also be found in icterus, and Ewing states no importance should be attached to the increase of diameter, unless 33 per cent. of the red disks show it. Megaloblasts are highly suggestive. The diagnosis must be made from the negative, as well as from the positive findings.

erentiation.—(*a*) From *metastatic bone-tumors*, in which the white re increased (not merely the polymorphonuclear but the neu- le and eosinophile marrow cells); (*b*) from *parasitic anæmia*, for the stools should be examined for parasites and their eggs; (*c*) if dominant, may suggest typhoid or meningitis; acute endocarditis e simulated by the fever, anæmia, heart murmurs and cardiac ciency of pernicious anæmia; (*d*) from *latent carcinoma*, the al cachexia, anorexia, nausea, vomiting, hæmatemesis, and de- or absence of hydrochloric acid may suggest gastric carcinoma, e blood examination with thorough physical examination usually nines the point; (*e*) *spinal disease*, Addison's disease, and hemor- : pleuritis, are usually excluded with ease; (*f*) *from other anæmias*. ollowing is constructed after Cabot's table:

	PERNICIOUS ANÆMIA.	CHLOROSIS.	SECONDARY ANÆMIA.
s Number.	Average number 1,000,000.	Rarely under 2,000,000.	Findings as in chlorosis. May be 1,000,000 or less, though rarely.
hape.	Poikilocytosis great, sausage-shape, etc.	Somewhat deformed.	
ize.	Average increased, some dark.	Diminished, light colored.	Various, not increased.
oglobin.	Increased per corpuscle yet many Ponfick's shadow corpuscles.	Reduced per corpuscle.	
aining.	Polychromatophile cells.		
uclei.	Micro-, megalo-, normoblasts. Megalo- more numerous than normoblasts and constitute majority of nucleated R. B. C.	Megaloblasts rare though recorded. Normoblasts more than megaloblasts.	Normoblasts far more than megaloblasts in malignant diseases. Normoblasts common (more than in chlorosis).
uleaux.	Not present, or decreased.		
r index.	High color index (not in chronic nor mild cases).	Index less than 1: always low: lower than in secondary anæmias.	Relatively low (this rule does not hold positively).
kocytes.	W. B. C. decreased, except in blood crises, hemorrhage, suppuration.	Usually normal.	Polymorphonuclear leukocytosis in malignant disease, when R. B. C. are low in number, *e. g.* one million, speaks for secondary anæmia; positive of more value than negative findings (*i. e.* no leukocytosis).
hocytes.	Lymphocytosis (relative).	Lymphocytosis.	Rarely increased (no decrease).
locytes.	Small number only.	Rare; less than in pernicious anæmia.	Myelocytes very rare.
eukocytes.	Rarely increased. Usually decreased.	Decreased.	Usually increased.
ulation.	Slow.	Rapid.	Often rapid.

Aplastic anæmia may resemble pernicious anæmia in its low blood count, reduced hæmoglobin or even its color index. It was named by Ehrlich because the bone marrow was white or yellow and aplastic. In the blood there are no evidences of regeneration such as nucleated red cells. The white cells may number only 200 per c.mm. and 80 to 90 per cent. of them are lymphocytes. There may be hemorrhages, fever, or hyperplasia of the spleen and lymph glands.

Leukanæmia (see DIFFERENTIATION OF LEUKÆMIA).

Treatment.—**1. Hygienic.**—The patient should have absolute rest in bed, and if possible, should be in a warm climate. An excessive nitrogenous diet is usually given, which is unwarranted because it increases tissue waste; besides, patients have an instinctive disinclination to nitrogenous foods, and albumins frequently produce diarrhœa. A vegetable diet is better tolerated, with bread, zwieback, beef tea, peptones, milk, honey, or porridge. Nutrient enemata are indicated in the gastric crises. In pregnant women, labor should not be induced.

2. Hæmatinic.—(*a*) *Transfusion* of defibrinated human blood has been practised (Quincke, Hoffman, Landos), though it is seldom beneficial. Human blood may be transfused *in toto* (Ziemssen). (*b*) Lépine recommends the physiological *salt solution*, based on Maragliano's findings that the serum in cases of pernicious anæmia has a globulicidal action on the red blood cells. The serum destroys the hæmoglobin in pernicious anæmia (the blood of a patient clears in a test tube on standing). Salt solution should be given frequently, and acts temporarily on the serum. (*c*) Phosphorus is without value and iron is often injurious. (*d*) *Arsenic*, recommended by Bramwell, is said to have cured the disease, though most cases have not been fol- lowed out thoroughly; it is, nevertheless, the best remedy known. It probably acts by irritating the blood-forming centres. Birsch-Hirs feld explains its action by assuming that it operates in some way upon the serum, which in this disease, destroys the corpuscles, while Litten holds that it increases the number of white cells.

PHYSIOLOGICAL ACTION OF ARSENIC.—(*a*) The smallest dose mere increases the appetite. (*b*) Small doses stimulate the appetite by ga tric irritation, by increasing secretion and stimulating peristalsi tissue waste and elimination of nitrogen is lessened. It is an alter tive, increases the muscular strength and endurance, improves t quality of the blood, the complexion, sexual powers, and lung capacit (*c*) Continuous administration produces constitutional effects, as puff ness of the eyes, which is first seen in the morning (Wier Mitchell this may be followed by general œdema, due to vascular and some times renal alteration. Administration should be stopped when puff ness is observed. (*d*) Large doses result in anorexia, nausea, diarrhœ abdominal uneasiness or colic, and sympathetic headache.

The circulation is stimulated by small doses. Arsenic is absorbed by the bloodvessels, and possibly unites with the red cells. Full doses lessen the force and rate of the heart, for they act directly upon the heart muscle. Arterial pressure is reduced by peripheral vasomotor paralysis. Respiration and digestion are stimulated by small and depressed by large doses, although some deny the stimulant action of any dose. Elimination is very rapid, and is effected mostly by the kidneys and intestinal mucosa, but also by the skin, bile, lungs, saliva, and mother's milk.

ADMINISTRATION.—Children tolerate large doses, though the aged stand the drug poorly, and their digestion suffers; after the fifth year, adult doses are given; girls tolerate the drug better than boys. When susceptibility is marked, a few drops of laudanum may be added, but its administration should always be stopped when (*a*) the eyes become puffy, or (*b*) itch, and (*c*) the tongue becomes furred. It may be given in the following forms:—(i) *Acidum arsenosum* (now officinal as arseni trioxidum) gr. $\frac{1}{30}$ p.c., with ext. glycyrrhizæ and (black pepper) piper āā gr. iv ("Asiatic pill"). (See formula under TREATMENT OF VALVULAR DISEASE, page 428). (ii) *Liquor potassii arsenitis* (Fowler's solution, 1786); for hypodermic use it must be sterilized, because fungi are likely to grow in it. Even with great care abscess may develop. Minims v equal gr. $\frac{1}{20}$ of ac. arsenosum. Treatment should commence with min. iij and each day the dose should be increased a drop until minims x or xij are given; rarely can larger doses (♏xv–xxv) be given. (iii) The cacodylate of soda (Clin) pills (āā gr. $\frac{1}{6}$) may be given one to five times daily.

3. Symptomatic Treatment.—This includes opiates for diarrhœa, cardiants for weak heart, and dilute HCl for gastric achylia.

(C). SECONDARY ANÆMIA.

1. Acute Post-Hemorrhagic Anæmia.

Etiology.—It may result (*a*) from trauma of large vessels; (*b*) from post-partum hemorrhage, abortion, or tubal pregnancy; (*c*) from hæmoptysis, aneurysm, cardiac disease, or tuberculosis; or from internal aneurysmal ruptures, as into the pleura; (*d*) from carcinomatous ulcerations in the uterus, gastro-intestinal tract, etc.; (*e*) from round ulcer of the stomach or duodenum; (*f*) from varicosities, as in the leg, stomach, or œsophagus, and liver cirrhosis; (*g*) from conditions of the blood, as epistaxis in typhoid; from pancreatitis, hemorrhagic states, or constitutional diseases.

Symptoms.—1. *The blood* is (*a*) *hydræmic*, the loss of fluid being supplied by the tissues, and its specific gravity is lowered. (*b*) The *red disks* are reduced to 50 per cent., or even to 20 per cent., are often irregular in contour (poikilocytosis), pale and poor in hæmoglobin; their diameter may be increased; generally there are some normoblasts. (*c*) The *hæmoglobin* is lessened even more than the red cells. (*d*) The leukocytes at first show a moderate increase; the polymorphonuclears are decreased, and the mononuclears increased. In sudden anæmia with rapidly resulting death, the red cells are seldom nucleated, and there is no leukocytosis, because the bone marrow makes no effort to regenerate the blood. (See Plate XIV, Fig. 3.)

2. There is *marked anæmia* of the *skin and mucous membranes*. Some œdema results from chemical changes in the blood, or malnutrition and increased permeability of the vessel walls. The pulse is weak, and becomes frequent with the least exertion. Nevertheless, the vasomotor system very readily accommodates itself to a loss of one-quarter of the blood volume; a greater loss produces cardiac weakness. Sometimes the heart action seems more violent and respiration deeper and more rapid than normal.

3. The digestive glands are less active.

4. *Albuminuria* develops in a few hours after a large hemorrhage. No obvious microscopic change in the renal epithelium is detected, whence it seems that albuminuria is due, either to undue permeability of the renal vessels, or possibly, to albumins absorbed from the tissues.

5. The *nervous* and *eye symptoms* may be marked. Amblyopia and amaurosis were known to Hippocrates; they are at first functional, but usually 8 to 10 days later degeneration or inflammation of the retina develops; as the blood pressure rises, retinal hemorrhages may follow. The patient often sees spots before the eyes. Occasional nervous manifestations include aphasia, or vertigo; delirium and hallucinations are chiefly terminal events; in some cases, lasting psychoses develop.

6. The *autopsy findings* in fatal cases are fatty degeneration, particularly of the heart, endothelium of the vessels, liver, brain, stomach, and kidney cells, which is usually caused by imperfect oxygenation. Hemorrhages in the uterus, gums, and retina, are due to impaired nutrition of the vessel walls. The bone marrow changes from a yellow to a red color, and abounds in normoblasts.

Prognosis.—The patient may survive the loss of more than one-third or one-half of the blood; Hayem reports recovery after the number of red cells fell to 550,000. Men stand hemorrhage better than women and young children, though the loss of a pint may cause death in arteriosclerotics. Recovery is more rapid in acute than chronic hemorrhage. In moderately severe hemorrhage, recovery may be practically complete within a week; water and albumin are readily yielded to the blood by the tissues; the red cells regenerate more slowly, and the hæmoglobin is the last element to reach normal. Increase in the coagulability of the blood is a favorable prognostic. According to Bierfreund, a loss of 10 to 15 per cent. of the blood is restored in eight days; of 16 to 20 per cent., in twenty days; of 21 to 25 per cent., in twenty-two days, and of more than 25 per cent., in twenty-nine days or more.

Treatment.—This has been considered under gastric ulcer, and intestinal hemorrhage in typhoid; the essentials are absolute rest, morphine to insure quiet, and salt infusions only in case of extreme cardiac failure.

Fig I

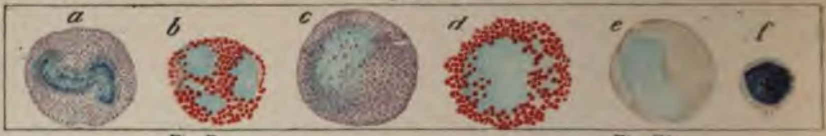


Fig II.

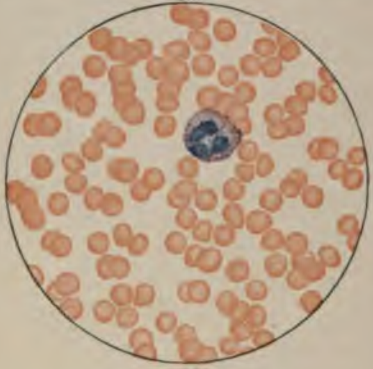


Fig III

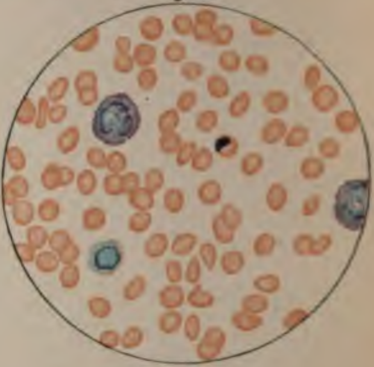


Fig IV.

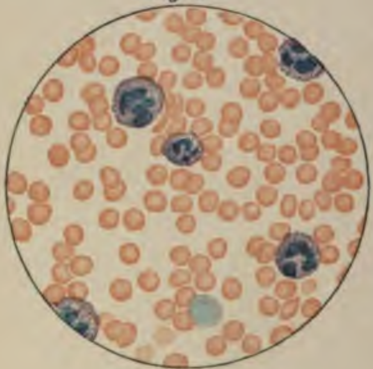


Fig.V.

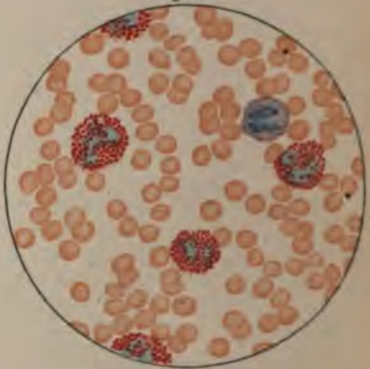


Fig VI.

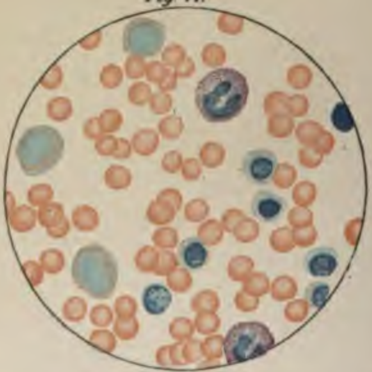


Fig.VII.

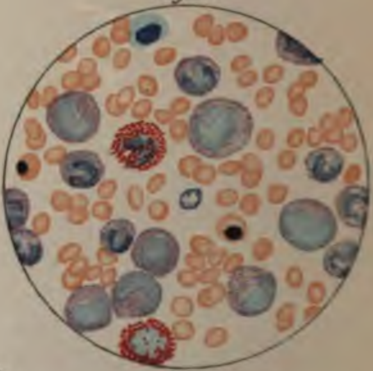
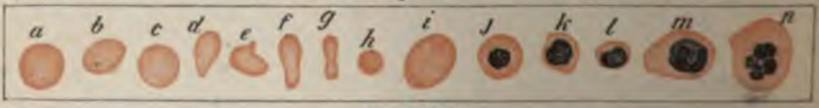


Fig.VIII.



DRAWN BY J.H.E. CHASE

PLATE XIV

BLOOD

[illegible]

Prepared by Dr. [illegible]

Fig. I. TYPES OF LEUCOCYTES

a. Polymorphonuclear Neutrophile. b. Polymorphonuclear Eosinophile. c. [illegible] (Neutrophile). d. Eosinophilic Myelocyte. e. Large Lymphocyte (large [illegible]). f. Small Lymphocyte (small Mononuclear).

Fig. II. NORMAL BLOOD

[illegible]

Fig. III. ANÆMIA POST-HÆMORRHAGIC (Secondary)

The reds are fewer than normal and are [illegible] somewhat irregular in form. The normoblast is seen in the field [illegible] small lymphocyte showing a marked post-hæmorrhagic anæmia with leucocytosis.

Fig. IV. LEUCOCYTOSIS INFLAMMATORY

The reds are normal. A marked leucocytosis is shown with [illegible] neutrophiles and one small lymphocyte. This illustration may also serve the purpose of showing the leucocytosis of malignant tumor.

Fig. V. TRICHINOSIS

A marked leucocytosis is shown, consisting of an eosinophilia.

Fig. VI. LYMPHATIC LEUKÆMIA

Slight anæmia. A large relative and absolute increase of the lymphocytes (chiefly the small lymphocytes) is shown.

Fig. VII. SPLENO-MYELOGENOUS LEUKÆMIA

The reds show a secondary anæmia. Two normoblasts are shown. The leucocytosis is massive. Twenty leucocytes are shown, consisting of nine neutrophiles, seven myelocytes, two small lymphocytes, one eosinophile (polymorphonuclear) and one eosinophile myelocyte. Note the polymorphous condition of the leucocytes, i.e., their variations from the typical in size and form.

Fig. VIII. VARIETIES OF RED CORPUSCLES.

a. Normal Red Corpuscle (normocyte). *b, c.* Anæmic Red Corpuscles. *d-g.* Poikilocytes. *h.* Microcyte. *i.* Megalocyte. *j-n.* Nucleated Red Corpuscles. *j, k.* Normoblasts. *l.* Microblast. *m, n.* Megaloblasts.

2. Chronic Secondary Anæmia.

Etiology.—Its possible causes are numerous; the chief types are (*a*) *hemorrhages* like those listed under acute post-hemorrhagic anæmia, but frequent and persistent, as chronic hemorrhoidal bleeding. (*b*) *Poor hygiene;* overwork, long hours in poorly-ventilated, sunless offices or factories; insufficient food, or food poor in iron; or worry. (*c*) *Exhausting discharges, or loss of albumin,* as frequent pregnancies with prolonged lactation, albuminuria, respiratory or alimentary catarrh, or protracted suppuration, with its attendant loss of nuclein. (*d*) *Toxæmias;* acute infections, as typhoid, diphtheria, rheumatism, or malaria, in which the activity of blood-making centres is diminished, and food is poorly assimilated; chronic infections, as syphilis and tuberculosis; gastro-intestinal, and possibly other intoxications; neoplasms acting as mechanical obstructions (as those of the œsophagus, or pylorus), affecting sometimes, the bone marrow by metastases deposited there, sometimes causing repeated losses of blood or elaborating toxins, which break down the tissue albumins; in neoplasms the reduction of the blood in red cells and hæmoglobin averages 55 to 66 er cent. of normal; parasites (oxyuris, ascaris, echinococcus, etc.); nd poisons (alcohol, lead, arsenic, or phosphorus) may produce næmia.

Symptoms.—The *blood is pale,* and its specific gravity is lowered. s a rule the red disks and hæmoglobin are reduced in rather close arallelism, though occasional exceptions occur. The hæmoglobin ay fall to 18 or 14 per cent. of normal, and the red cells may, in rare stances, approach the count of pernicious anæmia. The red cells ain unequally, and their umbilication is clearer, so that the hæmo- bin may even appear as a slight peripheral ring. They show less ikilocytosis than in acute post-hemorrhagic anæmia, and many of em are undersized. Some nucleated erythrocytes appear, but less in in the acute post-hemorrhagic form. The white cells vary with causal conditions. As a rule, the polymorphonuclear neutrophiles increased, and the eosinophiles (normally 4 to 6 per cent.), are creased. The latter are often increased in parasitic anæmias, up to or even 50 per cent. In some chronic anæmias, the leukocytes are minished.

As to *general symptoms,* it is frequently difficult to decide whether ey result from the anæmia, or from its cause. An anæmic skin, mus- lar adynamia, irritable weakness, and a tendency to œdema and morrhage into the skin, mucosæ, and serosæ, may be noted. The ppetite is poor or easily satiated, and tenderness over the stomach, rdialgia, nausea, and decrease or increase of the HCl may be ob- erved. The intestinal functions are usually normal, though the motor ower may be tardy. The aromatic sulphates of the urine are frequently increased. Rather exceptionally symptoms resembling those of pernicious anæmia may develop, as alteration of the bone marrow, fatty heart, cerebrospinal degeneration, atrophy of the gastro-intestin cells and retinal hemorrhage. The *differentiation* from prima anæmias has been considered under pernicious anæmia.

Treatment.—The treatment depends upon the cause, for whi thorough and repeated search should be made in doubtful cases. T hygiene of the home and place of work should receive consideratio Fresh air, sunlight, and proper food are often difficult to obtain for t lower classes. Iron and arsenic are valuable in simple types (Chlorosis and Pernicious Anæmia).

(D). LEUKÆMIA.

Definition.—Leukæmia (leukocythæmia) is a disease of hæmoge esis, characterized by an increase of the leukocytes, a decrease in t red cells and hæmoglobin, hyperplastic alteration of the bone marro lymph glands, and spleen, and by secondary infiltration into vario organs and tissues. It was discovered by Bennett, and independent by Virchow (1851).

Classification.—The older classification embraced (*a*) the splen and (*b*) the glandular (Virchow) types to which Neumann added (the medullary form; Béhier described (*d*) a gastro-intestinal form, a Phillippart, Nachter, and Kaposi (*e*) a dermal variety. This classi cation is clinical, but that of Ehrlich is based on the blood findin the splenic form is not recognized by him, and he classifies the disea as follows:

1. Lymphatic leukæmia. (i) The acute form, which runs a ve rapid course, and resembles an acute infection, with lymphæmi moderate or no splenic enlargement, fever, hemorrhages, lympha enlargement, and stomatitis. (ii) The chronic form, with lymphæmi enlarged spleen and glands, and a protracted course.

2. Myelogenic leukæmia with great polymorphism in the blo findings (*v.i.*).

Acute Lymphatic Leukæmia.

Acute lymphatic leukæmia (*acute lymphæmia*) includes nearly acute leukæmias. The term does not imply that the lymph glan alone are the cause, because there is considerable and growing evidenc that the bone marrow may be the seat of the causal change, and tha it may contain a parent cell for both leukocyte and lymphocyte. The chief pathological finding is *hyperplasia of the lymphadenoid tissue.* The course is that of a *rapid infective disease,* usually with an acute onset, lymphæmic blood findings, asthenia, and often fever; it is fatal in a few days to a few weeks. The disease occurs at any age, but especially in *children,* and 66 per cent. of cases are in males. The bacteriological findings are inconstant; the amœba described by Löwit lacks and is considered an artifact by most hæmatologists.

The disease is infrequent; Fussel and Taylor have collected but 56 cases, but it is certain that many cases escape detection.

Symptoms.—Prodromal symptoms may be observed, as headache, pain in the neck, spleen, joints, or head, epistaxis, mental obscuration, dyspnœa, or stomatitis, after which four cardinal findings develop:—

1. Evidences of *lymphadenoid change* are observed, as *lymph gland* intumescence, which is very frequent, although not invariable, is usually moderate, and sometimes disappears after the initial swelling; *splenic enlargement* occurs in 66 per cent., and is of slight degree, save in children, in whom it may be enormous; *tonsillar* hypertrophy occurs in 50 per cent., sometimes with hemorrhage and necrosis. Reed recently published a case of acute lymphæmia without glandular enlargement.

2. *Hemorrhages* are most characteristic and frequent; they occur (*a*) in the skin, as petechiæ or larger effusions, with necrosis or ulceration in areas of lymphæmic infiltration; (*b*) in the mouth, gums, palate, and pharynx, as petechiæ, erosions or extensive necrosis, developing in areas of lymphæmic infiltration; (*c*) in the retina in practically 100 per cent. with the same anatomical basis; (*d*) visceral hemorrhages, in the brain, cerebral nerves, labyrinth, and spleen; (*e*) from mucous surfaces, the vagina, urinary passages, and intestines; and (*f*) from the serous membranes.

3. *Necrosis* in the infiltrated foci of the mouth occurs in 70 per cent., and is *per se* highly suggestive in the diagnosis (Bradford and Shaw).

4. *Lymphæmia* is the *crucial finding.* The ratio between the white and red cells is 1 to 2, 10 or 20, though the absolute number of lymphocytes may reach 918,000 to the cubic mm. In general, the increase of leukocytes is less than in the splenomedullary form, and averages 350,000. The polymorphonuclears are usually normal or decreased, the eosinophiles constitute about 1 per cent. of the white cells, a few myelocytes may be found, but the salient increase is in the lymphocytes, and chiefly in the larger forms; the lymphocytes may constitute 90, or even 99 per cent. of the leukocytes. The red cells vary between 1 and 3 million, and some nucleation occurs (although frequently absent, Wertheim, Hayem, Neumann, Rieder). The hæmoglobin usually falls below 50 per cent., or much lower in peracute cases. (For varieties of leukocytes, see Plate XIV, Fig. I. For blood picture see Plate XIII, Fig. 2 and Plate XIV, Fig. VI.)

Other findings are pallor, frequent pulse, priapism, swollen and infiltrated liver, increased flow of urine, with increased urea and uric acid (increased nuclein destruction); albuminuria, nephritis and endocarditis are sometimes observed. Edsall found the nitrogen output enormously increased (from autolysis). Lymphomatous masses may cause dyspnœa. The fever is irregular, both in type and occurrence. Intercurrent sepsis reduces the splenic and lymphatic enlargement, the white cells decrease even to normal, and sometimes an ordinary polymorphonuclear leukocytosis replaces the typical blood findings. The *bone marrow* is *constantly involved,* especially in the long bones;

it is mostly red and jelly-like in color, more rarely red-gray the seat of hemorrhage. The typical granular neutrophile my are few, while large and small lymphocytes abound as in tl lating blood; this is known as "the lymphoid marrow." (vary as to whether the lymphatic or the medullary lesions primary change. Lymphatic deposits occur in the mouth, intestines (simulating typhoid ulcers), muscles, liver, kidn thymus. The patient is usually emaciated. Fatty and deg changes in the heart and other viscera are observed.

Diagnosis.—Though the splenic and lymphatic enlar petechiæ, stomatitis, fever, ulceration and hemorrhages are fr most suggestive, the blood findings alone are distinctive; they typhoid, purpura, scurvy, trichinosis, erythemata with mouth diphtheria (for which lymphæmia is often mistaken), sep severe acute anæmias, and chloroma (*v.i.*).

Chronic Lymphatic Leukæmia.

Chronic lymphatic leukæmia is rarer even than acute lym and runs a clinical course of from months to years, with lympl hyperplasia and lymphæmia; it is closely associated by som with malignant lymphoma and Hodgkin's disease. (Thus describes (*a*) the genuine lymphatic leukæmia with marked lyn and (*b*) lymphatic pseudoleukæmia with no striking dispr between the number of red and white cells, but with a relative of the lymphocytes compared with the other white cells. views have not yet attained general recognition and confirmati

Etiology.—No uniform or constant bacteriological cause l proven. Löwit described an amœba (hæmamœba leukæm [vivax]), which most authorities consider an artefact.

Symptoms.—The symptoms begin insidiously and the dise be first detected by (1) swelling of the *lymph glands*, usuall neck, which may reach enormous dimensions. The axillar are large, though the inguinal are less massive; glandular as a rule, extends from above downward. Sometimes the glands alone are swollen, and in very rare cases the lymphatic enlarged. The glands are discrete without periadenitis, are soft, being much less hard than in pseudoleukæmia. So moderate pain is experienced and there may be compression structures, as the vagus, trachea, and portal vein. The glands to grow until death, unless an intercurrent affection as er causes them to shrink temporarily.

2. *Splenic hyperplasia* and intumescence is practically though generally less extreme than in the myeloid type, or in H disease. The spleen often projects from beneath the costal arc inch or so, it grows as the disease progresses, and is usually tionate to the duration of the disease. In a case of Virchow, tl

was not enlarged. Intercurrent sepsis, cholera, erysipelas, etc., may cause the splenic tumor to temporarily disappear, together with the lymphæmia and the lymph nodes, but all three findings may persist in such infections. Diarrhœa, and treatment by arsenic or quinine, may likewise effect splenic reduction. Perisplenic adhesions and thickening are common, and the organ is soft or hard according to the duration of the disease. The tonsils are often swollen, in which hemorrhages, with or without necrosis, are frequently observed.

3. The changes in the *bone marrow* cause few symptoms; pain is rarely found. The essential character of the marrow alteration is undetermined.

4. The *blood* reveals an *absolute and a relative lymphæmia.* The red and white cells may have a ratio of 100 to 1, and yet the lymphocytes constitute 90 to 99 per cent. of all the white cells. They are mostly small lymphocytes (Pinkus), whereas the larger forms occur in the acute lymphatic leukæmia. (Ehrlich holds that large lymphocytes predominate.) Many of them are altered, degenerated, or swollen. The neutrophile polymorphonuclears (normally constituting 70 per cent. of the white cells), are reduced to 10, or even 2, or 1 per cent. Myelocytes are usually absent. The red cells average 4,000,000, but may reach 2,000,000, and seldom lower. The lymphocytosis may temporarily disappear during infection, and in one instance this was permanent. (See Plate XIV, Fig. 1.)

5. The *skin* may itch or be the seat of papular eruptions, or urticaria. Lymphomata form in the skin, sometimes of considerable size; or infiltrated reddish deposits may precede the lymphæmic blood findings. Œdema occurs, while hemorrhages are rarer. Facial growths are observed similar to those of chloroma (*v.* MEDULLARY FORM, DIAGNOSIS), or to the symmetrical swelling of the lachrymal and salivary glands.

6. The heart is degenerated, as are the vessels, from all of which hemorrhages may occur, as in the acute type. Intraperitoneal hemorrhages from ruptured adrenals are often reported. The nerve trunks may degenerate with bulbar, spinal, and nerve trunk symptoms. Retinitis leukæmica or optic nerve involvement may impair vision. The liver is swollen from degeneration, and from lymphatic infiltration. Dyspnœa is most common, and occurs early from the anæmia, mechanical compression of the air tubes, vessels or nerves, or from leukæmic changes within the follicles of the mouth and air passages. Renal infiltration is frequent.

Course.—The *course of the disease* is slow; it develops gradually and passes slowly into cachexia, though now and then extension in the glands is "explosive" (Trousseau). Remissions and intermissions of weeks or months are not uncommon. Intercurrent disease may be fatal, as pneumonia, and mechanical stasis, suffocation and acute exacerbations of the disease itself may hasten the course.

tween the twentieth and fiftieth years; 67 per cent. of c
males. Bacteriology has shown no cause, and Löwit's t
ing the hæmamœba leukæmiæ magna, has been vigoro
The disease is frequent among the Polish Jews, and t
working classes. A history may be obtained of malari
phoid, influenza, stomatitis, tonsillitis, digestive disturban
disturbed menstruation, the climacteric and trauma, th
probably only accidental occurrences. The disease has
three generations. Leukæmia has been also observed
dog, cow, pig, and cat.

Symptoms.—1. Blood.—The specific gravity is d
1,055 to 1,045–36; it is watery, or sometimes chocol
coagulates slowly (on account of the peptone present f
cells, although observations differ on this point). In ext
coagula are white (leukæmia) or slightly greenish. La
are observed at autopsy, and Virchow thought that he
abscess when he opened the left ventricle in his first aut
develops after puncture from lecithin and glyceroph
Microscopically the white cells are enormously increased
as 1 to 1, or in Robin's case, 2 whites to 1 red cell.
count which the author has seen was in Mercy Hospita
whites numbered 950,000, and the reds 1,900,000 (1:2
one must even look for the red corpuscles. Amœboi
greatly decreased. (Plate XIV).

The *characteristics of the white cells* are: (*a*) The *lar*
neutrophiles ("mark cells," myelocytes), which are fo
amount to 30 per cent. or more of the leukocytes. In t
are not absolutely pathognomonic, having been found
bers) in pneumonia, uræmia, chlorosis, skin diseases, s
normal blood. (*b*) *Mononuclear eosinophiles,* which
diseases, but in myeloid leukæmia are absolutely increas
necessarily relatively increased. They are absolutely n
diagnosis. (*c*) The "*mast cells,*" which must be absolu
they are polymorphonuclears with basophile granulati
not show with the tri-acid stain. Often they are relati

cells containing mitoses, fat globules, or red cells, may also be found. (See Plate XIV, Fig. I.)

The *red cells* are, as a rule, decreased to two or three million, though leukæmic patients are not always anæmic and an excess of red cells has been described by Quincke and Litten. Toward the end the red cells may decrease to even 300,000, and thus resemble pernicious anæmia. The reds may become degenerated, necrotic, polychromatic, or acquire amœboid movement, nuclei, and mitoses. Nucleation occurs more frequently than in any other anæmia (Ehrlich), normoblasts being the most common form. The hæmoglobin tends to crystalize. The color index is 1, or less than 1 (0.5 to 0.7). (See Plate XIII Fig. I. and Plate XIV, Fig. VII.)

2. Splenic Enlargement.—This is usually the first symptom to attract attention; it appears with the blood findings and is most constant; spleniculi, if present, are enlarged. Instead of a weight of 5 to 7 ounces, 18 to 28 pounds are reported; from the normal measurements, 3 to 5 inches, dimensions of 15 inches are frequently observed. Splenic pulsation was observed by Gerhardt. The form of the organ is preserved. The size varies (*a*) during intermissions; (*b*) without cause; (*c*) from administration of arsenic and quinine, and (*d*) from diarrhœa or hemorrhages. It is enlarged after meals. A systolic murmur is sometimes heard. The notches are usually clearly palpable, and the edges are plump, rounded, and hard. Crackling under the fingers and tenderness indicate soft adhesions (perisplenitis). Firm adhesions may develop and the capsule may thicken. Though at first soft, it later becomes very firm. The pulp and trabeculæ hypertrophy, and new lymphoid deposits are clearly marked against the reddish pulp. It may reach to the navel, or into the pelvis, displacing other organs. Pruss and Westphal, on puncture, found the Charcot-Neumann crystals. The author has seen three cases of leukæmic floating or wandering spleen, two of which had been operated on as uterine tumors. Rupture is a rare complication.

3. Glands.—The glands are enlarged in 33 per cent. According to the most recent views, the glands are heteroplastic, due to new-formed myeloid tissue, "metastases" from the bone-marrow. Though soft at first, they later become hard. Caseation and suppuration are most infrequent. As in the other types of leukæmia, recurrent compression of the vagus, trachea, portal vein, and bronchi, may result. Retrosternal dulness has been observed.

4. Bones.—Bone changes, recognized first by Neumann, occur in the spongy and long bones (ribs, tibia, sternum, and vertebræ). The bone is mostly rarefied and there may be tender places, or yielding areas which are more important than tender ones. The histological changes in the marrow are (*a*) the *lymphoid* (Neumann), in which the marrow resembles currant jelly, is reddish, and consists of small lymphocytes; it is sometimes hemorrhagic; the normal fatty appearance disappears. (*b*) The *pyoid;* which is seen mostly in the myelogenic type; the marrow is not transparent, but gray; its cells are rich in protoplasm with one or many nuclei. In both forms nucleated red cells abound, also eosinophile cells, large cells with neutrophile granules and large nuclei.

5. Vascular System.—The heart is moderately distended with white clots, and as Virchow described in his first autopsy, opening the heart was like incising an abscess. The muscle is pale and fatty, and nodules of leukæmic infiltration in the myocardium, foci of hemorrhage, and pericardial nodes and transudation are frequent. Cardiac dislocation from pressure by enlarged glands and spleen, anæmic bruits, venous pulsation, *bruit de diable,* palpitation, systolic murmurs from pressure by the glands on the vessels, and centripetal venous pulse (Senator) are observed. The radial pulse is rapid, and either soft or tense. Dyspnœa results from weak heart, vagus pressure, nodules in the lung, bronchitis, hydrothorax, pressure of the spleen on the diaphragm, tilting of the heart upward, and compression by the glands.

6. Respiratory Tract.—Nodules are frequently seen in the larynx, trachea, thyroid, and thymus, often with hemorrhage, sometimes resulting in tracheal, bronchial, or laryngeal stenosis. In the lungs (Deiter) lymphoid nodules may necrose and cavities like those of caseous tuberculosis (Boettcher) may appear. Small myeloid foci of leukocytes may resemble miliary tuberculosis (Virchow). Bronchitis with many eosinophile cells in the sputum, nodes along the pleural vessels, and pleural hemorrhage, transudation and exudation have been observed.

7. Fever.—Fever is less frequent than in the acute type, but occurs in the majority of cases. It is rarely high; temperature may be normal for months, or it may resemble recurrent fever, typhoid, sepsis, or malaria, though it follows no one type.

8. Digestive Canal.—Anorexia, thirst, stomatitis, or pharyngitis with ulceration of the lymphoid deposits, dysphagia from glandular pressure on the œsophagus, eructations and vomiting are occasional symptoms. Diarrhœa is very common, leucin and tyrosin being found in the stools. An enteric type is described by Béhier and others, in which the bowel symptoms are conspicuous; the lymphadenoid structures in the ileum are prominent, and Friedreich once confused this type with typhoid on account of the ulceration, enlargement of the spleen, and hemorrhage from the bowel. The abdominal lymph glands may be enlarged, though the others may remain normal. The *liver* is much enlarged, especially when the spleen is enlarged. In Welch's case it weighed 13 pounds, and in another it weighed 25 pounds. Its surface is smooth, its consistence increased, and its edges plump. Myeloid proliferations or deposits occur in the liver as in the spleen, and dislocate or deform the rows of liver cells. The portal lymph nodes may be enlarged. Icterus is infrequent and ascites is common from cachexia or lymph nodes (leukæmic peritonitis). The fluid contains many white cells and the myelocytes predominate as in the blood.

9. **Kidneys.**—The kidneys are frequently involved; there may be "myeloid" infiltration and cloudy, fatty, and amyloid alteration. The urine is decreased, pale, and strongly acid. The *uric acid* (Virchow) may amount to 45 to 105 grains (3 or 7 gm., Laache), though usually 20 grains (1.5 gm.), (normally 8 grains, or 0.5 gm.). Normally uric acid is as 1:50–80 of urea, but in leukæmia, it is 1:16 (Salkowski). According to Stadthagen, the uric acid results, not from decreased oxydation of the albumins, but from increased formation of the acid. The xanthin bodies are increased. Albuminuria is usual only in the last stages. The amount of urea depends on the degree of cachexia. Priapism occurs from nervous causes, as hemorrhage, or from thrombosis of the dorsal vein. Amenorrhœa is frequent in women.

10. **Nervous Symptoms.**—Nervous symptoms may develop, as delirium, neuralgia, headache, syncope or vertigo, mania or rarely bulbar palsy, facial paralysis, apoplexy, or symptoms like those of brain tumor.

The *Eye.*—Lymphoid nodules may develop in the orbit, possibly with exophthalmus. The *retinitis leukœmica*, first described by Liebreich, is not a genuine inflammation, but a leukæmic infiltration and occurs in 25 to 33 per cent.; the retina is pale, its arteries small, and veins large; the papilla is "washed" and yellow, or orange colored spots with red borders appear; hemorrhages also occur (Plate VI, Fig. 9).

The *Ear.*—Vertigo, deafness, tinnitus, and an atactic gait, may result from growths in the labyrinth; sudden deafness from hemorrhage is described by Steinbruegger, Pepper, Wagenhausen, and Lanois; the author saw three such cases within one year.

11. **Skin.**—The skin is less involved than in lymphæmia, but there may be acne, pigmentation, œdema, pruritus, lymphoderma perniciosa, diffuse or tumor-like infiltrations, or sweats.

12. **Hemorrhages.**—These are less frequent than in acute leukæmia or in pseudoleukæmia, though Mosler found them in 52 per cent. Epistaxis is the most common, and may be fatal. Stomach and bowel hemorrhages are next, and those from the respiratory, genito-urinary, and other tracts are rarer. They also occur in the skin, joints, muscles, subcutaneous tissues, or brain, where they may be single or multiple. The writer has seen three massive hemorrhages under the skin of the thorax, one in Leube's clinic, and two in his own practice. Death has followed the extraction of teeth (Chapelle) in a leukæmic subject. The writer saw Kolisko autopsy a case in which a slow ascending paralysis followed hemorrhage between the spinal pia mater and arachnoid.

Complications.—Complications are nephritis, pneumonia, endocarditis, amyloid degeneration, diabetes, and gangrene. Sussmann could collect but 25 cases of complicating tuberculosis; this number was increased by Dock to 27; at the time of writing, the author saw a case with E. W. Andrews, in which a left-sided tuberculous pleurisy developed; tubercle bacilli and mononuclear white cells were recovered from the blood-stained fluid.

Diagnosis.—The diagnosis was formerly a matter of great difficulty, because the difference between leukæmia and leukocytosis was made one of degree only, and various limits were placed (as a ratio of 1 leukocyte to 50 erythrocytes), beyond which leukocytosis was said to cease and leukæmia begin. The mere number of white cells is no criterion, because a ratio of 1 to 15 or 20 may be leukocytosis (as in Strauss' case of gastric cancer), and on the other hand, 1 to 200 may be leukæmia (von Noorden). Leukocytosis from digestion, fasting, marasmus, pregnancy, acute infections, or in the death agony, is of the ordinary polymorphonuclear neutrophilic type. (Plate XIV, Fig. IV.) Leukocytosis is a symptom, but leukæmia is a disease. It is clear, then, that increase in the variety of white cells is the definitive test. The *polymorphism* of the blood is striking; according to Lazarus, there must be (*a*) mononuclear leukocytes with granulations (myelocytes); (*b*) increased mono- and polynuclear eosinophiles; (*c*) absolute increase in the "mast cells," and (*d*) nucleated red cells must be readily found. The gross splenic, medullary, or lymphatic lesions are of but presumptive value in diagnosis, and the polymorphic blood picture alone differentiates from pseudoleukæmia, typhoid, purpura, anæmia, sepsis, or malaria. Not only can the disease, but also its type, be recognized from findings in the blood.

Special caution should be exercised in making a diagnosis (*a*) during remissions, when the blood is rarely morphologically normal, no matter what the number of leukocytes. In one of the author's cases, the leukocyte count dropped from 1,250,000 to 9,000 in a week, though the myelocytes still constituted 33 per cent. of the white cells. (*b*) Care is also necessary during acute infections when the leukæmic characteristics may disappear (but not always), as during typhoid, acute miliary tuberculosis, influenza, or sepsis. (*c*) The blood findings may become atypical just before death.

Similar Conditions.—1. *Chloroma* is considered a malignant type of leukæmia, or a "neoplastic hyperplasia of the red bone marrow," by Dock and Warthin, who in 1904, collated 39 cases. It is closely related to acute lymphæmia. Lymphomata form, causing orbital pain, tinnitus, deafness, temporal and orbital swellings, and such ocular symptoms as exophthalmos, strabismus and disturbed vision. The lymphomata are greenish, whence the name chloroma. It usually develops before the twentieth year. Retinal infiltration occurs with hemorrhages. Other findings are ataxia, pallor, sternal tenderness, hemorrhages, severe anæmia, often but not always changing to leukæmic characteristics, greenish urine, and greenish lymphomatous infiltration of the bone marrow, periosteum (sometimes with Charcot-Leyden crystals), the liver, or lungs, and greenish lymphadenoid changes. The hæmoglobin and red cells are reduced, even to 15 per cent. and 500,000 respectively. The leukocytes number about 50,000, but have been as numerous as half a million; they are chiefly lymphocytes. The affection is always fatal.

2. *Leukanæmia* is described by Leube as a severe blood disease, [illegible] which the myelogenous formation of both the red and white cells [illegible] disordered, *i.e.*, it combines some features of both leukæmia and pe

nicious anæmia. Its features are (*a*) a progressive anæmia, waxy color, and asthenia; (*b*) numbers of normoblasts, but especially of megaloblasts; (*c*) no abnormal pigments in the urine, no enlargement of the lymph glands (though since reported by Mattirolo), and no siderosis of the tissues; (*d*) no true leukæmic blood findings, but considerable myelocytosis and lymphocytosis; (*e*) firm connective tissue in the bone marrow, and (*f*) enlargement of the spleen, and prevertebral hæmolymph glands.

3. *Secondary Cancer in Bone.*—There are 13 cases in the literature, in which cancerous bone metastases have produced blood findings rather like those of pernicious anæmia and increase of the leukocytes (of which the myelocytes constituted 4 to 17 per cent.).

Prognosis.—The prognosis of leukæmia is bad. The former reports of recoveries are not wholly reliable. It may last 5 to 8 or even 10 years after a diagnosis has been made. One-half to 3 years is the usual length, and a little over 1 year is the average *course*. The *beginning is insidious*, and enlargement of the abdomen, dyspnœa, anæmia, and enlarged glands are the cause for seeking medical treatment. The nutrition is usually good. *Sudden death* may occur, either without discernible cause or from hemorrhage of the brain, hæmatemesis, marasmus, anasarca, diarrhœa, pleuritis, peritonitis, deep coma, pneumonia, or diarrhœa. *Remissions* (as in pernicious anæmia) occur, but the blood findings persist. Ewing observed 3 *acute cases*, and Billings and Capps published 2 and collated 7 others from the literature (which resembled an infection with hemorrhages, moderate splenic and lymphatic hyperplasia, severe anæmia, often necrosis of the throat and jaw, with 16,000 to 540,000 leukocytes, and often with low fever). A permanent cure after erysipelas has been reported by Richter.

Treatment.—*Hygienic* measures, rest and a careful diet, are of some benefit. Fats and carbohydrates cause dyspepsia, whence Litten gives animal foods, eggs, milk, raw chopped meat, koumyss, peptones, oysters, beer, wine, zwieback, spinach, Valentine's meat juice, and Brand's meat extract; an ice-bag to the spleen may mitigate pain over it. *Splenectomy* is most irrational, for leukæmia is a primary disease of the bone marrow. All of Collier's series of 16, and 94 per cent. of Bissel-Hagen's group of 35, died. The great danger is that of hemorrhage. Great *caution* must be observed in using *purges*, which, as in Addison's disease, may cause sudden collapse and death. Inhalations of oxygen, blood infusions, spleno- or organotherapy and iron are inferior to *arsenic*, which decreases the number of white blood cells, and the size of the spleen. Hemorrhages may disappear, and the number of red cells increase. Ebert gives arsenic with quinine. It is injected locally (by Mosler) into the glands and spleen, but in the author's experience, without success. Phosphorus may soften the glands. Special care should be employed in tapping an ascites, for peritonitis is prone to develop. The *x-rays* may prove decidedly beneficial. In 1 case already alluded to, the patient lived 19 months after the day he seemed to be dying. The way it helps is still a mystery. Schirmer says that

72 cases are reported (1905), and Wendell thinks over 90 per cent. were benefited. In some cases, the glandular and splenic enlargements, and the blood findings have wholly disappeared, as in 1 case observed by the writer. The *x-rays* seem to produce a leukotoxic substance (Capps and Smith).

(E). PSEUDOLEUKÆMIA.

Definition.—A disease resulting in chronic anæmia, or cachexia of which the chief symptoms or signs are enlargement of the spleen, o lymph structures, or both. The definition of the disease as "leukæmi appearance without leukæmic blood findings," no longer holds. Such conceptions would include glandular tuberculosis, Kundrat's lymphosarcomatosis, etc. It is also called Hodgkin's (1832) disease, pseudoleukæmia (Cohnheim), adenia (Trousseau), lymphosarcoma (Virchow) malignant lymphoma (Billroth), and anæmia (cachexia), lienalis o lymphatica (Wilkes).

Etiology.—The etiology is not clear. The disease occurs after loc lymphatic disturbances in the neck, following otorrhœa, coryza, extirpation of the glands of the neck, and tonsillitis. Like chloroma an leukæmia, it is observed chiefly (75 per cent. of cases) in males betwee twenty and thirty years of age. The disease is not uncommon in children, and often occurs in the lower animals. It also sometimes run through several members of the same family. Malaria, syphilis, scrofulosis, alcoholism, rhachitis, diarrhœa, typhoid, measles, and scarlatin are often noted in the patient's history, but their relation, if any, obscure. Suspected to be mycotic, many negative and variant examinations have been made. Though the disease has no well-establishe relation to leukæmia, as Cohnheim held, yet Fleischer, Penzoldt Mosler and Senator have observed transitions from pseudoleukæmi to leukæmia. Neumann held that the leukæmic blood changes depended solely on the hyperplastic marrow changes, and that as long as the hyperplasia involves only the spleen and glands, pseudoleukæmi results. Sternberg and others have maintained that it is tuberculosis a theory disproven by Dorothy Reed and Simmons.

Symptoms.—Its types are (*a*) lymphatic; (*b*) lienal, pseudoleukæmi splenica, splenomegalia; (*c*) mixed (*a* and *b*), and (*d*) myelogenic.

1. **Lymph Glands.**—Swelling of the lymph glands is generally the earliest symptom; the cervical glands first become enlarged, perhaps after an otitis or tonsillitis. The axillary glands become enlarged after a variable period, then the inguinal and other groups. They are soft and elastic; at first, the individual glands are round, or oval, discrete, smooth, and clearly palpable; later, with increasing size, they fuse into harder, irregular masses as large as the fist, or a child's head, particularly those in the neck. They may sometimes be painful from periadenitis, but never transcend the gland capsule, and seldom caseate or suppurate. From the neck, where the jugular veins may be compressed, the glandular enlargement often passes to the intrathoracic

glands, which are more often enlarged than the other internal nodes; this often occasions dyspnœa, compression of the superior cava, (whence the development of a large collateral circulation on the chest-wall), dysphagia, bronchial compression, and recurrent, or vagus paralysis. From the axillæ the glands under the scapulæ and pectoral muscles enlarge. The veins of the arm may be compressed, causing œdema. In the abdomen the retroperitoneal, mesenteric, periportal, iliac, and other lymphadenoid groups may enlarge greatly; according to their location, abdominal pain, œdema of the legs, paraplegia, hydronephrosis, ascites, and jaundice may ensue. *Histologically*, the lymphadenoid changes are (i) increase in the endothelial and reticular cells; (ii) hyperplasia of the lymphoid cells; (iii) formation of peculiar giant cells; (iv) fibrosis and chronic inflammation, and (v) great increase of the eosinophile cells (Dorothy Reed).

2. Spleen.—The spleen is enlarged in 75 per cent. of cases (Gowers). When enlarged, it reaches dimensions rarely attained in leukæmia, but maintains its form, is hard and tender, and presents the same histological appearance as in leukæmia, viz., nodes of lymphoid cells in a fibrous reticulum. The greatest swelling of the spleen is usually observed when the lymphatic glands are large and hard, yet splenomegaly may be the sole symptom. Some physicians doubt the correctness of the diagnosis when the spleen only is involved, but splenic hypertrophy alone, with relative increase in the lymphocytes, may be classed as Hodgkin's disease according to Pinkus.

3. Bone-marrow.—The bone-marrow is less frequently involved than in leukæmia. A myelogenous pseudoleukæmia has been recorded, but its exact status is still doubtful. The eosinophiles are abundant in the lymph nodes and bone-marrow.

4. The Blood.—The ratio between the red and white cells is normal. The hæmoglobin is decreased and the blood index is usually 1; the hæmoglobin may be rapidly decreased as in Litten's case, from 85 to 40 per cent. within a week. There is usually moderate anæmia, especially in advanced cases. Poikilocytes and microcytes often occur; megalocytes rarely, and normoblasts sometimes are found, but the eosinophiles are not increased. The blood plates are increased, but the Charcot-Leyden crystals are never found. Concerning the white cells, there is a relative increase of lymphocytes, the chief criterion of the disease, according to Pinkus and Ehrlich. The polymorphonuclear neutrophiles are decreased. These changes may not be observed in the earliest stage of the disease. The total of white cells is not essentially increased; in the case of Askanazy, the disease was pseudoleukæmia for two and a half years; when it became leukæmia, [illegible] lasted another one and one-half years. Limbeck found an increase in the polymorphonuclears in inflammation of the glands with fever, and the lymphocytes increased when the change was like that of lymphosarcoma. Leukocytosis may be explained in some instances as a leukocytosis of the death agony, or a terminal infection. [illegible] cases with decrease in the lymphocytes, which was caused by their decreased formation in the lymph glands.

5. Other Organs.—(*a*) The urticarious eruptions and *lymphatic growths in the skin* occur rather more frequently than in leukæmia; the lymphoderma perniciosa of Kaposi (*érythrodermie mycosique* of French writers) may be considered a type of pseudoleukæmia in cases with an absolute lymphocytosis. Erythema, purpura, furuncles, pemphigus, prurigo from irritation of the lymphatic deposits around the sweat glands, and pigmentation, have been observed. According to Dubreuilh (1905), 18 cases of prurigo are reported. Severe *sweats* are frequent. (*b*) Symptoms of *anæmia* occur, as a feeling of cold, vertigo, syncope, dyspnœa, œdema, hemorrhage, palpitation, anorexia, dyspepsia, and severe diarrhœa. (*c*) *Local pain* over the *liver* and *spleen* may indicate perihepatitis, or perisplenitis. Swelling of the liver may be marked, and is due to lymphoid deposits. Severe muscular pain has, in some instances, suggested trichinosis. (*d*) Gangrenous stomatitis, or pharyngitis, may occur in degenerated lymph nodes in the mouth, tonsils, and throat. Gastric and intestinal ulcerations and sometimes perforation are noted, though they are less frequent than in lymphosarcoma. Neusser noticed gastric crises like those of tabes. (*e*) Ross and Osler described lancinating pains in the feet, and œdema from involvement of the spine and cava by the lymph nodes. (*f*) Amyloidosis, bronchial catarrh, and pulmonary phthisis have been noted. (*g*) The uric acid in the urine is not increased. (*h*) The lymphatic hyperplasia may also involve the lymphatic structures in the tonsils, circumvallate glands of the tongue, the muscles, mammæ, eyelids, tibia, thyroid, or thymus; secondary deposits may be noted in the lungs, liver, heart, pleura, and more rarely in the brain, genitalia, and pelvis.

6. Fever.—Fever is common. Gowers and Murchison observed a chronic recurrent type, which was later described by Pel and Ebstein; the term is a misnomer, and classification of this febrile variety, as a separate form, is incorrect, for it has been observed in chronic malaria, lymphosarcoma, other malignant diseases, and in glandular and splenic tuberculosis which simulates Hodgkin's disease. The urine may show the diazo reaction with the fever. When the disease involves the internal glands only, the fever may simulate typhoid. Winnewarter stated that fever occurred when the glands decreased in size under the administration of arsenic. Fever is rather indicative of glandular tuberculosis, or of intercurrent (terminal) infections.

Course.—The clinical course is long. The glandular swelling sometimes decreases with, or without, therapeutic measures. The extension from the cervical to other lymphatic glands is generally gradual, but may occur with great suddenness, as in Trousseau's case. An acute form of Hodgkin's disease is not well established, although cases have been reported; many are sepsis, tuberculosis, or acute sarcomatosis. *Death* may occur from intercurrent diseases, but it most frequently results from chronic cachexia. Obstruction of the air passages occurs more frequently than in leukæmia. Death may also occur from twisting of the pedicle of the spleen, or from hemorrhages from the nose, uterus, stomach, intestines, or larynx, which sometimes resemble purpura hemorrhagica of Werlhof. Very exceptionally, transition into leukæmia is observed. It is a question whether patients with well-developed cases ever recover, though the disease is not so *fatal* as is leukæmia. Pleural effusions, diarrhœa, dysentery, and vomiting from pressure of the splenic tumor, are unfavorable complications.

Diagnosis, Allied Affections and Differentiation.—For pseudoleukæmia there are two criteria; these are (*a*) the lymphadenoid hyperplasia and chronic inflammation observed when an excised node is examined histologically; other kinds of cells, or other degenerations, excite suspicion that the disease is other than pseudoleukæmia; and (*b*) according to Pinkus, an absolute lymphocytosis, with a practically normal number of white blood cells. Differentiation from similar affections is as follows:

1. Glandular Tuberculosis (Sternberg, Noothaft, Goldman, Kanter, Delafield, Crowder, Askanazy, Waetzoldt).—Hodgkin's disease may be complicated by, or confused with, tuberculosis; in pseudoleukæmia the typical structure of the tubercle is lacking, but in tuberculous adenitis, there may rarely be a diffuse accumulation of large endothelioid cells between which lie tubercle bacilli (Ziegler); after a long course, caseation and giant cell formation may result; the forms of glandular tuberculosis are (*a*) Ziegler's large cell infiltration, (*b*) purely hyperplastic lymphoid proliferation, or (*c*) anatomical tubercles. Of late, the number of reported instances of glandular tuberculosis simulating Hodgkin's disease has been increasing enormously. The author examined 2 cases of supposed pseudoleukæmia in which tubercle bacilli (without anatomical tubercles) were found in numbers in the spleen and lymph nodes. In the neck, tuberculous adenitis is more often in the submaxillary than in anterior and posterior glands, in the latter of which pseudoleukæmia is more common. Tuberculosis is more frequently a local than a general adenitis, and is more often attended by secondary suppuration. Türck believes that fever and glandular tenderness indicate tuberculosis, and that node-like swellings of the lymph vessels in the mouth are symptomatic of pseudoleukæmia. Excision of a node or operation determines the question.

2. Anæmia Infantum Pseudoleukæmica.—This disease was first fully described by von Jaksch, though earlier observed by Senator; it is known in Italy as *anæmia splenica infettiva dei bambini;* it is called "anæmia splenica," or "pseudopernicious anæmia of children," by Ehrlich. It is a profound anæmia in which the blood findings suggest pernicious anæmia; that is, there are normoblasts, megaloblasts, decrease of the red blood cells (1½ to 3 million), and sometimes poikilocytes, or polychromatophilia; there is often increase of the leukocytes, but they rarely number more than 50,000. It occurs in young children from nine to eighteen months of age; the convalescence is usually very slow, or death may result. Its symptoms are hardness and e ment of the spleen, swelling of the liver (less than in leukæmia a than the swelling of the spleen), signs of profound anæmia and rhages into the skin or from the nose, mouth, stomach, or int The status of the disease is not yet determined, because the lymp are frequently increased in children, and leukocytosis, in gener less significance than in adults. The diagnosis is especially d when it occurs in syphilitic children; syphilis, rickets, and dys are common antecedents.

3. **Anæmia Splenica.**—Doubt has been expressed whether it be classed as an essential anæmia, liver cirrhosis, chronic splen as a splenic form of Hodgkin's disease. (*a*) The *spleen is enlar* capsule thick, its stroma increased, and the endothelium of the sinuses sometimes proliferated. The cause is unknown; possib toxic. (*b*) There is an *anæmia* of the *secondary*, or chlorotic, the red cells average 3,500,000, the hæmoglobin is even more r and the whites are decreased to 3 or 4 thousand. (*c*) *Hemorr* particularly from the stomach, are frequent. (*d*) In many case is a terminal *liver cirrhosis* (Banti's disease), with icterus and (*e*) Its course is *very chronic*, covering years. In some cases th profound alterations of nutrition, as stunted growth, clubbed f or pigmentation of the skin (in 8 of Osler's 18 cases). In severa Dock and Warthin found portal thrombophlebitis and hyper of the hæmolymph glands. Of Lichty's cases 5 recovered, 12 s improvement, and 7 died.

4. **Lymphosarcomatosis.**—This is sometimes most difficult ferentiate, and some clinicians, as Hoffmann, class the two di together. According to Kundrat, lymphosarcoma is largely disease, with local glandular involvement, especially of the c glands, leading to infiltration of the throat by contiguity; the may begin in the tonsils, the air-passages, the portal glands, etc. phosarcoma ulcerates and transgresses the gland capsule; this occurs in leukæmia or in pseudoleukæmia. The glands in leuk and pseudoleukæmia may compress neighboring structures but actually invade them. Unless lymphosarcoma extends beyon capsule of the glands, it cannot be distinguished by physical exa tion unless it lies in parts which can be easily inspected, as the The spleen may be enormously enlarged. According to Pinku leukocytes are increased in pseudoleukæmia while in lymphosa they are decreased.

5. **Other Splenic Enlargements.**—The most common of thes Cirrhosis of the liver, malarial spleen, sometimes carcinoma, chl with enlarged spleen (Chvostek), chronic polycythæmic cy (characterized by (*a*) enlarged spleen, (*b*) cyanosis, and (*c*) incre the red cells) and finally miliary tuberculosis of the spleen, a mycosis, syphilis, rhachitis or echinococcus which are rarer (see Diseases of Spleen).

6. Multiple Myeloma.—This is characterized pathologically by lymphoid hyperplasia of the bone marrow, occurring in the ribs and body-ends of the long bones chiefly, and more often in the proximal than in the distal bones; no metastases occur. The bones soften or sometimes sclerose. Clinically, it occurs in males over forty-five years and is characterized by severe, *deep-seated pain* in the body, chest, spine, neck, and more rarely in the extremities. Prominences in the bones, *spontaneous fractures*, especially of the sternum and ribs, and kyphosis, from softening of the vertebræ, may result. The growths may rupture externally. The *x-rays* may clearly define them. They sometimes pulsate. Severe anæmia, marked nervous disturbances, as neuralgia, motor and sensory paralyses, which usually develop late and involve the opticus, hypoglossus, facialis, cord (paraplegia dolorosa), and sciatic nerve; fever and the *Bence-Jones' albumosuria* are observed. The anæmic blood findings show no increase of the white cells. To call this disease a myelogenic form of pseudoleukæmia, or myelogadenia, there must, according to Pinkus, be a lymphocytosis.

7. Other Affections.—Syphilis of the lymphatic glands (lymphoma gummosa); symmetrical lymphoma of the lachrymal and salivary glands of Mikulicz, which are simple lymphoma reacting to arsenic, and resembling pseudoleukæmia; and secondary carcinoma, or sarcoma must be differentiated. *Difficulties* of diagnosis are met when the enlarged glands in internal parts produce topical symptoms, thus simulating hypertrophic cirrhosis, when it begins in the spleen.

Treatment.—**1. Local Applications.**—These are useless, excepting the *x-rays;* William A. Pusey states that two of his cases, seen by the author five years ago, wholly recovered. In some cases the rays have no influence on the spleen and glands, and in others the cachexia increases, though there may be improvement in the local findings. Inunctions of green soap, given once or twice daily, are sometimes beneficial, and in one instance the author saw marked temporary benefit.

2. Drugs.—*Arsenic* is most efficacious (*v.* PERNICIOUS ANÆMIA for details); it acts on the glands more than on the spleen. Some clinicians believe that the fever which it sometimes produces, reduces the adenopathy. Winnewarter and Billroth recommended its parenchymatous injection into the glands, but the results are not very encouraging. Iodides are seemingly injurious. Berberin hydrochlorate gr. xv increases the appetite and contracts the spleen (Litten). For hemorrhages Litten uses a decoction of quercus cortex, which rather disturbs digestion; equal parts of formalin and rectified spirits locally (which may irritate a delicate skin), and guacamphol gr. iij to iv once or twice daily may be given.

3. Surgical Treatment.—Extirpation of the glands is seldom beneficial. The removal of the spleen is attended by constantly decreasing mortality. In 1900, Bessel-Hagen collected 360 operations for various indications; the average mortality was 38 per cent. (for splenomegaly, 61 per cent. before 1891, and since then, 13 per cent.). In 1901, Harris and Herzog reported 2 operated cases, and collected 17 others, the mortality being 20 per cent. In Lichty's 25 cases, 8 were operated on with a 37 per cent. mortality. Scott has added 6 cases, of which 33 per cent. died.

Carstens, in November, 1905, collected 739 cases of splenectomy for various diseases of the spleen; 72 per cent. recovered.

THE HEMORRHAGIC DISEASES.

The hemorrhagic diseases may be classified roughly as follows:

(A). Acquired types.
- 1. Purpura.
- 2. Scurvy.

(B). Congenital (hereditary) type. 3. Hæmophilia.

(F). PURPURA.

Purpura includes a number of affections, more or less similar in kind and differing chiefly in degree, some of which are symptomatic, though others are independent but obscure affections.

Symptomatic purpura is observed as a symptom in:—(*a*) Various *infections*, as endocarditis, scarlatina, measles, smallpox, rheumatism, typhus, sepsis (strepto-, pneumo- and staphylococcus, and bac. pyocyaneus and coli), anthrax, cholera, yellow fever, plague, syphilis, or tuberculosis. (*b*) *Toxic* conditions, as nephritis, cholæmia, snake-bites, and drug-poisoning (from copaiba, ergot, belladonna, quinine, mercury, iodides, bromides, or arsenic). (*c*) *Cachexiæ;* old age, malnutrition, carcinoma, sarcoma, leukæmia, pseudoleukæmia, or pernicious anæmia. (*d*) *Nervous* states; cerebrospinal lesions, as tabes, myelitis; neuroses (bloody stigmata of hysteria); neuralgias, or vasomotor fluxions. (*e*) *Mechanical* conditions, as trauma, embolism, or stasis.

Purpura simplex (Zeller, 1684), is purpura in which the cutaneous hemorrhages are the chief symptom. Its *etiology* is obscure. It develops most often in middle-aged males. As in the other forms, whose description follows, there is no constant nor characteristic *pathology*, though vessel alteration (thickening, hyaline, and fatty degeneration), thrombosis, hemorrhage, and pigmentation sometimes occur. In the blood itself no change is observed other than its tardy coagulation; this requires 10 to 30 minutes, instead of 3 to 5, which is the normal time. The *eruption* begins as small red spots, which gradually become green and black; they occur largely on the external surfaces of the limbs, and more often on the legs than on the arms.

trunk. They do not fade upon pressure. The initial eruption may be macular, papular, or urticarious (purpura maculosa, papulosa, or urticans), but soon becomes blood-tinged. Constitutional symptoms are usually absent; there may be slight fever, gastric disorder, or diarrhœa (Graves). The usual *duration* is 1 to 2 weeks, the *prognosis* is good, and the *treatment* hygienic rather than medicinal. Relapses are common.

Purpura (or peliosis) rheumatica is a form described by Schoenlein, and still generally listed by itself because of the articular symptoms. As joint symptoms also occur in Werlhof's disease (*v.i.*), scurvy and hæmophilia, its separate classification is somewhat arbitrary. Most cases develop in males between fifteen and thirty years of age. It has no relation to rheumatism proper, being a pseudorheumatism. Its pathology is obscure.

Symptoms.—(*a*) As *prodromes*, indisposition, low fever, muscular pains, colic, and particularly sore throat, may be noted. The developed disease is manifested chiefly by various forms of purpura, and by symptoms in the joints; (*b*) the *purpura* may occur before, with, or after the joint-symptoms. It occurs as simple hemorrhages of the skin, which vary from red to green-black according to their age, or as urticaria, erythema, pemphigus, or œdema, which become stained with blood; the legs are most often involved, largely on the extensor surfaces. (*c*) *Articular symptoms* are most common in the knees and ankles, being less frequent in the elbows, and rare in other joints; there may be only stiffness and pain, or there may be actual swelling; if the joints are first involved, their symptoms frequently abate with the appearance of the purpura, and may last but 5 to 10 days. (*d*) *Other symptoms* are inconstant, as fever, which may rise to 102° or 103°, splenic tumor, albuminuria, or nephritis. The author once had 3 cases at one time in the Cook County Hospital, in which there was severe sloughing of the tonsils. Osler (1904) described 29 cases in which *visceral lesions* occurred with purpuric, urticarious, and erythematous symptoms; they occurred chiefly in children, and relapses were frequent; (i) digestive complications occurred in 25 cases; vomiting in 15; colic in 8 (resembling renal stone or appendicitis); blood in the stools in 8, and diarrhœa in 15; (ii) acute nephritis occurred 14 times, and 5 cases died of uræmia; (iii) arthritis occurred in 17 cases; (iv) swelling of the fauces, œdema of the larynx, aphasia, transient hemiplegia and least often endocarditis, may occur.

The **diagnosis** chiefly concerns affections characterized by hemorrhages of the skin, articular symptoms and perhaps temperature. Acute leukæmia, septicopyæmia and endocarditis must be excluded. Genuine rheumatic polyarthritis is usually differentiated with ease by the earlier migrating, inflammatory joint involvement, the temperature, sweating, characteristic cardiac complications, and the later, rather accidental and wholly secondary occurrence of the purpura.

The **prognosis** is generally good, though relapses are common and sometimes periodical. Its course is rarely longer than a week or two.

Treatment is hygienic and symptomatic. The joint-symptoms somewhat to acetanilide and salicylates, though the latter exer specific influence.

Purpura hemorrhagica (*Morbus maculosus Werlhofii*, Werlhof, 1 closely resembles the sub-types already described, but differs them in that not only are the skin and joints the seat of hemorr but also the mucous membranes, serous membranes, and perhap viscera.

Etiology.—Of this little is known; staphylo-, strepto- and pneu cocci, Bacillus pyocyaneus and coli, and one like Friedländer's pneu bacillus have been found. It occurs oftener in young weakly wo than in robust adults. Exposure to cold or dampness, and ma trition seem to be predisposing causes. It usually develops a apparently primary affection, but it may occur in some of the condi listed under symptomatic purpura.

Symptoms.—Perhaps after inconstant and variable prodrome fever, vomiting, or vertigo (*a*) *hemorrhages* appear on the *extrem* the face is not often involved; they occur as spontaneous pete blood-stained bullæ, or vibices, or may result from slight traum by pinching the skin. (*b*) The *mucous membranes* bleed to a va degree; the nose and mouth bleed most often; less frequently is hæmoptysis, hæmatemesis, enterorrhagia, hæmaturia, and me rhagia, in various combinations, sequences, and degrees. The junctivæ may be blood-stained. The writer saw one rapidly fatal in which there was hemorrhage from the middle ear. (*c*) The *s* *membranes*, and the joints, particularly the knees, ankles, and elb are often implicated. Ecchymoses in the endo- and pericard pleura, peritoneum, and meninges are common findings at aut (*d*) *Other hemorrhages* are much less common, as retinal hemorrh hemorrhage into the brain, cord, or testis. The connective tis fasciæ and bones are practically exempt.

There may be *general symptoms* and *complications*. As to the bl there is delayed coagulation and some anæmia, but the red cells hæmoglobin are rarely much reduced; some nucleated red cells be found, as well as leukocytosis, reduction in the blood plates, some methæmoglobinæmia. Fever is inconstant and atypical. pulse rate may be raised; palpitation, delirium, vertigo, albumin hepatic enlargement, splenic tumor, subicterus, and nephritis occasional symptoms. In J. S. Cutler's case, there was extensive i tinal ulceration; perforation has been also reported.

Diagnosis.—Most errors in diagnosis result from confusion those purpura cases which are symptomatic of sepsis, hemorrh exanthemata, or blood affections. The difficulties in diagnosis often great; in an anæmic old man, who suddenly vomited blood passed bloody urine, the diagnosis of purpura was made; the aut revealed a round ulcer of the stomach, and a papilloma of the blad *Scurvy* is distinguished by its gingivitis and muscular hemorrha and *hæmophilia* by reason of its hereditary facts. *Henoch's pur* is a type which occurs chiefly in children, and is characterized (*a*) by cutaneous lesions, which resemble erythema nodosum rather than purpura; (*b*) by hemorrhages of the mucous membranes; (*c*) by gastro-intestinal crises of vomiting, diarrhœa, and abdominal pain; (*d*) by pains in the joints, and (*e*) by frequent relapses, often over a number of years (*v. s.* Osler's group).

Prognosis.—The prognosis is uncertain. According to Gross, 50 per cent. die. Unfavorable symptoms are sudden onset, high temperature, and extensive hemorrhages. *Purpura fulminans*, of which Styler (1906) collected only 14 cases, may be fatal in 24 to 48 hours. Recurrence is not uncommon.

Treatment.—This has advanced but little beyond Werlhof's therapy of giving dilute sulphuric acid ♏x, and quinine gr. v after meals. Everything of a supportive nature, as sufficient nourishment, rest in bed, iron and arsenic, are helpful. The hemorrhages are arrested with difficulty, and the author doubts the value of drugs in their control. Turpentine and ergot may be given (as in hæmoptysis *q.v.*). Adrenalin solution to the nose and mouth, gelatin by mouth (see NEXT SECTION), and especially calcium chloride gr. v, t. i. d., to increase the coagulability of the blood, are indicated.

(G). HEMORRHAGIC DISEASES OF THE NEW-BORN.

Syphilis hemorrhagica neonatorum appears soon after birth. In some instances the syphilitic child may be born with hemorrhages in the skin, mucosæ, internal organs, and from the navel. Occasionally deep jaundice develops. The cause is probably vascular disease; Mrăcek found endarteritis, especially of the capillaries, in 14 per cent. of 19 cases. In other cases an added sepsis is apparently causative.

Winckel's disease, epidemic hæmoglobinuria, is (*a*) an affection of the new-born of unknown causation; (*b*) it appears epidemically in hospitals; (*c*) it develops in the first four days of life; (*d*) it is characterized clinically by fever, gastro-intestinal symptoms, icterus, dyspnœa, cyanosis, albuminuria, methæmoglobinuria and large mortality, and (*e*) pathologically by internal hemorrhages, fatty degeneration, and splenic tumor.

Morbus maculosus neonatorum is no clearer etiologically than Werlhof's disease in adults; some cases come under congenital syphilis; others with fever, jaundice, and epidemic occurrence under infection (pyogenic cocci, pneumococci, a bacillus like Friedländer's, pyocyaneus, and colon bacillus), and still others seem embolic, thrombi forming in the auricles when the heart is weak (Landau). Injuries to the head, causing hemorrhages in the fourth ventricle and cerebral peduncles, and other vasomotor disturbances (von Preuschen, and Pomarski) are questionable causes. It occurs once in 500 to 700 births.

Symptoms.—In the first week of life, hemorrhages occur from the navel and mucous membranes; in Townsend's 50 cases they occurred

in the following frequency: from the bowel (melæna), 40 per
navel, 36 per cent.; mouth, 28 per cent.; stomach, 28 per cent.
nose, 24 per cent. Death occurs within a week in 62 per cent. D
in 78 cases, found the mortality was 55 per cent.

Treatment.—Iron, ergot, and adrenalin are useless. Diffuse s
lants should be given when there is a weak, rapid pulse. Injecti
gelatin are dangerous, for it possibly contains some ptomaine, a
local use and administration by mouth are safer.

(H). SCURVY.

Definition.—A general disease, more often endemic than spo
caused by neglect of hygienic conditions, and characterized by
vitis, hemorrhages, inflammations, cachexia and chronicity of cou

Etiology.—It was first observed in the Crusades, and was desc
most fully by Vasco de Gama; in his voyage around the Cape in
33 per cent. of his sailors were stricken with scurvy. It has devel
not only among sailors, but among prisoners, Canadian lumbe
Pennsylvania miners, and among the workers on the Chicago Dra
Canal, where in 1893–95, the author saw 100 cases. Insufficient, m
onous, possibly spoiled, food causes the majority of cases, some
in conjunction with lack of fresh air, impure water, damp qua
and over-exertion; a lack of fresh meat, vegetables (potassium
malic and citric acids), and fats predispose to scorbutus, thoug
lack of no single group is solely responsible. The Chicago Dra
Canal cases were all Poles, and lived on coffee, bread, and pres
and fresh meat. It develops principally in middle-aged males
poorer classes. The disease is becoming less frequent. In
Admiral Gleary had 2,400 cases in the English fleet, and in 1849,
were 260,444 cases in Russia, and 60,958 deaths. It is still
endemic in Russia, where it is regarded as an infection.

Symptoms.—Scorbutus begins slowly, and progresses insidi
its earliest symptom is (1) *anæmia* (cachexia scorbutica). The
scaly, pale, lustreless, and may become pigmented. The mucous
branes are rather livid, and the expression is apathetic. The
findings are not characteristic; coagulation is slow, the red cell
hæmoglobin are proportionately reduced, and leukocytosis is a
except with intercurrent inflammation or severe hemorrhage. En
tion, mental depression, muscular weakness, fatigue, anorexia,
tion of HCl in the gastric juice, intestinal fermentation, constip
palpitation, weakness with irregularity of the heart's action, a sy
functional murmur, and œdema about the ankles accompan
anæmia. Splenic enlargement, albuminuria, peptonuria and o
are common.

2. *Gingivitis* begins with turgescence of the gums, which is du
serohemorrhagic infiltration, usually noticed first about the
teeth; the gums are spongy, painful, particularly on eating,

readily, and may become so swollen as to hide the teeth. Gingivitis is absent when there are no teeth or roots. The breath is extremely fœtid. In severe cases the gums necrose, and bleed profusely; deep ulcers, on healing, leave cicatrices; the teeth may loosen and fall out, the salivary and cervical lymph glands and tongue may swell. Necrosis of the jaw is uncommon.

3. *Hemorrhages* may occur (*a*) in the *skin,* especially on the extensor surfaces of the legs; small hemorrhages often occur, first about the hair follicles; they are at first small petechiæ, but later become larger and stretch the skin. In less frequent instances, the first eruption may resemble acne, herpes, or pemphigus, hemorrhages appearing later in these exanthems. The trunk is less frequently involved, and the face is usually exempt. As the hemorrhages increase, the legs are diffusely mottled with red, green, and black, and are swollen, tense and somewhat warmer than normal. Similar stains may be noted running upward in streaks along the lymph vessels. Some of these changes are due to (*b*) muscular hemorrhages (*scorbutic sclerosis*). They occur mostly in the calf and thigh, less in the buttocks, or abdominal wall. The legs are large, extremely hard, blood-stained, and later pigmented. As the disease regresses the muscles usually recover their function, but in some instances ulceration (scorbutic ulcers), suppuration, muscular cicatrization, or pressure atrophy and shortening of the tendons may leave permanent deformity and contractures. (*c*) *Mucous membrane hemorrhages* are less common than in purpura or hæmophilia, though epistaxis is fairly frequent. In some cases hemorrhages occur in the conjunctiva, air passages, and stomach, but in more cases in the bowels, perhaps with intestinal ulceration. (*d*) Of serous membrane hemorrhages, articular hemorrhage (hæmarthrosis) is the most frequent. Next in order of frequency are those in the pleura, pericardium, peritoneum and meninges. They are usually sudden in onset, extreme in degree, and unfavorable in outcome. (*e*) Parenchymatous hemorrhages are least common, as in the eye-tissues, brain, and other viscera. Subperiosteal and epiphyseal hemorrhages are rare, unless Barlow's disease is included (see NEXT SECTION). Scorbutic ulcers in soft tissues may reach the bone, causing necrosis.

4. Scorbutics are subject to *various inflammations.* (*a*) Painful *joint* swellings are the most frequent, and occurred in 35 per cent. of the author's cases, and chiefly in the knees; they may be serous or hemorrhagic, or in severe cases the cartilages or bones may become eroded and ulcerated; sometimes suppuration intervenes; deformity and ankylosis are possible sequences. (*b*) Hemorrhagic pericarditis and pleuritis are very dangerous. (*c*) Onychia, paronychia, choroiditis, and panophthalmitis are occasional incidents. Fever is uncommon, except from extreme inanition or terminal infection.

Course and Prognosis.—The onset is insidious, the course chronic, and convalescence protracted. Most cases recover, after which recurrence is not frequent. Death may occur (*a*) from cachexia, and the autopsy then shows fatty degeneration of the heart, liver, and bloodvessels; (*b*) from profuse hemorrhages, into the pleura, pericardium, gastro-intestinal tract, or rarely into the brain, or meninges; (*c*) from infections, as dysentery, pleurisy, pneumonia, or septicopyæmia, probably of the terminal type, and promoted by the reduced physiological resistance.

Diagnosis.—The diagnosis is usually made without difficulty from (*a*) the anæmia or cachexia, (*b*) gingivitis, (*c*) tendency to hemorrhage, and (*d*) inflammation, (*e*) the chronic course, and (*f*) the temporary nature of the condition (as contrasted with the most permanent tendency to hemorrhage in hæmophilia).

Treatment.—(*a*) *Hygienic;* avoidance of "cured" or smoked foods is both prophylactic and curative. In Nansen's polar expedition, which lasted three years, not one case of scurvy developed. His meats were cooked, and plenty of cooked and raw fruits and vegetables were used. Berries, sauerkraut, potatoes, radishes, cress, apples, oranges, and lemons are especially valuable. A monotonous dietary is particularly to be avoided. (*b*) The *anæmia* (*q.v.*) should be treated by strychnine, quinine, iron, and arsenic. Yeast ℥ij two or three times daily, is recommended. (*c*) The *gingivitis* is treated by occasional local applications of silver nitrate (10 per cent.). Cocaine may be painted on the gums before meals, so that the patient may be able to eat, but great care in its use must be taken, lest the habit be contracted, or toxic symptoms develop. (*d*) *Symptomatic treatment;* hemorrhage is benefited by rest only. Stimulation is required in intercurrent pericarditis and pleuritis. Drastic cathartics are to be avoided. Complicating arthritis necessitates rest and the application of cold, but rarely aspiration or drainage.

(I). INFANTILE SCURVY—BARLOW'S DISEASE.

Möller (1852–62) described the affection as acute rhachitis, and Ingerslev (1871), Jalland (1873), Cheadle and Gee (1878), but particularly Barlow (1883), described a cachexia with hemorrhages which occurred in the very young. De Bruin observed 61 cases in Holland in 1893. In 1898, 379 cases were collected in the United States.

Etiology.—Unlike scurvy in adults, the hygienic conditions are good in 80 per cent. of cases, and it occurs largely in the better classes. (*a*) The obvious *cause is feeding* with malted, condensed or cooked milk or baby foods; these foods lack some unknown substance which is essential to health. The malady is very rare in breast-fed infants. In 713 cases (S. Weiss, 1905) only 20 or 2.5 per cent. occurred in infants at the breast. (*b*) Ninety per cent. of cases occur between the sixth and twelfth month (Heubner's and Neumann's 94 cases).

Symptoms.—These are not always typical; there may be anæmia ("cachexia") without hemorrhages, or hemorrhages without anæmia, though in typical cases they occur together.

1. *Subperiosteal hemorrhage* is the leading symptom. There may be at first some vague symmetrical swelling over the epiphyses, which later becomes more obvious, though no real œdema is noted. There is pain and tenderness usually in the thighs or legs, which are drawn up, flexed or everted, immobile and semiparalytic (pseudoparalysis). At autopsy, the anatomical findings are subperiosteal hemorrhages; a colloid and fibrillar alteration in the marrow of the diaphyses; arrest of ossification; decrease of the osteoblasts; rarefaction of the bone, sometimes leading to fractures (spontaneous or after slight trauma), or to epiphyseolysis (usually at either end of the femur or the upper end of the tibia); the joints are normal. Seventy per cent. of the lesions is in the thigh, 24 per cent. in the leg, and 6 per cent. in the orbit; the arms, ribs, spine, skull, sternum, and scapula, are not often involved. Rib involvement results in depression of the sternum.

2. The *cachexia* is evidenced by the anæmia (usually moderate and sometimes attended by slight lymphocytosis), sallow skin, asthenia, anorexia, constipation, irregular temperature (in 66 per cent.) or subnormal registration and the almost invariable severe sweats.

3. *Gingivitis* is noticed when the teeth have already erupted or are about to erupt. In 73 cases (Heubner and Neumann), the gums were normal in but 15 per cent.

4. Other symptoms embrace hemorrhages in the skin in 15 per cent., from the kidneys in 9 per cent. (hemorrhagic nephritis), and rarely in the conjunctivæ, muscles, stomach, bowels or meninges. The spleen is rarely swollen.

Diagnosis.—Diagnosis is not difficult, after one typical case has been seen; it is based upon the etiology, subperiosteal hemorrhages, gingivitis and anæmia (cachexia). Confusion with genuine paralysis is unnecessary, for the immobility is voluntary to escape the pain of movement. Syphilitic bone disease (syphilitic pseudoparalysis or Parrot's disease) is rather similar as to the crepitus, epiphyseolysis and pain, but other syphilitic stigmata or scorbutic symptoms are generally present.

Prognosis and Treatment.—Proper treatment secures marked and immediate improvement in the great majority of cases. Death is uncommon; in unfavorable cases, it occurs in the third or fourth month of the disease.

The diet consists of raw cow's milk, potato purée, and beef juice (not broth or extracts); orange juice, apple sauce, pears, cooked berries and spinach are most beneficial. Treatment by drugs is generally superfluous.

(J). HÆMOPHILIA.

Definition.—A congenital and hereditary affection, characterized by deficient coagulability of the blood, and a consequent tendency to various profuse hemorrhages, which are spontaneous or traumatic, and are most difficult to arrest.

Etiology.—Its real cause is unknown, and the few pathologic changes described in the blood and the bloodvessels are insufficie factors. Sahli believes that there is an hereditary deficiency in t fibrin-forming substances (thrombokinase). (*a*) *Heredity* is the on clear etiological fact, which Fordyce noted in 1784; as Grandidi remarks, hæmophilia is "the most hereditary of all hereditary diseases Osler quotes Brown as observing hæmophilia through seven generation Sometimes a generation may be exempt. (*b*) Broadly speaking, t *males* of the "bleeder families" are the "bleeders," and the femal transmit the disease to their male offspring without manifesting t disease themselves. In 200 families of "bleeders," Grandidier foun 609 cases in males, and 48 in females (8 per cent.); Stahel in 1,8 families found only 24 cases in males. In the Mampel family, fir reported by von Chelius in 1827, Lossen (1905) found 212 membe of which 111 were males; of these exactly one-third were bleeder and 48.7 per cent. of the male bleeders died of hemorrhage; none the females were bleeders and all transmission of the disease occurr through the women. (*c*) The disease occurs most frequently in Ge many, England, North America and France. Consanguinity and fami tendencies (gout, rheumatism, or tuberculosis), have been suggest as causes. "Bleeders" are generally healthy individuals with so delicate skins.

Symptoms.—In 75 per cent. of cases, *hemorrhages develop* in the *fi* *two years of life;* rarely do they first appear after puberty or in t third decade, though isolated observations are recorded of perso having their first hemorrhages after the seventieth year. The hem rhages are spontaneous or traumatic; they most often occur as cap lary oozing from a mucous surface; deep interstitial bleeding is m unusual. They are copious and hard to still. Epistaxis is the m common (50 per cent.); hemorrhages from the gums (12 per cent intestine (12 per cent.), lungs, kidneys and stomach (each 6 per cen are next in frequency. Uterine hemorrhage and arthropathies a not infrequent. According to König, the joint findings are (*a*) hæmarthros, with pain, swelling and fluctuation; (*b*) then panarthrit whose gross pathology resembles tuberculous arthritis, with flexion the joint; (*c*) regression, or ankylosis and deformity, which rath resembles arthritis deformans. The knees and elbows are most oft affected. Subcutaneous hemorrhages may occur. The first blee ing may occur from the navel, or may result from vaccinatio extraction of teeth, circumcision, from a trifling prick or cut, fro delivery, or from rupture of the hymen on the wedding night. I various attacks, one part after another may bleed or the hæmophil disposition may be confined to one part, as a kidney (renal hæmophilia Hæmophilia differs from purpura, in that the bleeding seldom occur into a serous membrane (excepting the joints), or into the viscera The amount of blood lost may be enormous, and in one case amounte to 24 pounds in eleven days. The coagulation test of the blood may show that coagulation is delayed to thirty or forty-five minutes (Wright); the clot, when formed, is porous.

Of *general symptoms*, there are few except changes in the joints (like those in subacute rheumatism), a neuropathic tendency (neuroses and neuralgia, particularly of the fifth nerve), and anæmia (though hæmophilics tolerate loss of blood better than other individuals, show it less, and recover from it more readily).

Diagnosis.—The diagnosis may be for a time uncertain, when hemorrhage develops after an operation, or occurs from one organ only, as the kidney. The family history clearly stamps the affection, and readily differentiates it from scurvy, purpura and hemorrhages in the new-born.

Prognosis.—The prognosis is grave. Any hemorrhage may prove fatal. Sixty per cent. of cases die before the eighth year, and only 11 per cent. reach the twenty-second year; if middle life is reached, the danger gradually lessens. It is more favorable in girls, despite the apparent menstrual dangers. De Bons (1905) found 69 excessive hemorrhages among 150 deliveries in hæmophilic women; he believes pregnancy has a rather favorable effect on the disease. De Lee has reported premature detachment of the placenta in hæmophilia. The rudimentary or local forms are more favorable than general hæmophilia.

Treatment.—1. Prophylaxis.—Grandidier lays down the following rules: (*a*) women, with an hæmophilic family tendency, whether they are "bleeders" or not, should not marry. (*b*) Men, if not "bleeders" themselves, may marry. (*c*) Men should not marry when their family history indicates that male "bleeders" have begotten hæmophilic children. It is obvious that in hæmophilic families, general measures to elevate the physiological resistance, and avoidance of trauma or any but life-saving operations, are indicated. Vaccination is rarely contra-indicated.

2. Control of Actual Hemorrhage.—This is almost impossible. (*a*) Wounds should be cauterized or enlarged, as Fordyce discovered that in hæmophilics large bleed less than small wounds. Other surgical hæmostatic measures need not be further considered. The Esmarch constrictor controls some hemorrhages of the extremities. (*b*) Styptics, as Monsell's solution, or tannic preparations, are useless. (*c*) Adrenalin, locally and internally, has no apparent affect. (*d*) Calcium chloride is somewhat more beneficial, grs. iij to v t.i.d. should be given; it should be given intermittently, because protracted administration decreases the coagulability of the blood. (*e*) Gelatin may be given by mouth or in unusually severe hemorrhages by sub-cutaneous infusion (*v.* HEMORRHAGES OF THE NEW-BORN). (*f*) Saline cathartics are sometimes apparently efficacious, possibly by diverting blood from the seat of hemorrhage. (*g*) Very acute anæmia necessitates injection of salines under the skin; when the hemorrhage is over, regeneration of the blood is remarkably rapid. (*h*) Joint involvement is treated by rest, immobilization, and rarely aspiration.

SECTION VII.

DISEASES OF THE DUCTLESS GLANDS.

(A). DISEASES OF THE SUPRARENAL GLANDS.

ADDISON'S DISEASE; OTHER AFFECTIONS OF THE SUPRARENAL GLANDS.

Definition.—A disease characterized (*a*) pathologically by lesions (usually tuberculous) in the adrenal glands or by degenerative changes in the adjacent abdominal sympathetic ganglia, and (*b*) clinically by langor, muscular and mental adynamia, digestive disorders, pigmentation of the skin and a fatal issue.

Etiology.—1. **Suprarenal disease** was first associated pathologically with melanoderma by Addison, in 1855. By far the most common lesion is (*a*) chronic fibrocaseous *tuberculosis* of the adrenal glands; the tubercles of the adrenal glands are usually bilateral, destroy most of the suprarenal structure, and frequently involve the semilunar ganglia and solar plexus by their attendant formation of connective tissue; in rare cases only is the adrenal tuberculosis primary, though cases were recorded by Symes and Fisher; there is usually a primary focus in the lungs (in over 50 per cent.), in the genito-urinary, alimentary or glandular system. (*b*) Less common are tumors, simple atrophy, fibrous induration (usually tuberculous), cirrhosis (26 cases recorded), gumma, amyloid degeneration, acute inflammation (as by contiguity from perinephric abscess), acute hemorrhage, embolism, echinococcus, traumatic adenoma, or other very rare lesions. In what way suprarenal lesions produce Addison's *melasma suprarenale* is still a disputed question, for our knowledge of adrenal physiology is incomplete. It seems (i) that the glands contain a substance (sphygmogenin) which maintains vascular

tonus, and stimulates the vasomotor centres and nerves, th
and respiration; when secreted in excess, as in hypernephro
blood tension may be much elevated (Fraenkel, Neusser, I
and this may induce cerebral hemorrhage; (ii) the glands ap
elaborate a substance which is necessary to life; this internal s
probably renders less toxic some substances produced by other
e.g., the "fatigue stuffs" of muscle metabolism. Chemically,
mogenin, pyrokatechin, neurin and lecithin have been found
holds that the active glandular substance is epinephrin.

There are, however, cases of Addison's disease, in which the a
are normal. Lewin's statistics from 500 cases are as follo
typical cases, the adrenals are diseased in 88 per cent. and ap
sound in 12 per cent.; on the other hand, disease of the adr
attended by pigmentation of the skin in 72 per cent., and una
by it in 28 per cent. There is no wholly satisfactory explana
such cases. Perhaps compensatory hypertrophy or accessory
may prevent symptoms in some cases of adrenal disease.

2. **The adjacent sympathetic structures**—the solar plexus an
lunar ganglia,—may be involved directly, or indirectly as by
changes in the adrenals, and pressure from contiguous aneu
lymphomata. Pigmentary atrophy, hyaline degeneration an
cell infiltration of their vessels, and degeneration extending
to the cord, or downward along the nerve fibers have been de
Some writers explain Addison's disease as a disease of the sym
system, though others endeavor to reconcile conflicting facts by
ing that there is decreased adrenal activity and also sym
disturbance.

3. Minor etiological factors are (*a*) *age;* most cases occur
the fifteenth and fortieth years; the youngest patient recorde
congenital case, and the oldest one was eighty years; (*b*) *sex;*
cent. occurs in males; (*c*) *social* status; 90 per cent. is found
laboring classes.

Symptoms.—1. Marked *physical and mental adynamia* is
the initial symptom and persists, with increase after possible
sions, from onset to death. The insidious langor, weakness,
lack of initiative and muscular relaxation contrast sharply w
healthy appearance of the patient, at least in the early stages.
symptoms are probably toxæmic, partly from reduced interna
tion and partly from the tuberculous lesion. Obstinate e
somnia and headache, and somewhat later attacks of vertigo, t
spots before the eyes and syncope are frequent. The inte
functions are somewhat weakened, neuralgias occur in the epiga
intercostal spaces, back and extremities, periodic attacks of irri
are frequent, and the deep reflexes are usually somewhat reduc

2. *Digestive symptoms* generally appear next. The tongue i
what coated and moist; there are anorexia, thirst, a sense of abd
fulness or pressure, eructations, and sensitiveness over the ab
Paroxysmal pains in the epigastrium or back radiate widely, es

into the chest; they resemble tabetic crises and are most severe. *Vomiting* is the most distressing, ominous, and intractable alimentary symptom; it may follow eating or occur independently of it. Though at first intermittent, it later hastens the fatal issue. Early constipation gives way to terminal diarrhœa.

3. *Pigmentation* (melanoderma, bronzed skin, *melasma suprarenale*) develops after the adynamia and alimentary disturbance. Its cause is obscure; Neusser refers it to involvement of the sympathetic nerves or ganglia. It is found in the deep layers of the rete Malpighii, to which it is apparently brought from the blood by leukocytes. At first the *skin* has a dirty, yellow-gray color, and small areas appear which gradually fuse, and then slowly become darker and more diffuse; extended pigmentation may occur with focal areas of deeper discoloration. Pigmentation develops particularly (*a*) where it is normally deepest, as about the nipples, anus and genitalia; (*b*) where pressure or friction is greatest, as over folds of the skin, over the clavicle, spine, ischia, scapulæ or waist (from belts or corsets); (*c*) where there is exposure to the sun, as in the face, neck or arms; (*d*) where accidental irritation occurs, as by blistering, eczema or parasitic affections. The soles of the feet, palms of the hands, nail-beds and hair are seldom stained. Previous scars may or may not be stained; sometimes they are surrounded by a zone of deeper discoloration. The skin is dry, is sometimes bathed with profuse perspiration and may have a fish-like odor. Scattered spots of pigment may atrophy (vitiligo) and impart to it a strange appearance. The *mucous membrane* of the lips, gums, cheek and palate also show pigmentation, but usually later than the skin. The spots and horizontal ink-like stains on the cheek where the teeth touch it are particularly of diagnostic value. The conjunctivæ are pearly white, and are usually said to be exempt, though frequently small aggregations of pigment can be seen on careful examination (Leva). Staining of the larynx, nymphæ, vagina, glans penis, serous membranes and internal organs is infrequent.

4. *Other findings* embrace the following: (*a*) The *heart* shares in the general adynamia; it frequently shows brown atrophy at autopsy; its tones and apex are weak. Pain over the heart is frequently reflected from the abdomen; palpitation and dyspnœa are common, but functional murmurs are rare. The pulse is compressible and somewhat accelerated, seldom slow or arrhythmic. The peripheral vessels and abdominal aorta pulsate actively. Atheroma is common, even in young subjects (Kolisko). (*b*) *Anæmia* was considered characteristic by Addison, though Nothnagel and others have demonstrated its infrequency; in a recent case, the writer observed a red-cell count of 2 million. Increase in the red cells (polycythæmia rubra), and in the hæmoglobin results from concentration of the blood, which is due to vomiting and diarrhœa. The lymphocytes are somewhat increased. (*c*) The *lymph nodes* and Peyer's patches are sometimes hyperplastic. The spleen, thyroid, thymus and pineal glands are often enlarged,

gia; (*c*) pigmentation of the skin and oral mucosa; (*d*) c ness and (*e*) the chronic lethal course.

As Addison's disease usually indicates the presence of the advisability of injecting tuberculin may be considered however, may easily occur from tuberculosis in other tissu tumors may cause metastases in the brain or (like tumors o prostate and mammæ), in the long bones, skull and spine

Clinical Course.—The cardinal symptoms described pro with occasional intermissions, and in nearly all cases, after a year or two; the terminal symptoms are often v may be delirium, hallucinations, convulsions, incoercible diarrhœa, irregular respiration, small pupils, occasional ingitic symptoms and finally collapse and coma. The vary: (*a*) *acute cases* may resemble typhoid, digestive dis poisoning; (*b*) *chronic cases* sometimes last ten or th (*c*) *sudden death* may occur before pigmentation develop renal lesion at autopsy disclosing an anomalous type (*fo* Addison's disease; (*d*) the course may be modified by intestinal *tuberculosis.*

Differentiation.—*Pigmentation* occurs in numerous oth (*a*) poisonings by silver (*argyria*), in which the reduced a gray deposit in the extracellular tissue of the skin, sweat junctiva and nail-beds; by *arsenic,* which seldom stains in *pellagra,* which is endemic and intermittent in occu exacerbations in the spring and fall, is preceded by eryth associated with tuberculosis, but with eosinophilia, di voracious appetite and is exempt from mucous membr tation (Neusser). (*b*) Chronic *icterus* (which, howeve scleræ and urine), *cyanosis* (arteriosclerosis and heart sometimes pigmenting the oral mucosa), various physi mentations, and dirt and parasites as in tramps (vagab *Abdominal diseases,* dyspepsia, gastric ulcer, tumor (phomata, and Hodgkin's disease), hypertrophic cirrhosi exophthalmic goitre or rarely scleroderma. In these conditions, pigmentation of the oral mucosa is rare.

Treatment.—1. **Supporting and Tonic.**—Absolute rest is imperative because of exhaustion and proneness to lethal syncope. Alcoholic stimulation is poorly tolerated. Careful feeding is necessary because of the sensitiveness of the stomach and bowels. Strychnine, arsenic, nitroglycerine, faradization and galvanization may be given.

2. **Symptomatic.**—Narcotics for pain, gastric sedatives for vomiting (*v.* GASTRITIS), astringents and opiates for diarrhœa (*v.* ENTERITIS) should be administered; strong cathartics are always contra-indicated, for they may precipitate fatal collapse; one case in the Cook County Hospital died suddenly after taking a dram of compound jalap powder.

3. **Organotherapy.**—Adrenal extract by mouth has surely helped some cases and apparently arrested the development of others (Oliver, Sansom, Lloyd, Jones, Osler, Ringer, Langlois, Dupaigne). In Adam's review of 97 cases, one-third were benefited, and one-sixth recovered. The medullary portion of the gland is best. It may increase the vascular and muscular vigor or even lessen pigmentation. Some practitioners advocate the hypodermatic administration of adrenalin chloride, ʒj of 1 to 1,000 in salt solution, on alternate days. Some advanced cases are unfavorably affected by organotherapy.

Other affections of the suprarenal glands are of slight clinical interest. (*a*) In *developmental anomalies*, the glands are sometimes absent or hypoplastic, chiefly with other defects, as encephalocele, etc. (*b*) *Hypertrophy* in one gland may compensate for hypoplasia in the other. *Supernumerary* glands are not uncommon. (*c*) *Atrophy* is usual in advanced age. (*d*) *Degenerations* embrace the parenchymatous, fatty, hyaline and amyloid types. (*e*) *Circulatory disorders* are hyperæmia, embolism, thrombosis and hemorrhage, which latter may occur in trauma in the new-born; tumors, inflammation and the hemorrhagic diathesis. (*f*) *Inflammation*, acute and chronic, is uncommon. (*g*) *Granulomata*, chiefly syphilitic and tuberculous, are rarely primary. (*h*) *Tumors*, carcinoma, sarcoma (20 recorded, Affleck and Leith), and hypernephroma (*v.* KIDNEY TUMORS) are chiefly of anatomical importance. In 23 published operations, the mortality was 50 per cent., recurrence developed in 21 per cent., and 29 per cent. recovered (in nine months to six years). At the Johns Hopkins Hospital, three tumors of the suprarenal glands were successfully operated on (Osler). (*i*) Parasitic disease (echinococcus) is extremely infrequent.

(B). DISEASES OF THE SPLEEN.

Lesions of the spleen are usually secondary.

ACUTE SPLENIC TUMOR.

This occurs (*a*) in acute infections, due either to the local action of microörganisms and their toxins, or to toxæmic paralysis of the splanchnic nerve, which normally controls the vascular supply of the organ; it is most common in typhoid, malaria, recurrent fever and septic conditions; (*b*) in acute stasis, usually from portal obstruction; and (*c*) in embolism (*v.i.*) and trauma. *Symptoms* are infrequent; much pain usually indicates involvement of the capsule (perisplenitis). On objective examination: (*a*) The spleen may sometimes be seen, during deep inspiration. (*b*) Palpation alone is wholly reliable. By the bimanual method and with the examiner sitting at the right side of the patient, the edge of the spleen comes in contact with the fingers and slips sharply down below them; splenic crenations are seldom palpable. A palpable spleen always indicates enlargement, or much less often, downward dislocation by pleurisy, emphysema, pneumothorax or thoracic tumor. (*c*) Percussion, which is valuable in outlining the upper splenic level, is unreliable in determining enlargement, for confusion with fæcal accumulations is frequent.

CHRONIC SPLENIC TUMOR.

Chronic enlargement is symptomatic of (*a*) chronic infections, as malaria, tuberculosis and syphilis, (*b*) continued portal stasis, (*c*) repeated embolism, (*d*) blood affections, as leukæmia, pseudoleukæmia or splenic anæmia, and (*e*) rickets, amyloidosis, neoplasms, cysts, polycythæmia (*v.i.*), arthritis deformans and chronic acetanilide poisoning.

EMBOLISM.

Embolism occurs frequently in the spleen, because of the wide arteries and slow current; it ranks second in frequency to kidney embolism. It usually develops in the course of acute endocarditis, chronic valvular lesions or arteriosclerosis and seldom follows local lesions as sinus thrombosis or splenic vein thrombosis. The *symptoms* vary somewhat as the embolus is simple or infective; sudden chill, fever, pain in the side, acute splenic tumor and perisplenitic friction may develop, which with a cause or embolic symptoms in other organs justify a diagnosis of embolism. Frequently no distinctive symptoms occur.

If septic, symptoms of abscess may be present (*v.i.*). Treatment is wholly symptomatic.

ABSCESS (SPLENITIS APOSTEMATOSA).

Abscess of the spleen may result from embolism, trauma or invasion by contiguity. Swelling, pain, perisplenitic friction or possibly unevenness of surface or fluctuation may mark its development. It is more often hidden by the symptoms of the causal focus. It may rupture into the pleura, kidney or peritoneum. Aspiration may determine the diagnosis, and surgical evacuation is then indicated.

PERISPLENITIS.

This affection may be primary or secondary, simple or suppurative, an isolated lesion, or part of a chronic peritonitis (see "ICING LIVER," CHRONIC PERIHEPATITIS). It usually accompanies the various splenic affections already enumerated. Without local peritonitic friction its diagnosis is impossible. There is no efficient therapy.

AMYLOID SPLEEN.

Its etiology is considered under amyloid liver and kidney. Pathologically its two types are (*a*) the sago spleen, in which there are amyloid deposits in the vessels of the Malpighian tufts and (*b*) the more important, diffuse amyloid infiltration. It is diagnosticated by (i) the etiological factors, (ii) its usually large size, hardness, smoothness and thick edge, and (iii) coincident amyloidosis of the liver and kidneys.

SPLENIC ANÆMIA.

Splenic anæmia (*v.* DIAGNOSIS OF PSEUDOLEUKÆMIA). Springthorpe observed six cases in one family. Umber advises operation in every case, because he believes that the spleen destroys the red cells. The *x*-rays have been of therapeutic value.

TUMORS AND CYSTS.

New growths are very infrequent. They are more often secondary than primary. They include carcinoma, sarcoma (of which 19 primary cases were reported by Adolph, 1905), enchondroma, and lymphangioma. They are rarely differentiated from splenic anæmia and like affections. The chronic endothelioma of Gaucher clinically resembles splenic anæmia; pathologically, large endothelial cells replace the splenic tissue; this can be seen under the microscope or macroscopically, as in Stengel's case.

Echinococcus cysts are found in but 0.2 to 3 per cent. of echinococcus disease, and usually with concomitant cysts in the liver. Dermoid cysts are exceedingly uncommon. Only 13 non-parasitic cysts are found in the literature.

Granulomata (see TUBERCULOSIS AND SYPHILIS).

RUPTURE OF THE SPLEEN.

Rupture may be traumatic or spontaneous; the latter variety has been observed in embolism, malaria, typhoid, acute leukæmia, and pregnancy. It is more common in India, and tropical climates than in temperate zones. Aspiration and even palpation have occasioned rupture. The treatment is surgical. Up to 1905, 46 splenectomies for rupture were reported (Faltin).

MOVABLE OR FLOATING SPLEEN.

Permanent mobility (*lien mobile*) and dislocation are due to the same factors observed in enteroptosis, with which it may be associated: (i) lax, elongated or ruptured ligaments (ligamentum gastrolienale, phrenicolienale, and most important, ligamentum phrenicocolicum); (ii) relaxed abdominal walls; and (iii) enlargement and increased weight of the spleen operating with the above factors.

Symptoms.—Symptoms are often absent. Pain or a dragging sensation is frequent, which is either local or radiates to the thorax, left shoulder or legs, similar to that observed in neurotics or women with uterine or ovarian disease. There may be headache, insomnia, psychical alteration, dyspepsia, constipation, menstrual anomalies, vesical tenesmus, and paræsthesia in the legs; most of these are neurotic. *Objectively:* (*a*) The most important sign is *palpatory detection of the spleen in an abnormal location.* (i) It is located most often in the left ilio-inguinal region, near the ilium, but may lie just below the left costal arch, near the navel, in a hernial sac, in the pelvis, or above the pubes. (ii) Its hilum usually points upward, either directly, if it lies across the vertebræ or in the pelvis, or with slight inclination to the right or left if it lies to one side. (iii) The form, notches and possibly the pulsating vessels are those of the spleen. (iv) In size it is usually enlarged, either from some causal hypertrophy or some later congestion; torsion of its pedicle may lead to softening, atrophy, gangrene or even liberation of the spleen as a free body. (v) As a rule it can be replaced, and its position changes with posture; perisplenitis, causing a friction rub, may result in firm adhesions to the colon, bladder, rectum, or uterus, thereby causing great or insuperable nostic difficulty; in such cases the spleen is dislocated, but ceases wander." (b) The *spleen is absent from its normal location,* the nic area being replaced by tympany from the colon (which changes ulness when water is introduced into the colon). (c) *Pressure ptoms,* intestinal obstruction, gastric dilatation, vesical and rectal pression may be noted.

iagnosis.—Diagnostic confusion with floating kidney, and pedun- ted ovarian tumors is rather infrequent, when the abdomen and s are carefully examined. The author knows of three laparotomies rmed for supposed uterine or renal disease, in which abundant sions rendered differentiation impossible. On the other hand, rd, immovable suprapubic tumor was correctly diagnosticated ukæmic spleen from the glands and blood findings.

reatment.—(a) *Bandages* are more difficult to apply than for able kidneys, though some cases can be benefited by them. (b) spleen may be replaced by operation, and its vicinity well packed gauze; the resulting adhesions often retain the viscus. (c) *nopexis* (Rydygier) may be performed, the spleen being sewed lace or deposited in a peritoneal pocket. Stierlin (1897) collected perated cases, in which the mortality was 6 per cent. (d) *Splenec-* (see PSEUDOLEUKÆMIA) may be performed as a last resort, the tality being about 27 per cent.

SPLENOMEGALY, WITH CHRONIC POLYCYTHÆMIA, AND CYANOSIS.

his condition has been described particularly by Osler, Weintraud Türck. About 34 cases are reported up to 1905. (a) The *red are increased,* even to 9 or 13 million (polycythæmia rubra or globulism); the hæmoglobin may reach 150; the white cells number n 4,000 to 31,000. The viscosity, specific gravity and volume of blood are increased. (b) There is *chronic cyanosis,* without adequate se and (c) *splenomegaly.* There may be headache, constipation, iting, and an hemorrhagic diathesis. The arterial tension may be eased and the heart may hypertrophy. De Lee sent the author it appeared to be a *forme fruste,* a case with splenomegaly and nosis but without polyglobuly. The *etiology* is obscure; in about third of the cases it was tuberculosis of the spleen and 8 of the 11 rated cases recovered; stasis may be the cause, *e.g.*, a malignant wth compressing the superior vena cava; it seems probable that nost cases the cause is an undue activity of the bone-marrow. The *tment* is x-rays, venesection, iodide of potash or splenectomy.

(C). LYMPHATISM (STATUS LYMPHATICUS).

Lymphatism is a condition in which there is hyperplasia of th various lymphoid and kindred tissues, and a tendency to sudde death. It is also known as the *constitutio lymphatica*, *status thymica* and *status lymphaticus.*

Its *etiology* is most obscure. The affection is rare and occurs mo often in women and children. Hedinger observed it as a famili affection.

Symptoms.—1. There is *lymphoid hyperplasia.* (*a*) The *lymph glands* of the chest and abdomen are more often involved than the external, cervical, axillary or inguinal glands. (*b*) The *tonsils*, nasopharyngeal, lingual and intestinal lymphadenoid tissues are frequently hyperplastic. (*c*) Moderate *splenic enlargement* is usual. (*d*) The *thymus* is swollen, sometimes giving retrosternal dulness. (*e*) The *bone-marrow* often changes to a reddish color.

2. *Associated findings* may include hypoplasia of the heart and aorta, thyroid enlargement, poor development, infantilism, rickets, tetany, and laryngismus.

3. *Sudden death* is often the first evidence of the condition and may occur without seemingly adequate cause. (*a*) It has occurred during or after *anæsthesia*, especially with chloroform. In one case, a patient with enlarged glands died suddenly after receiving but 20 drops of chloroform. (*b*) Operations with *local anæsthesia* have also been fatal. (*c*) Administration of *antitoxin* has caused death, as in the well-known case in Prof. Langerhans' family. (*d*) It may occur while *bathing* or after *falling in the water.* (*e*) Sudden deaths during convalescence from *acute infections*, or some collapses during hydrotherapeutic treatment (Escherich) may be attributed to the lymphatic constitution. (*f*) Children with eczema may die suddenly; lymphatism causes 75 per cent. of these deaths. It is said to be due to cardiac excitability (Kundrat), to lessened physiological resistance with a tendency to heart paralysis (Paltauf), and to a toxic condition (Blumer, who suggests the term lymphotoxism.)

(D). DISEASES OF THE THYROID GLAND.

GOITRE.

Definition.—Hypertrophy of the thyroid gland. Sporadic cases are common in Europe and America. Goitre occurs endemically in Switzerland and Italy, though we have no knowledge of its cause; the drinking-water has been thought causal. It is usually acquired, and is seldom congenital.

There are *three main forms:* (*a*) the *parenchymatous* or follicular ype, with more or less symmetrical enlargement, and formation of olloid material in the hyperplastic follicles; (*b*) the *cystic*; and (*c*) *ascular* or congestive type, with dilatation of the bloodvessels. The ast form borders closely on those almost physiological variations n the organ, which are observed at puberty, in defloration, pregnancy, hlorosis and psychical trauma.

Symptoms.—1. The *thyroid enlargement* may be diffuse, as in the oft, symmetrical and moderately vascular form, or partial or nodose, mplicating but one lobe or the isthmus. In most cases there are only ocal findings.

2. *Compression* symptoms are not common. The carotid artery nd jugular vein are pushed to one side, and the sternomastoids ecome thin. The trachea may be compressed, its lumen becoming haped like a sword scabbard; asthma, dyspnœa, bronchiectasis and ilatation of the right ventricle follow stenosis of the air-passages. aryngeal paralysis occurs in over 10 per cent. Fraenkel states that n 25 per cent. of cases in which there is tracheal stenosis, there is lso laryngeal paralysis. From the isthmus or lower horns of the hyroid, goitres may grow in a retrosternal or retroclavicular direction 5 to 10 per cent.) causing compression of the vagus, sympathetic erves and vena cava. A goitre may disappear into the chest with ach inspiration and appear on expiration (*goitre en dedans*). It may ecome incarcerated in the chest.

3. An *aberrant or accessory thyroid* may occasionally form a tumor, nywhere from the tongue to the aorta, and from the spine to the nterior chest wall, in the retropharyngeal or retroœsophageal tissues (causing dysphagia or dysarthria), in the trachea, larynx, root of the tongue, pleura or mediastinum; it may compress an entire lung. Storrs (1904) collected 32 lingual goitres; there are three cases in the literature of retrosternal accessory thyroids, which became cystic and calcarious (Collins, 1905), and fourteen which were intratracheal (Pfeiffer, 1905).

4. *Sudden death* may occur in large goitres; it may be due to pressure on the vagus, spasm or œdema of the glottis, sudden hyperæmia of the gland and pressure on the trachea, hemorrhage dissecting into the chest, or status lymphaticus.

Diagnosis.—Confusion is possible with *tumors* as sarcoma, carcinoma or adenoma, for whose differentiation and symptoms reference to surgical treatises should be made. There are 23 cases in the literature of *echinococcus cysts*. In 83 per cent. of cases, *cancer* develops from a preëxisting goitre; early metastases are specially common and characteristic; only 10 per cent. recovered among 110 operations (Madelung). There are 20 cases recorded in which *metastases* throughout the body occurred *from benign tumors* of the thyroid (chondroma or myxoma). Thyroid inflammation (*strumitis*) is usually secondary (metastatic) or traumatic, rarely primary; it may occur in sound or goitrous glands; it is most often due to the streptococcus but may be caused by the colon, typhoid, pneumonia, diphtheria or other organisms.

Treatment.—In regions where goitre is prevalent, the drinking water should be boiled. *Medical treatment* is of uncertain value. Iodine may be used externally or by injection (Maunoir, 1825). Potassium iodide (Coindet, 1820) is beneficial in some subacute cases, according to Bruns, in 75 per cent.; Kraus found it particularly beneficial in the vascular type, with cardiovascular symptoms, as moderate tachycardia and full arteries. The *x*-rays are valuable in some cases. Thyroid extract is occasionally beneficial. *Surgical treatment* is indicated in large or growing goitres. Reverdin's collection of 6,103 operations shows a mortality of 2.8 per cent., and in Kocher's series, now several thousand, but one death occurred in each 600 operations. See CACHEXIA THYREOPRIVA, under MYXŒDEMA.

EXOPHTHALMIC GOITRE.

Definition.—A malady probably due to excessive activity of the thyroid gland, and characterized by three cardinal symptoms, exophthalmos, enlarged thyroid and tachycardia, and by other accessory symptoms, as tremor, increased metabolism, and ocular, vascular, digestive and other symptoms. It was first described in England by Parry (1786), and Graves (1835), and later in Germany by Basedow (1840). The claims of the Italians, that Flajani (1800) first described the affection, are ill-founded.

Etiology.—Its etiology is obscure, but (1) the most probable cause is *over-activity or perversion of thyroid secretion* (Moebius, Johnston and Greenfield). Exophthalmic goitre contrasts sharply with myxœdema (*v.i.*) of which the anatomical basis is thyroid atrophy. The outlines of this theory can be best presented by comparing these antithetic conditions;

EXOPHTHALMIC GOITRE.	*vs.*	MYXŒDEMA.
(*a*) Hypertrophy of gland; excessive internal secretion (hyperthyrea or thyroidism).		Atrophy; deficient (hypothyrea) or absent secretion (athyrea).
(*b*) Increased nervous excitability.		Dulness, apathy, cretinism.
(*c*) Vascular erythrism; flushed, moist, warm skin; rapid pulse.		Skin dry, thick and cool, and pulse slow.
(*d*) Increased diuresis and active metabolism.		Decreased.
(*e*) Thyroid extract increases symptoms; it may produce in normal individuals symptoms like those of exophthalmic goitre, tachycardia, tremor, even exophthalmos.		Thyroid extract improves or cures

Surgical (partial) excision relieves, and often cures.	Some cases result from removal of thyroid. Transplantation of new gland improves or cures.
Exophthalmic goitre (glandular hypertrophy with excessive function) may occasionally become myxœdema (insufficient function from glandular atrophy).	

. *Other etiological explanations* are less probable, as (*a*) vasomotor rosis (Charcot); (*b*) lesions in the corpus restiforme of the medulla, ed on experiments by Filehne; (*c*) lymphatic disease and (*d*) dis- e of the parathyroid glands.

. *Disposing or exciting factors* are: (*a*) *Sex;* in Eschner's series 200 cases 80 per cent. were women (95 per cent. in other series). roids, pregnancy, and puberty are sometimes promoting factors. *Age;* most cases occur between the twentieth and thirtieth years. rrett (1904) could collect only 42 cases in persons under fifteen rs. (*c*) *Psychical* trauma, as fear, excitement or concussion, are netimes apparent causes. (*d*) Anæmia, debility, excesses, neuro- hic family tendencies and occasionally chorea, hysteria, epilepsy psychoses favor its development. (*e*) After thyroidectomy, symp- ms resembling those of exophthalmic goitre may develop; this stoperative thyroidism is rare.

Symptoms.—Cardinal Symptoms.—1. *Tachycardia* is usually the rliest cardinal symptom, and is almost always present. The pulse nges from 100 to 120, and in severe cases to 140 (160 or even 200). is associated with other cardiovascular symptoms, as (*a*) increased rdiac action, manifested by a diffuse apex and cardiac impulse, arp tones sometimes heard at a distance of several feet (Graves), d hypertrophy chiefly of the left ventricle which is ultimately com- cated by dilatation; (*b*) a functional (systolic) murmur over the ex; (*c*) palpitation, which usually antedates tachycardia and is an dence of intoxication (thyroidism); (*d*) vascular symptoms; a pulse her weaker than the apex beat; active arterial pulsation in the otids, and abdominal aorta; systolic capillary pulsation in the gers, forehead or even in the retina, palate, liver and spleen; even peripheral veins may pulsate; (*e*) irregular and failing heart action urs late and only in severe cases. Hemorrhages, notably epistaxis, not uncommon.

2. *Exophthalmos* is usually a later symptom than tachycardia or netimes than the goitre. It is present in about 90 per cent. of cases d results from dilatation of the orbital vessels. In developed cases e protrusion of the eyeballs, and the staring, fixed expression are very aracteristic (exophthalmos may also occur in extreme myopia, tumors the orbit, and sympathetic irritation). The condition is generally lateral, though often unequal; in one instance, the author saw uni- eral exophthalmos with homolateral goitre. The eyelids are unable cover the eyeball entirely, and a rim of the white sclera shows out the iris. The protrusion may be very extreme, even amounting

to dislocation of the bulb; in these cases it may be necessary
together the lids to prevent corneal ulceration and loss of the
panophthalmitis. Under this topic may be mentioned the *as*
eye-symptoms; they are (*a*) von *Graefe's sign,* which is the fai
the upper lid to follow the eye-ball when it is rolled downwar
present in at least 33 per cent., and is of considerable diagnosti
though present also in Thomsen's disease. (*b*) *Stellwag's sign,*
is rarely absent, is widening of the interpalpebral fissure wit
quent winking; it also occurs in mania, and in old age. (*c*) *M*
sign consists of weakness of the internal recti, whereby conv
is less complete, or unsustained, when a finger on which the e
focused is brought close to the nose; it occurs also in other con
as paretic dementia. (*d*) *Becker's sign* consists of pulsation
retinal vessels. (*e*) In *Joffroy's sign,* the patient's forehead r
smooth (instead of becoming wrinkled) when he glances
with the head bent somewhat downward. Eye paralyses and
or pupillary changes are infrequent.

3. *The goitre* is rarely an initial symptom, and usually d
later and in a remittent fashion, *i.e.*, swelling, recession and r
swelling. It is present in 92 per cent. (*a*) Its typical form is *v*
though it may develop upon any of the ordinary forms of go
in an acute fatal case, seen in 1905, with Dr. Dorsey of Stre
which exophthalmos, tachycardia, and thyroid thrills and m
developed in a chronic cystic goitre. Acute thyroidism may dev
thyroid malignancy. The goitre is *smaller* than the ordinar
chocele; it is diffuse or oftener more developed on the right s
accentuation of physiological proportions); and *microscopicall*
the dilated vessels alluded to above, and hyperplasia of the pare
atous tissue, in which the colloid matter is scanty or resem
embryonal form (not staining with eosin); in the later stage
fibrous tissue may develop. (*b*) It is usually *soft, elastic, var*
size from time to time, *pulsating* on inspection, *thrilling* on pal
and revealing on auscultation a loud systolic bruit, or a doub
mur which, according to Guttmann, is pathognomonic.

Accessory Symptoms.—(*a*) *Tremor,* described first by Base
classified as a cardinal symptom by some writers, as Char
Marie. It is present to some degree in most cases, and is cons
in 50 per cent. It is characteristically fine, regular and rapid, n
ing 8 or 9 vibrations to the second. It affects the wrist mo
the fingers; sometimes it affects the trunk. (*b*) There may be
symptoms. Some *cerebral symptoms* are usual, as headache,
impaired memory, insomnia, irritability, depression or change
position. Psychoses are infrequent, as the mania which is som
observed in fatal cases. Choreiform movements were descri
Kahler. Muscular weakness is usual, as "giving way of the
(Charcot), and weakness of the respiratory muscles (scant insp
increase in the chest measurement, Bryson's sign). The refle
often increased. Rheumatic pains may be noted. *Vasomotor*

toms are common, as violent reddening of the face, subjective warmth, dry mouth, sweats of a peculiar, penetrating odor, or intermittent swelling of the joints. Rarer complications are cerebral paralyses, tabes, syringomyelia or other neuroses. (*c*) *Respiratory* symptoms include dyspnœa and dry cough. The author has seen two incipient cases diagnosticated as phthisis. (*d*) There may be *digestive* symptoms. Severe (nervous) vomiting and diarrhœa may simulate organic disease; their persistence is ominous. (*e*) *Cutaneous* symptoms may be marked. Pigmentation may be extreme (Drummond, Chvostek). Pigmentation of the eyelids, vasomotor œdema, urticaria (particularly in acute cases), vertigo, itching, and falling of the hair may also occur. A peculiar fulness above the clavicles may develop, sometimes with pain in the neck. The electrical resistance of the skin is decreased, perhaps to one-fifth of the normal (Charcot, Chvostek, Vigouroux); it is probably occasioned by profuse sweating and has no pathognomonic importance. (*f*) There may be *constitutional* symptoms. Emaciation and anæmia are common, especially in blondes. Metabolism is almost always increased; this is evidenced by the nitrogenous excretion, increased diuresis and the frequent, slight, but wholly atypical, elevation of temperature; these symptoms may be intermittent (Huchard). Amenorrhœa, albuminuria, glycosuria, persistence of the thymus gland, and the occasional hyperplasia of the spleen are difficult to explain.

Diagnosis.—The diagnosis is unequivocal when the cardinal triad of symptoms is present, reinforced by the secondary symptoms. Atypical forms (*formes frustes*) may cause confusion, but careful watching of the clinical course usually settles all doubt. The affection is often incorrectly diagnosticated in young girls at puberty, in whom thyroid turgescence, moderate tachycardia and vascular excitability occur without particular significance. Incipient tuberculosis and neurasthenia may be simulated.

Course and Prognosis.—(*a*) The onset is usually insidious and the course chronic. Remissions are very common. Chronic cases sometimes begin rather acutely. Acute cases are unusual; they occur chiefly in men; Lloyd records a case which was fatal in three days, and Trousseau one which was fatal in eight days. The author has seen three cases in which death occurred in from two to five weeks. The acute type is usually fatal, though Solbrig mentions a recovery after ten days. (*b*) *Death* occurs in 12 per cent. of cases; it is almost always due to cardiac failure, from hypertrophy passing into dilatation. (*c*) The outlook is best in the less typical (rudimentary) forms. (*d*) Complete recovery is rare, though 20 to 50 per cent. of cases improve (relative recovery). (*e*) Unfavorable prognostics are its occurrence in the very young and in men, emaciation, extreme tachycardia, persistent vomiting or diarrhœa, fever, corneal disease, lymphatism and intercurrence of myxœdema (*v.i.*).

Treatment.—**1. General.**—*Rest* is the first indication. Travel, sojourn at the various springs and resorts, gymnastics and excitement are always injurious. A simple diet and interdiction of all stim (alcohol, coffee, tea, tobacco) are essential. A quiet life at a moderate elevation or at the sea-side is very beneficial.

2. **Drugs.**—Except for symptomatic indications, drugs have a limited use. The heart tonics are disappointing; digitalis u fails to slow the heart, and strophanthus and spartein sulpha rather more efficient but do not adequately control tachycardia. donna, in the largest physiological doses (until it produces my and dry mouth), is recommended by Gowers; some cases ar tinctly benefited, and in personal experience, the annoying palpi and abdominal throbbing are most relieved. Ergotin, qu arsenic, iron and strychnine are of uncertain value, but the seems rather more beneficial; iron is apparently useful only in chl girls (Moebius) and is directly injurious in men, for it aggr digestive and vasomotor disturbance. Constant application of a bag to the heart is very helpful; it relieves palpitation and som slows the heart rate.

3. **Electricity.**—This is chiefly valuable as a suggestive re according to some writers; galvanization is particularly recomm by Erb, who uses the anode over the cervical spine and the ka over various peripheral areas. Electrotherapy effects only tra results.

4. **Organotherapy.**—Thyroid extract aggravates the sympto all save a very few cases. Thymus extract has apparently helped cases, but most recent reports concur with the rather negative r obtained by Mackenzie and Kinnicutt. The use of serum or from dethyroidated animals is still in the experimental stage; Bau (1895) demonstrated that the thyroid gland contained an iodine which Ross proved was the main functional constituent of the c The active element of the gland presents two clinical phases: neutralizes toxins (failing in which, myxœdema results), and (*b*) *itself neutralized*, Basedow's disease results; to neutralize the h thyrea of this disease, it was suggested that the serum of thyr tomized animals be used (Ballet, Enriquez, Lanz, Burghart Blumenthal); some good results have recently been obtained (Ro

5. **Thyroidectomy.**—The earlier objections to partial thyroide on the grounds that many operated cases were ordinary goitr that improvement often occurs spontaneously or medicinally, ar weakening because of convincing surgical statistics. The av mortality is about 15 per cent. (Mayo, Oppenheim). In Ko large experience of 167 operations (1906), naturally on severe only 5 per cent. died. The Mayos' last series shows 8 per cent. tality; they consider general preferable to local anæsthesia. may also occur under local anæsthesia which latter is a very unple procedure. Removal under nitrous monoxide gas gave excellent r in two of the author's cases. The results of operations by K Kümmell, Friedheim and others show approximately the foll results; mortality 5 per cent. (as against 12 per cent. under m

treatment), complete recovery (which is rare under expectant therapy) in 70 per cent. and partial recovery in 25 per cent. Kocher prefers trying the x-rays first and then proceeding by stages, first ligating the thyroid arteries (after which tetany may occur and once within half an hour), and later performing partial thyroidectomy; the danger of a "heart" death is avoided by relatively early operation. Hirst states that the mortality of operation, for any indication, in an exophthalmic subject, is increased 15 per cent.

Division of the cervical sympathetic is inadvisable.

MYXŒDEMA.

Definition.—Myxœdema is a chronic constitutional disease, characterized (i) by loss of thyroid function (athyrea), (ii) by myœdema (an elastic œdematous change in the subcutaneous tissue), (iii) by cachexia or (iv) by cretinism (mental failure). It was described by Gull (1873) as a cretinoid manifestation, and later by Ord (1877), in England, and in France by Charcot, Bourneville, d'Olier and others. The symptoms vary as to cause and combination, whence (*a*) cretinism, (*b*) adult myxœdema, and (*c*) operative myxœdema will be considered separately.

Etiology.—The thyroid may be aplastic, hypoplastic, cirrhotic from an infective sclerosing thyroiditis (syphilis, tuberculosis, rheumatism, or erysipelas), or hypertrophied (goitre) or it may be removed by operation, but in all cases the thyroid function (internal secretion or antitoxic action), is suspended or lost.

CRETINISM.

Cretinism may be congenital, in which instance life is not long protracted; it usually develops in the first year, and is noticed near the time of weaning (infantile form); it may develop between the fourth year and puberty (juvenile form). The endemic form occurs in regions where goitre is prevalent, in Switzerland, Italy, France, Great Britain, Spain and Sweden; according to Kocher, when both parents are goitrous, the child is invariably a cretin, is myxœdematous and may also have goitre. The sporadic form may develop from thyroid aplasia, sclerosing thyroiditis or from goitre. In 1901, 100 cases were collected in America.

Symptoms.—In the last part of the first, or in the second year of life, retardation of mental and physical development is apparent. The head is disproportionately large, the fontanelles persist, the forehead is narrow, and the base of the skull is shortened. The face is turgid, pale, and imbecile, the lids are swollen, as are the nose, the protruding tongue, and pouting lips. Dentition is retarded and caries is frequent. The skin loses its tone and is dry, and the hair is thin.

The neck is short, which condition is accentuated by supraclavicul
deposits of fat. The abdomen is large and pendulous; umbili
hernia is very frequent. The body is dwarfed and the extremiti
are short, stumpy, and weak or helpless. Metabolism is slow, t
gaseous interchange being reduced to 50 or 60 per cent. of the norm

Diagnosis.—(*a*) The mental alteration is marked, though mild co
pared with the grimaces, grinding of the teeth, and compulsory mo
ments caused by *gross lesions of the brain.* (*b*) In hyperplasia of t
bone cartilages (*chondrodystrophia fœtalis*), there is great enlargeme
of the head and joints, dwarfing of the body, and shortness of t
extremities (micromelia), but no essential mental change; court fo
were probably of this type. (*c*) *Infantilism* concerns only the infant
appearance of the genitalia, and the absence of the primary and secon
ary evidences of sex; general malnutrition, aplasia, and cretinism a
absent, though some cases of infantilism are due to thyroid hyp
plasia.

MYXŒDEMA OF ADULTS.

Symptoms.—Myxœdema spontaneum adultorum, or *cachexie pach
dermique* (Charcot) is characterized by (1) thyroid atrophy, (2) my
œdema (pachydermia), (3) intellectual and physical weakness, an
(4) certain accessory symptoms. It is most frequent in England an
France. Most cases occur in persons between thirty and fifty yea
of age.

1. Thyroid Atrophy.—This is difficult to detect clinically. It
less a clinical than a histological condition, as evidenced by the lac
of function sometimes observed in gross enlargement of the glan
Magnus Levy, in 34 cases observed by him, found the thyroid ha
and large in 5 cases. Eighty-six per cent. occurs in women, and
is possibly related to the uterine functions or to the physiologi
congestive, thyroid enlargement observed in women. It may
hereditary or familial. It sometimes occurs in a family (or in indiv
uals) in which there are cases of exophthalmic goitre.

2. Myxœdema.—In a few instances, much mucin has been fo
in the skin and subcutaneous tissues. These structures are inela
semigelatinous and semiœdematous, but do not pit upon pres
the skin is pale, waxy, dry, rough, firm, and its electrical resist
is increased; the hair and teeth may fall out and the nails thi
and break. The facies is somnolent, coarse, and swollen, and
features are individually changed, rather resembling those of a cr
the cheeks are patchy-red, pendulous and tremble like jelly, and
tongue is big. The supraclavicular tissue is enlarged in lumps
rolls, though this is also observed in healthy persons (Verne
pseudolipomata). S. Kuh observed a painful swelling over the cla
alone, which rather resembled keloid. The hands and feet are i
trated. The swellings may change rapidly. The oral mucous m

brane is dry and thick, sometimes also the pharynx, larynx and even the rectum; polypi are frequent and secretion of tears is scanty.

3. **Mental Weakness.**—Intellectual and physical weakness is apparent. Somnolence is more frequent than insomnia. Charcot compares the condition to hibernation. Headache is common. The patient is apathetic, irritable, weak in memory, and slow in thought, speech and movement; sometimes delirium and mania precede the ultimate dementia. The gait is clumsy ("hippopotamus gait") and muscular fatigue follows moderate exertion.

4. **Other Symptoms.**—The accessory symptoms are variable, as cardiac hypertrophy, arteriosclerosis, palpitation; small, sluggish pupils, tremor, vertigo, night terrors, sensitiveness to cold; indigestion and constipation; anæmia; amenorrhœa; scanty urine with little urea, and low specific gravity, sometimes albuminuria, and less often glycosuria; hemorrhages, especially from the uterus; and subnormal temperature. The thymus is usually enlarged, sometimes the hypophysis; Hunt describes it with adrenal tuberculosis.

Course.—The course is chronically progressive, and covers ten to twenty years. Remissions may occur in the warm weather, from change to a milder climate or during pregnancy. In the last stage, the myxœdema often disappears. Transient cases of acute myxœdema have been observed by Sollier, Osler, and others. Osler in 1898, reported an acute case with melæna, mania, glycosuria, tachycardia, and death in six months. Sudden death is occasional but most patients die of intercurrent tuberculosis or acute infections.

CACHEXIA STRUMIPRIVA (THYREOPRIVA) OR OPERATIVE MYXŒDEMA.

Horsley reported that myxœdema, tetany contractures, apathy, and coma occurred in the monkey after complete thyroidectomy; when kept warm, the monkeys did not manifest myxœdema, but a species of cretinism. Cachexia follows extirpation in man and herbivora; convulsions develop in carnivora, resulting from the fact that the antitoxic elements of the thyroid are not present to neutralize the nuclein of ingested meat. The toxæmia strumipriva is manifested by anæmia (2 million red cells), leukocytosis (30, to 38,000) and degeneration in the walls of the bloodvessels. Kocher (1882) observed cachexia strumipriva in about 17 per cent. of 408 cases, more often after the complete than after the partial operation. Reverdin made similar observations, and Eiselberg reported it in about 23 per cent. of complete excisions made in Billroth's clinic. Accessory thyroids (*v.* GOITRE) may avert this operative complication. The author knows of two instances in which removal of an accessory lingual thyroid (under mistaken diagnosis), caused myxœdema. Schiff (1884), and later Eiselsberg succeeded in curing operative myxœdema by transplantation of a thyroid. Bircher (1889) first successfully made a trans-

plantation in the human being. Unfortunately, the function of t
transplanted gland is very seldom maintained, though recently C
tiani reports excellent results.

In some cases, ablation of the thyroid gland produces tetany (*teta*
thyreopriva). Vassali and Generali maintained that absence or ab
tion of the thyroid causes cachexia, and trophic disorder, and t
absence or ablation of the parathyroid glands causes tetany (with
cachexia). The parathyroids have maintained life thirty-seven y
when the thyroid was congenitally absent, and Kishi believes
after thyroidectomy, the parathyroids actually become thyroid t
In thyroidectomy the upper parts of the lateral and middle
should be removed in order to spare the parathyroids which are
alongside the lower part of the lateral lobes.

Treatment of Cretinism, Myxœdema and Cachexia Thyreo
For these allied conditions we possess in the thyroid extra
the few actual specifics of medicine, and most cases of cretin
myxœdema and operative myxœdema can be cured—in a se
seldom employ this term. Horsley's pupil, Murray, fir
thyroid extract hypodermatically; this method soon gav
administration by mouth, advocated by Horwitz of Copen
and Mackenzie and E. L. Fox in London. The pow
should be prepared from the glands of young sheep,
soon atrophies in older animals. Beginning with gr. ij
may be increased to gr. v or even xv. In a short tin
of increased metabolism appear, as loss of weight, in
increased urea and elevation of bodily temperature;
stimulated, myxœdema and cretinism disappear,
full and lively, the skin moistens and regains its
regard the results are magical. In some instances t
ism), are apparent, as tachycardia, nervousness
palpitation, flushing, sweating, tremor, less oft
rarely exophthalmos, softening of the bones,
rhachitic subjects, or even death; these sympt
of exophthalmic goitre (hyperthyrea); thyroi
frequent in myocarditic and arteriosclerotic
that administration of arsenic lessens the liab
after all is an infrequent condition. Larg
winter than in summer. At first large dos
after relief is obtained, smaller doses shou
to ij, or whatever size experience in the ind
Relapses are common after withdrawal
cases it becomes rather a food than me
warm baths, and sometimes pilocarpin
matically, are of decided benefit. A
also advocated.

(E). DISEASES OF THE THYMUS GLAND.

The **thymic functions** are unknown. Among other hypotheses, it is said that the thymus is concerned in neutralizing infection, that it concerns certain vegetative functions as the growth of bone, and that it bears some relation to cerebral function. Its secretion in the first two years of life is milky and contains iodine, cells, and a molecular substance. It weighs two to four drams (7 to 13 gm.) at birth, about an ounce (24 to 30 gm.) in the second year, after which it wastes, weighing but one dram (3 gm.) at twenty-five to thirty years of age. There is an intimate, though obscure, relation between the thymus, thyroid, pituitary body, parathyroids, testes, ovaries and possibly also the bone-marrow. The thymus wastes as the testicles and ovaries develop. Thymic extract causes a fall in blood pressure, cardiac acceleration and, in large doses, fatal collapse.

(*a*) **Hemorrhages** into the thymus may be found in cases of congenital syphilis and in asphyxia of the new-born. (*b*) Thymic *cysts* have been described. Chiari claims that 50 per cent. of them are merely accentuation of the normal findings, in which the lymphoid elements overgrow into the convolutions of the gland. (*c*) *Abscess* of the thymus has been described by Dubois (1850), but is often mistaken for softened gummata, cysts and postmortem softening. (*d*) The most frequent *tumors* are sarcoma and lymphosarcoma. Less frequent are dermoids and granulomata.

(*e*) **Persistence of the gland after puberty** is an infrequent and wholly obscure condition; sometimes it may be identified by an area of dulness reaching upward from the third rib and an inch or so to either side of the sternum.

(*f*) Some cases of "persistent thymus" are **hypertrophy**, which is the most common thymic lesion. (i) Plater (1614) associated enlargement of the thymus with *sudden death*. Grawitz, Jacobi, and others have described cases of thymic hyperplasia which has caused sudden dyspnœa, usually in infants, followed by early death; the idea is advanced that the gland presses on the vagus, cava, or trachea. The cases are thought to be lymphatism, as are the cases described by Nordmann, Recklinghausen, Rolleston, Paltauf and Keyer, who collected eight cases of sudden death in robust young men while bathing, by Kundrat, who found thymic hyperplasia in 10 cases of death from chloroform, and by Schlömicher's similar series. (ii) The existence of *thymic asthma* (Kopp, 1830), also known as Millar's asthma, was denied by Friedleben (1848). Though asthma thymicum is probably a nervous affection (laryngismus stridulus) in most cases, still in some rare instances, the enlarged gland may actually flatten the trachea (Summa and Benecke), as the distance between the sternum and spine is but little more than an inch; Siegel, in a case of thymic asthma, stitched

Acromegaly (large extremities), first described by
a nutritional affection, probably caused by disease o
and marked clinically by unusual overgrowth of t
of the face, hands and feet, and by general symptom
sexual disturbance.

Etiology.—The hypophysis is almost always dis
Woods-Hutchinson collected 262 cases; in 77 aut
was diseased in 73. The process may be hyperplasia
malignant sarcoma, less commonly colloid degenerat
ening or hemorrhage. Dean Lewis found hyperplas
phile cells, which confirms Benda's theory that acr
excessive activity of the cells in the anterior lobe o
From its association with acromegaly, the gland ha
be the "growth centre." The growth may compress t
cavernous sinus, sympathetic nerves, bone, dura o
According to Sternberg, the sexes are about equall
50 per cent., the affection begins between twenty
per cent. between twenty and forty, and in 14 per c
years of age.

Symptoms.—**1. Acromegaly.**—Enlargement of t
usually the first clear symptom, but is preceded by para
in the extremities, which are often extreme, and are
(*a*) A remarkable change occurs in the *face;* the s
protrude, the maxillæ enlarge and the nose, ears,
chin attain massive proportions. The general cont
hexagonal. The teeth separate, and the tongue son
beyond them. The eyes by contrast appear small.
the change in the bone is hypertrophy without inflan
increase in the vascular furrows, widening of some
(with narrowing of others), and some roughening
localized protuberances which are not osteophytes; t
and larger, especially the sphenoid, supraciliary rid
occipital protuberances; the orbit may be narro

the "spade-like fingers." The palms of the hands and soles of the feet become flattened. The *x*-rays show simple hypertrophy of the periosteal and subperiosteal tissues, and also of the soft parts; in some cases the soft parts are more hyperplastic than the bones. A common statement in the history is that the patient "must each year buy a larger-sized hat, gloves, or shoes." The nails are thin and small. The antithesis of acromegaly is micromelia (Hutchinson). (*c*) The *changes* in the trunk are less conspicuous. The sternum is increased anteroposteriorly, and the ribs and clavicles are thickened. The anteroposterior dimension of the thorax is increased, the head is held backward, and the dorsal kyphosis is rather characteristic. The muscular weakness, and lean arms and legs contrast sharply with the first impression of strength imparted by the massive face and extremities. The breathing is largely abdominal. (*d*) The *skin* is hyperplastic, and also the mucous membrane of the nose, tongue, pharynx and larynx. The larynx is enlarged (Marie), which may explain the deep, low, rough, monotonous voice.

2. Nervous Manifestations.—(*a*) Symptoms of *brain tumor* (hypophysis) are common, as headache, vertigo, mental alteration, vomiting, periodic loss of consciousness, bitemporal hemianopsia (early and frequent), optic atrophy (40 per cent.), ocular paralysis, anosmia, auditory disturbance, and exophthalmos. Hertel in 175 cases found eye symptoms in 52.5 per cent. (*b*) *Changes in character*, anxiety, apathy, and insomnia which deepens into terminal somnolence, are common; severe pains, paræsthesia, acroparæsthesia, and subjective warmth are frequent.

3. Constitutional Symptoms.—(*a*) *Glycosuria* is more frequent than Hansemann's figures indicate (12 per cent.). Its relation to acromegaly is uncertain, probably being a coördinate manifestation. Sometimes polyuria occurs without glycosuria. (*b*) *Profuse sweats* are common, even with glycosuria; the skin is sometimes pigmented; obesity is common. (*c*) *Sexual disturbance* is the rule; in women amenorrhœa is practically constant, and is often preceded by dysmenorrhœa; in men impotence is not unusual; the genitalia are much oftener infantile than hypertrophied. (*d*) The *heart* is often *dilated*, and *hypertrophied* from the frequent concomitant *arteriosclerosis*, deformed chest, and rarely from actual overgrowth (cardiomegaly); there may be cyanosis as the heart weakens. The vessels are wide and thick. Splanchnomegaly may be early observed (intestines and other viscera). In most cases the lymph nodes, particularly the cervical, are palpably enlarged, the tonsils, spleen, thymus, and sometimes the thyroid are hyperplastic. Gigantism (*v.i.*) occurs in over 20 per cent. of cases.

Course and Prognosis.—(*a*) Acromegalic subjects seek advice for the enlargement of the extremities or tonsils, sweats, pain, eye-symptoms, or polyuria; (*b*) remissions are common, the constitutional symptoms and, to a slight extent, the enlargement subside; on the other hand, a fitful advance may be noted. (*c*) The average course is *chronic*, covering ten to twenty years, though relatively benign occur, lasting thirty or even fifty years, and acute malignant cas known, which are always due to sarcoma; they may develop in weeks, and average two to four years in duration. (*d*) *De* *inevitable*, though it may occur from various causes; it may suddenly as in brain tumor or from gradual marasmus; di coma, intercurrent infection, cardiac insufficiency, intestinal ca or pyelonephritis may explain the fatal issue.

Diagnosis.—In this connection, Strümpell quotes Charcot's r that physicians in general see and diagnosticate only what the seen and learned to diagnosticate; the diagnosis is made at a by one who has seen a single case; undue regard to a single sym as the skin changes, amenorrhœa or glycosuria, may cause err the grouping of acromegalic findings is unmistakable.

Differentiation.—1. In the *cranium progenium*, the lower ja trudes and its teeth are directed forward; the lower jaw is hyperp the upper jaw is hypoplastic. Cranium progenium may oc acromegaly, cretinism, healthy individuals after variola, in degen and in idiocy.

2. *Gigantism* occurs in over 20 per cent. of acromegaly (partic when acromegaly develops early in life). Forty per cent. of are acromegalic (Sternberg). The interrelation of these conditi incompletely understood. Gigantism proper implies symmetry of growth; there are normal "giants" and giants with acrom leontiasis (*v.i.*), facial hemihypertrophy, and hereditary sy Bassoe's report of the autopsy of the giant Wilkins covers the subject (*Journal of Mental and Nervous Diseases*, 1903).

3. *Diffuse hyperostosis* (Starr, Putnam, Prince) occurs in the y involves all the skull bones, and seldom the extremities or exophthalmos, blindness, deafness, bilateral facial palsy, dyspl dyspnœa, and other basilar symptoms are noted, which culn fatally with stupor, idiocy, epileptiform convulsions, and paral the extremities. A sub-type (leontiasis ossea, Dana, 1893) occ the form of tumor-like protuberances on the skull and other Prince (1902) collected 20 cases from the literature. He mai that it is a form of osteitis deformans.

4. *Osteitis deformans* (Paget's disease, 1887) is a combined rar and hypertrophic osteitis. It begins slowly in subjects over years old, with pains and deformity of the thighs, legs, and the legs bow widely outward and somewhat forward, the curve vical and upper dorsal spine becomes kyphotic, the thorax na transversely and widens anteroposteriorly, the abdomen is dian shaped and crossed by a sulcus, and the head hangs forward. soft parts do not hypertrophy as in acromegaly, the face is involved, the head is larger above than below, and the arms are altered. About 70 cases are on record.

5. *Hypertrophic osteoarthropathy*, known since the time of Hi rates, was especially described by Bamberger and simultaneous

Marie, in 1889. The ends of the fingers and perhaps the toes become clubbed or "drumstick" in appearance. While the nails are thin and small in acromegaly, in hypertrophic osteoarthropathy, they are large, thick, wide and curved downward. The alteration typically involves the terminal phalanges, but also in severe cases the ends of the ulna, radius, tibia and fibula. The pathology is a toxæmic, chronic periostitis, with hypertrophy and osteophytic growth. The most common cause is chronic lung disease (chronic phthisis, bronchiectasis, emphysema, or fibrosis), whence Marie's name of *osteoarthropathie hypertrophiante pneumonique;* it may develop in congenital heart disease or aneurysm, or, much less frequently, in icterus, gastric dilatation, syphilis, cystitis, or various infections. Adult males are most often affected.

The treatment of *acromegaly* is merely symptomatic. Extract of the hypophysis has been given, and in a few cases with temporary success.

SECTION VIII.

CONSTITUTIONAL DISEASES.

(A). DIABETES MELLITUS.

Definition.—An independent disease of metabolism characterized: (*a*) by a permanent *lessened capacity* of the organs fixing, storing and consuming *grape sugar;* (*b*) by excess of sugar in the blood (*hyperglycæmia*), resulting in (*c*) grape sugar in the urine (*glycosuria*); (*d*) *causal changes* in *the pancreas.* (The term mellituria includes the presence of all kinds of sugar in the urine.)

Willis (1622–75) first differentiated between diabetes mellitus and diabetes insipidus, and Dobson (1774), of Liverpool, fully described the clinical symptoms.

The liver, the reservoir for carbohydrates, may contain even 14 per cent. of glycogen. It collects carbohydrates from the portal vein, stores them as glycogen, and again imparts them to the blood as grape sugar, for use in the tissues. The muscles also manufacture glycogen from the grape sugar of the blood, and are more tenacious of it than is the liver. Glycogen may also be produced from the albuminates. When there is a temporary carbohydrate shortage, the reserve store of glycogen is drawn upon. Fat is converted in the liver into sugar, when neither the carbohydrates or the albuminates suffice to maintain the normal proportion of sugar in the blood. The fate of carbohydrates when ingested in excess is: (*a*) glycogen storage (up to 10 ounces—300 grams); (*b*) conversion into fat; (*c*) alimentary glycosuria from hyperglycæmia, when the sugar in the arterial blood exceeds 0.2 per cent.; this is known as "alimentary glycosuria," which is physiological, wholly distinct from diabetes mellitus, and occurs when the blood-sugar exceeds the assimilation limit. The test for alimentary glycosuria consists of giving 3 ounces (100 gms.) of grape sugar, after a light breakfast of bread and coffee. If more than 1 per cent. of sugar appears in the urine, or if 2 or 3 per cent. of the entire amount administered is found, it is indicative of diabetes rather than of glycosuria. Most sugar absorbed goes to the liver, but some absorbed by the lymphatics appears in the urine, whence its presence is not necessarily a sign of diabetes; in liver cirrhosis, sugar may reach the blood with the collateral circulation. Traces of sugar (0.01 to 0.02 per cent.) are normally present in the urine, but are not detected by the usual tests, whence healthy urine is regarded as free of sugar.

Etiology.—1. The *pancreas* (*q.v.*) has long been considered an etiological factor in diabetes. Recently Opie discovered *atrophic changes* in the cells of the islands of Langerhans; they are determined only by microscopic examination. These cells are thought to produce an *internal secretion,* concerned in breaking up sugar in the tissues and muscles. This internal secretion from Langerhans' islands, is wholly distinct from the external secretion of the pancreatic juice voided into the intestine. Lépine some years ago spoke of a "glycolytic ferment," which entered the blood from the pancreas and broke up the sugar molecules; in diabetes resulting from the absence of the ferment, the sugar is not broken down. This ingenious argument has received much experimental corroboration, since (*a*) total pancreatic extirpation in animals and man produces glycosuria; (*b*) after partial extirpation and suture of the remaining portion to the abdominal wall, diabetes fails to appear, but develops if this portion is later removed; (*c*) when about a tenth part is left, a mild form of diabetes ensues, and glycosuria occurs only after the ingestion of carbohydrates; (*d*) subsequent destruction of the remaining portion of the gland produces a severe form; (*e*) when more than one-tenth of the functionating gland is left behind no diabetes ordinarily results; (*f*) diabetes does not occur in simple ligature of the pancreatic duct, excluding the juice from the intestine, nor when the fluid escapes through a cutaneous fistula, whence the argument that there is an internal secretion. (*g*) Cohnheim's experiments (1903) suggest that a ferment or pro-enzyme, secreted by the pancreas, joins another ferment secreted by the muscles, and that the united ferments break down the sugar molecules to produce heat and energy. In 167 patients, Thoinot and Delamare found "Langerhans' insufficiency" in 80 per cent., and chiefly in "lean diabetics."

2. There is a *neurogenous diabetes,* the relations of which (if there are any) to the pancreas are wholly obscure. This and the following types might be considered as symptomatic diabetes. Nervous diseases, as Claude Bernard's well known puncture of the fourth ventricle, may possibly alter the circulation in the liver. Among them are apoplexy, tabes, brain tumors (particularly those located near the base), general paralysis, multiple sclerosis, meningitis, syphilis, softening of the brain, diseases of the cord, vagus and sympathetic system, trauma, traumatic

neurosis, paralysis agitans, mental diseases, Basedow's disease, and acromegaly. Hoffman distinguishes between (*a*) *neurogenous diabetes*, which occurs chiefly in men, with no tendency to furuncles, carbuncles or cataract, with no connection with gout, and which passes away if the brain affection is curable, and then always in a comparatively short time, though in some cases it causes death within two years; and (*b*) *diabetes of the obese*, which occurs as frequently in women as in men, often produces carbuncles, is often connected with gout, and is very chronic, often lasting ten or twenty years.

3. *Obesity* may be a cause; according to von Noorden, "There are (*a*) cases in which the consumption of sugar and its conversion into fat are simultaneously restricted—glycosuria which varies in severity, and in degree of emaciation (ordinary diabetes); (*b*) cases in which only the consumption of sugar, and not its conversion into fat is interfered with—obesity without glycosuria (masked diabetes); these pass readily at a later period into (*c*) cases in which the consumption of sugar is restricted, and the collecting of carbohydrates in the fatty tissues is also more or less interfered with—obesity with consecutive glycosuria (ordinary diabetes of the adipose)."

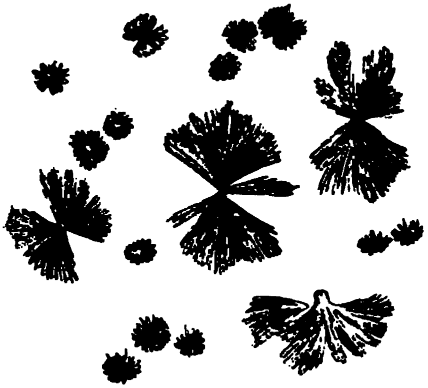
4. *Gout, arteriosclerosis*, various *liver lesions* (cirrhosis), and possibly acute infections, have apparent though obscure relations with diabetes. Adrenalin may produce glycosuria (Blum and Herter). Renal diabetes (glycosuria without an excessive amount of sugar in the blood) may occasionally develop in nephritis, renal hemorrhage, chyluria, and from administration of the glucoside, phloridzin. *Lactosuria* may occur in late pregnancy, the puerperium and nurslings; von Noorden's hypothesis is that in the "puerperal state the capacity of the tissues for breaking up milk-sugar is diminished, an instance of adaptation to an end in that the cells of the mother refuse a material which is preeminently suited to the nutritive wants of the nursling."

5. *Predisposing factors* are (*a*) *sex;* men (3:2) are especially subject to diabetes; (*b*) *age;* though it occurs at any age, its frequency is as follows: In persons from thirty to forty years, 18 per cent.; forty to fifty years, 25 per cent.; fifty to sixty years, 26 to 30 per cent.; in Stern's collection of 117 infantile cases, one was apparently congenital. (*c*) *Heredity.* A family history of obesity, consanguinity, neuropathic taint (20 per cent.), or gout is especially significant; cases are reported in which the grandfather was diabetic, the son gouty, and the grandson again diabetic (hereditary alternating diabetes). (*d*) *Race.* Diabetes is comparatively rare in the colored race, and is especially common in Jews. It is rather less common in America than in Europe. (*e*) *Occupation.* Diabetes is more often seen in private than in hospital practice. Wealth and culture increase ten-fold the predisposition to diabetes (von Noorden). It is frequently found in scientists, lawyers, musicians, poets, teachers, statesmen, merchants, speculators, and in those leading a luxurious or self-indulgent existence with sedentary habits; it also results from overwork, excitement, over-eating and over-drinking. Diabetes is apparently increasing in frequency.

Symptoms.—1. **The Urine.**—*Glycosuria.*—Sugar in the urine is the leading symptom of diabetes. A daily excretion of 1 to 3 drams (5 to 12 grams), is not uncommon; the maximum amount recorded is 1,500 gm.; the largest quantity of sugar is voided late in the morning or after 6 P.M. A percentage of from 1 to 4 is not uncommon, 8 or 9 per cent. is rare, and the maximum is 20 per cent. (personal observation). The estimation of a diabetic's capacity to consume sugar suggested simultaneously by Seegen and Traube is as follows: Accordingly as the urine, after a diet absolutely free from carbohydrates, is free from sugar or not, a distinction is made between (*a*) mild and (*b*) severe cases of glycosuria. (*a*) *Mild glycosuria* is characterized by the urine becoming sugar-free within a few days after carbohydrates are withdrawn, and by sugar reappearing when carbohydrates are again ingested. The test diet consists of ham, eggs, roast beef, cucumber with vinegar, olive-oil, and salt and pepper; brandy with Appolinaris water; coffee without milk or sugar; clear bouillon; butter, green salad, green vegetables, and sardines; this diet is free from carbohydrates. With increasing additions of starch, sugar at some time is again voided, *i.e.*, the limit of tolerance or of assimilation is reached. (*b*) *Severe glycosuria* is characterized by continued excretion of sugar, in spite of the withholding for days of all carbohydrates from the diet, and signifies (i) that even those carbohydrates which are slowly formed from the albumin are not wholly consumed; (ii) that the functions of glycogen storage, and (iii) of splitting up of sugar in the tissues are seriously affected; of this type there are many degrees. Fat and alcohol never increase the severity of glycosuria. Muscular exercise and massage usually decrease it. It is often increased by psychic disturbances and may be suspended or lessened during an acute intercurrent infection, especially in typhoid; chronic diseases with fever, especially pulmonary tuberculosis, have less influence. It is reduced or absent in the final stages of diabetes, and may be absent in diabetic coma. Glycosuria very often disappears when granular atrophy of the kidney develops (Frerichs, Stokvis, Fürbringer); the diabetes is then cured in some unknown way. Gouty diabetics as a rule pass no sugar during an attack of gout (diabetes alternans).

TESTS FOR SUGAR.—(*a*) For *Fehling's test*, two solutions are kept separate; one contains copper sulphate, 34.64 gm. in 500 c.c. of water and the other, sodium and potassium tartrate 173 gm., and sodium hydrate 125 gm. in 500 c.c. of water. Equal parts of these solutions are heated, but not boiled, and the urine is added drop by drop. The volume of urine should never exceed that of the solutions. Reduction of the blue copper solution to the yellow-red suboxide of copper occurs when sugar (its aldehyd group) is present. Chloral, phenacetin, chloroform, formalin, morphine, uroleukinic, and homogentisinic acids (the probable causes of alkaptonuria), glycuronic acid, cascara, rhubarb, and salicylates, may also reduce Fehling's solution, particularly after boiling. In doubtful cases, or as corroborative tests, (*b*) the *fermentation test* may be used; yeast is shaken thoroughly

PLATE XV.



Crystals of Phenyl-glucosazone. (Musser.)

(Oc. 4, Obj. D.) Drawn by J. D. Z. Chase.

ith a quantity of urine, the air allowed to bubble up, and the mixture laced in a saccharometer,—a tube closed at the top, and graduated or estimation of percentages. The sugar, fermenting, is measured y the amount of carbon dioxide formed. A control test should be nade. (c) The *polarization test*, showing dextro-rotary reaction, may e employed. (d) The very delicate and reliable *phenylhydrazin test* f E. Fischer may be used. Cipollina's method is the simplest: 5 ninims of pure phenylhydrazin, eight drops (0.5 c.c.) of glacial acetic cid, and a dram (4 c.c.) of urine in a test-tube are gently boiled for ne minute with frequent agitation. Four or five drops of sodium ydrate solution are added, the mixture is boiled again, and allowed o cool; in a few minutes (to a half an hour) yellow rosettes of henylglucosazon precipitate; maltose and certain pentoses, the crystals f which melt at different degrees, may alone cause error. Plate XV.

QUANTITY OF URINE AND SPECIFIC GRAVITY.—The urine is almost lways acid even after fermentation (lactic acid), and has a sweet aste. As a general rule the quantity of urine secreted during the ight is less than that secreted in the day-time; this is an important oint because in other *polyurias* frequently ⅔ to ¾ of the entire amount f urine is secreted during the night (Lecorché and Quincke). A ecretion of ten quarts is not rare. The largest amounts recorded are 6, 17 or 28 quarts in a day. Higher figures suggest simulation, atients having been known to add sugar to the urine. *Pollakiuria* frequency of urination) is common. The urine is *pale*. If dark and f uratic deposits are present, the prognosis is good (Seegen and Pavy). oam often remains long on its surface and the sediment is very carce or is absent. Prout considered that the onset of diabetes began t the time the urine ceased having a turbid, uratic deposit. An in-rease in the amount of sugar usually brings an increase in the amount f urine, but ordinarily the amount of urine rises less rapidly than oes that of the sugar. The specific gravity is higher, the greater he amount of urine secreted.

Amount in liters or quarts.	Specific gravity.	Sugar.
1.5 to 2.5	1.025 to 1.030	2 to 3%
2.5 to 4	1.030 to 1.036	3 to 5%
4 to 6	1.032 to 1.046	4 to 7%
6 to 10	1.036 to 1.046	6 to 9%

Two or three per cent. of sugar may cause no change in specific gravity. The average is 1,030–40, it is rarely more than 1,050, and the highest record is 1,074; very high specific gravity suggests fraud; the lowest records are 1,002 and 1,008. If under the influence of meat diet, the urine becomes entirely or nearly free from sugar, the quantity approaches normal. The specific gravity remains high because the urine is rich in meat end-products. There is no absolute parallelism between the percentage of sugar and the specific gravity; (a) glycosuria is often preceded by premonitory polyuria. (b) Though diabetes may be cured, polyuria may persist without glycosuria; this may be caused by the habit of drinking large quantities of water. (c) There are some cases in which though there is a large percentage of sugar, only a pint or a pint and a half (500 to 800 c.c.) of urine is secreted; this was first described by Peter Frank a century ago as "diabetes decipiens." Crystals of sugar may be seen on the shoes, clothing, or in the vessel.

Nitrogenous Constituents of the Urine.—Diabetic patients usually excrete considerable urea; 2 ounces (50 to 60 gm.) are not infrequent, and over 5 ounces (163 gm.) have been noted; larger figures are reached in no other disease; it is occasioned by the abundant ingestion of proteids, and possibly by fermentation of the tissue albumins; the condition is pathological when the amount of nitrogen in the urine exceeds that in the food. Naunyn and von Mehring deny that there is an increase in the urea save in severe and exceptional cases.

Ammonium, normally 8 to 15 grains (0.5 to 1 gm.) *per diem*, may increase to 20 or 180 grains, although the highest figures are seen exclusively in diabetic coma. The organism excretes increased amounts of ammonia in order to neutralize pathological acids (acidosis). The diabetic diet is acid; phosphoric acid forms from the ingested meat, and much sulphuric acid is produced by oxydation of sulphur in the albumin molecule. The quantity of creatinin may reach 20 to 30 grains (1.3 to 2 gm.) *per diem*, because of the diet, and tissue waste. The amount of uric acid is nearly normal.

Albuminuria is found in from 30 to 40 per cent. of cases; it is almost always slight, and is due (*a*) to cystitis, pyelitis, intercurrent diseases as gangrene or infection, pulmonary tuberculosis, and in some cases without relation to diabetes as in the arteriosclerotic and gouty types of diabetes, to liver cirrhosis, etc.; (*b*) to diabetes in nervous individuals, and (*c*) to circulatory disturbances; (*d*) in uncomplicated cases of diabetes it may be due to the action of the sugar, β-oxybutyric and other acids and toxins on the kidneys. Albuminuria is a frequent forerunner of diabetic coma, either as a coördinate manifestation or a determining factor (decreased renal permeability with retention in the blood of toxins). Diabetes may develop into chronic interstitial nephritis, and the sugar then disappears.

Non-nitrogenous organic substances. Lipuria has been observed. Acetone, aceto-acetic acid and beta-oxybutyric acid are most frequent; they may be derivatives of albumin or of fat. *Beta-oxybutyric acid* is absent in diabetics who are in good physical condition, and not infrequently also in those who are suffering from marked loss of albumin. The urine may be odorless, or may smell like chloroform, fruit or wine. Sometimes the daily amount excreted is only a few grains; at other times it reaches 2 to even 7 ounces (50 or 226 grams). Its abnormal excretion once begun, is uninterrupted, sometimes lasts years, and shows a constant tendency to increase. It is of the gravest prognostic significance because in most cases, at the end of days, weeks or months, fatal diabetic coma develops. An excretion of more than an ounce indicates imminent coma. Acidosis is most accurately estimated by the output of ammonium; according to Magnus Levy, 2 gm. of ammonium correspond to 6 gm. of β-oxybutyric acid, 5 gm. to 20 gm., and 8 gm. to 36 or 40 gm. β-oxybutyric acid is thought by some to be the parent substance of acetone or diacetic acid. *Acetone and diacetone* are present in the urine in diabetes, fevers, cancer, inanition or auto-intoxications, and indicate malnutrition. When the amount of acetone progressively rises from 15 grains upward, the outlook is unfavorable. It is not proven that the acetones cause coma, but they at least accompany acidosis. They impart a fruity or wine-like odor to the urine and breath. *Denniges' test* for acetone (quoted from Simon's *Clinical Diagnosis*) is as follows: 20 grams of concentrated sulphuric acid are poured into 100 c.c. of distilled water, when 5 grams of freshly prepared yellow mercuric oxide are added. The mixture is allowed to stand for twenty-four hours. This reagent is added to 3 c.c. of urine, drop by drop, until the precipitate thus formed no longer disappears on stirring. When this point is reached a few more drops are added. After two to three minutes the precipitate is filtered off. The clear filtrate is further treated with about 2 c.c. of the reagent, and 3 to 4 c.c. of a 30 per cent. solution of sulphuric acid, and placed in a vessel with boiling water. In the presence of an abundant amount of acetone a copious white precipitate forms immediately; while in the presence of traces only (less than 1:50,000), a slight cloud develops on standing for several minutes. The precipitate is almost entirely soluble in an excess of hydrochloric acid. If albumin is present, the urine becomes turbid at once when the reagent is added. In that case the test is continued as described, attention being directed to the coarser precipitate which occurs later. To such urines large amounts of the reagent must be added, the idea being to precipitate everything that can be precipitated with the reagent, before heating. Oppenheimer claims that it is a delicate test, and gives well-pronounced reaction with a dilution of 1:20,000.

Gerhardt's test for diacetone is as follows: a dram of urine is treated with a strong solution of ferric chloride added drop by drop. A precipitate of phosphates is filtered off, and more of the iron solution is added to the filtrate. If a bordeaux-red color now appears, another portion of the urine is boiled, and similarly treated. If in the second test no reaction is obtained, a third portion of the urine is treated with sulphuric acid and extracted with ether. A positive reaction, when the ethereal extract is tested with ferric chloride, the color disappearing upon standing for twenty-four to forty-eight hours, indicates diacetic acid, particularly if the urine is rich in acetone.

Mineral Constituents.—Sodium chloride, sulphuric and phosphoric acids, and the ethereal phosphates are abundant; they are caused by the increased amount of food ingested. The oxalates are increased in mild cases. Much lime is excreted, which suggests osseous waste.

Pneumaturia.—The butyric acid bacillus, the Bacillus coli, ye fungus and other bacteria may cause fermentation in the bladd producing carbon dioxide, hydrogen, and methane gas.

2. Direct Consequences of the Glycosuria and Polyuria.—(*a*) T exaggerated *thirst* (*polydipsia*) is always secondary to the polyu (*b*) *Nutrition* is affected. Much sugar is lost to the tissues; quarts of urine with 5 per cent. of sugar produces loss of 400 calor one-seventh of the total. Therefore the nutritive requirements a great, the appetite is excessive (*polyphagia*) and in severe cases emaci tion and fatigue are pronounced. The loss of sugar must be met b increase in albumin and fat. Increase in weight may follow reductio in the sugar. The temperature in uncomplicated cases is subnorma from inanition.

3. Complications.—Complications and other symptoms result fro various causes: (*a*) the sugar in the blood, (*b*) impaired nutrition a lowered physiological resistance; both conditions predispose to (infection, particularly by the bacillus tuberculosis, (*d*) poisoni (acidosis), and other ill-understood conditions, which promote dege erations and toxæmia.

1. Nervous.—(*a*) *Intellectual depression*, insomnia, headache a neurasthenic symptoms are common, though psychoses are less f quent. In rare instances focal symptoms may develop (as in uræmi without anatomical findings. Brain symptoms may result from co cident syphilis, arteriosclerosis, etc. (*b*) *Diabetic coma* occurs in per cent. and causes about half the deaths in diabetes; it is most f quent in youth, and in acute and severe types; Naunyn holds th it is favored by a strict antidiabetic diet. All cases of coma whi develop in diabetes are not diabetic; patients may succumb (i) cerebral *apoplexy*, tuberculosis, embolism, thrombosis, sepsis, *uræmia*, (ii) to *heart failure*, which may cause sudden death, or co with death after a short time or (iii) *diabetic coma* proper, which probably due to toxæmia (acidosis), and closely resembles expe mental acid poisoning. Most writers believe that β-oxybutyric ac is causal, though in three of the author's cases it was not prese in the urine (Long). Coma is imminent when the daily output ammonium exceeds 45 grains (3 gm.), for this measures the acido It may occur suddenly without obvious cause, or may follow sev exertion, excesses, excitement or gastro-intestinal disorders. In so cases there are prodromal headache vertigo, stupor, restless anxiety symptoms resembling those of acute alcoholism. The pupils a dilated, the reflexes abolished, the pulse is small, and averages 1 to 120, and the temperature is usually subnormal. Convulsions a most uncommon. The breath has the wine-like acetone odor. Th breathing is characteristic; inspiration is deep, long-drawn and energetic and expiration is short and slightly sighing; in this, *Kussmaul's dyspnœa*, the inspiratory energy contrasts strongly with the general bodily weakness; respiration may be normal in rate, but is generally somewhat accelerated; there is no stridor, nor, at the onset, any

yanosis. The urine is generally albuminous and almost always conains small granular casts (Külz and Adelhof); the addition of a few rops of ferric chloride gives the Burgundy wine color; sugar is resent, though it may occasionally disappear as the coma develops. artial consciousness may return, but, with few exceptions, death ccurs within a few hours to two days, with subnormal temperature, owed respiration and cardiac weakness. The autopsy reveals no nstant brain changes. (*c*) Degeneration (toxæmic) in the posterior lumns of the cord may develop. (*d*) The *peripheral nerves* may be fected. *Neuralgia* is common and may indicate incipient neuritis, may appear and disappear, varying with the amount of sugar in blood and urine. It is often an early symptom; it may be uniteral or more often bilateral, most frequently affects the sciatic rves, and is not uncommonly a most obstinate and recurrent symp- *Neuritis*, as a general rule, occurs in the severe forms of diabetes, d in its later stages. It may be manifested only by obstinate neugia, painful paræsthesia in the legs, muscular cramps or in 30 to per cent. of diabetics, decrease or abolition of the patellar reflexes. euritis may involve the cranial nerves, mostly the abducens, then e oculomotor, hypoglossus, spinal accessory and facial, or with reater frequency the spinal nerves; the lower are affected more ften than the upper extremities, and, like diabetic neuralgia, bilateral volvement of the crural or sciatic nerves is the most common type. euritis may be single or multiple (polyneuritis). Its symptoms are hose of neuritis or polyneuritis (*q.v.*), as paralysis, altered sensation, bolished reflexes, reaction of degeneration and trophic disturbances, s falling of the hair and nails, glossy skin, and perhaps also herpes r perforating ulcer of the foot (*mal perforant*). In severe cases tabes s simulated, *pseudotabes diabetica* (see MULTIPLE NEURITIS). (*e*) The *pecial senses* may be involved. Retinal diseases occur under three orms: (i) albuminuric retinitis, accompanying contracted kidney; ii) *retinitis centralis punctata* with characteristic small, shining central pots, usually with hemorrhagic points, and always bilateral; (iii) the rdinary hemorrhagic type (see Plate V, Fig. 1). Retinitis occurred in 9 per cent. of Galezowski's 114 cases. Neuritis, neuroretinitis and etrobulbar neuritis with consecutive atrophy are progressive, and occur nore frequently in severe than in mild cases, sometimes as early, ometimes as late, complications. Twenty per cent. of diabetics with etinitis die within one year, and 60 per cent. live over two years Nettleship).

Amblyopia and amaurosis are complications of early diabetes, and ften first induce the patient to seek medical advice. Paralysis of the cular muscles occurs in 7 per cent. of cases; accommodation paralysis s the most frequent type, and then that of the external rectus; the most probable cause is neuritis; Mauthner assumes that there is a nuclear hemorrhage. Cataract occurs in 4 per cent. of cases. Other ocular changes are rare. Aural complications include external furuncles and otitis media, which has a tendency to hemorrhage an diffusion, as mastoiditis.

2. CIRCULATORY.—(*a*) The *blood* may become somewhat conce trated, for much water is required to eliminate the sugar; the amou of sugar (normally 0.1 to 0.15 per cent.) is increased in diabetes t 0.3 or 0.5 per cent. Fat in the blood (lipæmia) is a frequent finding what appears to be fat is sometimes albumin (Futcher) or cholestearin in combination with fatty acids; Frazer in one case found 14 per cent. of fat. *Bremer's test* is based on changes in the hæmoglobin; thick smears of blood are made on a slide, which is heated and treated with 1 per cent. congo red solution for two minutes; diabetic blood will not stain, but the control slide of normal blood stains; this reaction, according to Bremer, often results during remissions in the glycosuria and in the preglycosuric stage; it sometimes occurs in other conditions, as Hodgkin's disease. *Williamson's test* is based on the fact that diabetic blood decolorizes a weak alkaline solution of methyl blue. It is as follows: 20 c.c. of blood, 40 c.c. of distilled water, and 1 c.c. of 1 to 6,000 aqueous solution of methylene blue are added to 40 c.c. of liquor potassæ, and are placed in test-tubes in a beaker of water, which is boiled for four minutes; the blue color disappears. The *alkalescence* of the blood, which is reduced in various conditions of inanition, is greatly lessened in diabetes, particularly when β-oxybutyric acid engages the ammonium and other salts and when coma develops. (*b*) The *arteries* are frequently sclerosed in diabetes. Arteriosclerosis causes many common symptoms, as cardiac hypertrophy or dilatation, angina pectoris, cardiac asthma, hemiplegia, albuminuria, senile gangrene, and intermittent claudication. (*c*) The *heart* may be altered in various ways (*v.s.*). Hypertrophy occurs in about 10 per cent. and is less ominous than atrophy, which occurs in uncomplicated diabetes of the young. Heart-failure may occur after moderate exercise, or may develop precipitately, ending in coma or asphyxia and correctly separated by Frerichs from genuine diabetic coma. Von Noorden observed five instances of sudden death in 140 fatal cases. "Functional heart symptoms" occur in 10 per cent. o cases. The pulse is usually accelerated, and its tension is elevated Cardiac or renal dropsy is uncommon in diabetes.

3. RESPIRATORY.—(*a*) *Tuberculosis* is most frequent in the poor classes, and in the young, but much less so in older, obese and gouty subjects; about 25 per cent. of diabetics contract tuberculosis. Hæmoptysis is very rare (Seegen and Leyden); the sputum is abundant; it is usually stated that the tubercle bacilli are scant or absent; in the secondary necrosis, to which all diabetic tissues are prone, the marked clinical and pathological lung-findings may sharply contrast with the small number of tubercle bacilli found in the sputum. The course is rapidly fatal. (*b*) *Gangrene* is much less common than tuberculosis. Fever, pulmonary hemorrhage, and purulent sputum are common. Fœtor is less conspicuous than in other forms of pul-

onary gangrene. (c) Intercurrent pneumonia may develop, and is
ually fatal.

4. Digestive.—(a) The *mouth* is generally dry and acid (from
composition of sugar); the saliva is scanty and poor in ferment;
omatitis is common, and the growth of aphthæ is promoted by the
id reaction of the mouth, though it is preventable by cleanliness.
he teeth may decay from alveolar periostitis or drop out from tropho-
eurotic changes. (b) The *stomach* is affected with remarkable infre-
uency, considering the oral findings, and the polyphagia. Marked
norexia is uncommon, though the patient may refuse fatty and
itrogenous food. The stomach is usually dilated. (c) The *bowels*
re generally costive. Schmitz claims that obstinate constipation pre-
isposes to coma, though in the author's experience, most diabetics
re constipated. Intestinal or gastro-intestinal catarrh sometimes seems
o precipitate coma, but it may be merely an associated symptom.
d) The *liver* is usually enlarged (hyperæmia and fatty degeneration),
nd sometimes tender. Gall-stones are concomitants in 10 per cent.
f cases, according to Bouchard, but Naunyn is correct in denying
hat they have any intrinsic relationship to diabetes. French authors
Hanot and Chauffard) have described a pigmentary cirrhosis in
iabetes (diabète bronzé), which has already been referred to under
iver cirrhosis, and chronic pancreatitis; this syndrome probably
develops in the following sequence: (i) hæmolysis, (ii) pigment deposit
in the liver, pancreas, and skin from the altered blood (hæmochroma-
osis), (iii) chronic interstitial inflammation, chronic hepatic cirrhosis,
nd (iv) chronic pancreatitis, causing diabetes. (e) *Pancreatic* symp-
oms are few, because the external pancreatic secretion is essentially
ormal, and only the internal glycolytic secretion is affected.

5. Genito-urinary.—(a) Albuminuria may be present (see Urine).
e kidneys are frequently hypertrophied and hyperæmic; various
generations may be noted, including Ehrlich's glycogenic degener-
n. Interstitial nephritis may follow and replace diabetes, chiefly
gouty and corpulent individuals. (b) Cystitis is not rare; the urine
actually ferment in the bladder, just as it readily decomposes
e urinal; this may lead to pyelonephritis or pneumaturia (*v.s.*).
tis may cause temporary disappearance of the glycosuria. (c)
e female, menstruation may be disturbed or suspended. The
l inclination is usually lessened in severe cases, but may be
sed in mild cases from genital pruritus (*v.i.*). Conception may
even in advanced cases, but in 33 per cent. there is spontaneous
n or premature delivery. In severe diabetes interruption of
ncy is indicated, but care must be taken that diabetes gravi-
is not confused with lactosuria; the maternal danger in unin-
d pregnancy is great, from exacerbation of the disease, coma,
and infection, though Stengel has recently concluded that the
s overdrawn. Fungi readily develop about the labia and
this is promoted by lessened general resistance and local
with the saccharine urine. Aphthæ, leptothrix and other

parasitic deposits are frequent, particularly in careless subjects; pruritus pudendorum, vulvitis, vaginitis, urethritis (which may simula gonorrhœa), furunculosis and even gangrene may develop. (*d*) I the male, the sexual appetite may be increased early in the diseas but later is replaced by impotence, which is probably due to spina degeneration. Phimosis, balanitis, small erosions near the urethr urethritis, itching at the meatus causing tenesmus, curious nodes i the corpora cavernosa, furuncles and necrosis may first engage th physician's attention. Genital symptoms in men are less conspicuou than in women.

6. CUTANEOUS.—The skin is dry, rather harsh and itches whe there is much polyuria and emaciation. Sugar is said to be prese when the patient sweats. Itching is caused by toxæmia irritating t cutaneous nerves, as in uræmic and cholæmic pruritus. Skin eruptio include pityriasis tabesentium, urticaria, erythema, eczema, bull acne, purpura and herpes. Infection is favored by reduced phys logical resistance and possibly by hyperglycæmia; furunculosis (10 25 per cent.) is due largely to the staphylococcus, and may initi lymphangitis, phlegmon or gangrene; erysipelas is promoted by duced general and local resistance. Gangrene is most frequent late or middle life, and in mild cases; it usually begins on the t and extends upward, either as dry or moist gangrene; it is said result from obliterating endarteritis, but in the writer's experienc occurs three times less frequently than the ordinary plaque-like athe oma. Unfortunately gangrene is usually progressive. Wounds i diabetics heal better than in pre-antiseptic times, but delayed healin infection, and gangrene are still frequent. Enlargement of the lymphatic glands is seldom absent in severe, progressive forms with emaciation; the glands are hard, painless, and probably secondary to ski infection or irritation.

Course and Prognosis.—The onset may be acute or chronic, an the course may be short (covering weeks) or protracted and remitten (covering twenty years or more). Various complications modify th duration and outcome. Von Noorden's summary of the gener *prognostic signs* covers the ground completely. *Favorable* signs ar (*a*) onset late in life; (*b*) long duration prior to the time of observation, with no grave complications nor any considerable emaciatio (*c*) traumatic causation; (*d*) syphilitic origin; (*e*) the occurren invariably of mild forms of diabetes in the family of the patient; (precedent and co-existent obesity; (*g*) co-existent uric acid diathesi (*h*) slight glycosuria and tolerance of moderate amounts of carbohydrates; (*i*) marked oscillations and increasing tolerance of carbohydrates; (*j*) circumstances which permit of dietetic and gener hygienic prescriptions. *Unfavorable* prognostics are: (*a*) onset i early life, especially in childhood; (*b*) great loss of strength, despit a brief duration of the diabetes; (*c*) a history of severe forms in the family; (*d*) early appearance of grave secondary diseases and complications; (*e*) a high degree of glycosuria, with complete intolerance of carbohydrates; (*f*) excretion of β-oxybutyric acid; coma; (*g*) poor circumstances which render impossible the dietetic treatment, or the avoidance of bodily and mental overwork.

Diagnosis.—The diagnosis is certain only when the urine in every case is examined as a routine procedure, because only then can the lighter cases, which are amenable to treatment, be discovered. In many cases the thirst, polyuria, emaciation, itching, and the complications *per se* are suggestive. *Pentosuria;* 16 familial or hereditary cases are recorded. Pentose reduces Fehling's solution, even after fermentation of the urine. Bial's test is made as follows: a small amount of the following solution, orcin 1 gm., liq. ferri chloridi 25 drops, and 30 per cent. hydrochloric acid in 500 c.c. of water, is heated to boiling, and added to the urine which turns green if pentose is present.

Treatment.—1. **Diet.**—Regulation of diet, dating from Rollo one century ago, is the most essential point. The carbohydrates normally supply nearly half the bodily requirements for food and energy. The diabetic loses a large part by their escape through the kidneys. Furthermore, their ingestion implies a waste of digestive energy, an irritation of the tissues by the unused sugar, and a growing decrease of the tolerance of the tissues for them. A man weighing 70 kilograms (150 lbs.) requires a diet representing 2,500 calories, *i. e.,* roughly 35 calories for each kilogram. The dietary must therefore regard not merely the glycosuria, but the maintenance of body-weight and energy. It is useless to suppress the symptom, glycosuria, when the patient emaciates. The object in view is to limit the carbohydrates, and replace them with albumin and fat. Naunyn and Weintraud have proven that exclusion of carbohydrates for some weeks increases the tolerance for carbohydrates taken afterward; *i.e.,* the "sugar-consuming function" is rested and in part restored.

General Outlines of Dietary.—Explicit written directions should be given to each patient. *All diabetics may eat the following foods: meat,* the muscular parts of beef, veal, mutton, and game, and the tongue, lung, heart, brain, marrow, and sweet-breads; also fatty livers, which contain but little glycogen; meat peptones, somatose and kindred preparations, gelatin, and meat jellies; *fish,* caviar, cod-liver oil, clams, oysters, lobsters, and crabs; *eggs* (10 to 20 per cent. of fat); animal and vegetable *fats,* bacon, suet, olive oil, cocoa butter; butter contains 85 per cent. fat, and should be used on meats, eggs, or spinach or melted in milk; rich cream; all *cheeses* (3 to 30 per cent. fat), especially Swiss varieties; *vegetables,* cress, tomatoes, cabbage, cauliflower, sprouts, artichokes, mushrooms, truffles, olives, onions, garlic, celery leaves, lettuce, cucumbers and radishes; of *fruits,* only whortleberries, young raspberries, and green gooseberries should be eaten, though fruit sugar (levulose) is better tolerated than starch or cane-sugar. *Desserts,* (soufflés) of eggs, gelatin, and lemon; tea, coffee, diabetic cocoa (Rademann's) and von Hoevel's saccharin chocolate, may also be taken.

To some cases, and in definite amounts, von Noorden allows the following (after determining the limit of tolerance, as outlined under URINE [glycosuria, *v.s.*]); beans and peas (ʒj); turnips, carrots, celery bulbs, wax beans (℥j); radishes (℥j); walnuts 6; hazel-nuts 10; almonds 8; (no chestnuts); apples, pears, apricots, peaches (℥j to ij); berries (℥ss to j); cooked apples, plums, pears, peaches, cherries (ʒj); milk (8 to 9 ℥), (of which many practitioners allow a larger measure).

Gradual withdrawal of carbohydrates is tolerated better than abrupt restriction. Glutin flours differ but little from ordinary flours in their percentage of starch. Aleuronat flour contains but half the starch of ordinary flour. Von Mehring believes that fat is best absorbed when some carbohydrates are given with it. If some starch can be given, Mossé prefers to allow *potatoes* which are more easily assimilated and contain but 16 to 24 per cent. of starch (bread contains 55 per cent.). Ingestion of more than 5 ounces of *butter* daily may increase the β-oxybutyric acid, but even 7 ounces may be taken if the butter is carefully washed in cold water, which extracts the lower fatty acids (von Noorden). Saccharin should be used in place of sugar, gr. iss daily; it is 280 times as sweet, and is antifermentative, but an excess may occasion dyspepsia. Naunyn advises an occasional *fast day,* and Austin Flint kept his cases in bed, and without food from Saturday night until Monday morning. As the disease is eventually progressive, a schematic diet should not be insisted upon; reasonable restriction, with *occasional dietetic treats* is less often violated by the diabetic, who is always hungry for bread and potatoes. The patient's weight, color and strength are of more significance than the urine. Though *alcohol* is a protoplasmic poison, it aids in the absorption of fat, prevents tissue waste, contributes energy and heat, and is a nervous sedative and cardiac tonic.

Special diabetic treatment of the individual forms (according to von Noorden); (*a*) *mild forms of glycosuria. In elderly persons* it is not necessary to determine with great accuracy the sugar tolerance. The patient may eat bread, potatoes and vegetables according to his desire, for the quantity of sugar (1 to 2 per cent.) in the urine is usually insignificant. The older the patient and the more corpulent, the more careful should the physician be about reducing the body-weight; fatty foods should be increased, butter being used freely on bread, potatoes, vegetables and meat; and the patient should have bacon, eggs, fatty cheese, light wine, coffee, tea and carbonated water (total of one to one and a half pints). Muscular *exercise* is essential, but should be regulated according to the bodily strength and condition of the heart. A course at Carlsbad is beneficial for obesity, gout or mild circulatory weakness. In *young persons,* carbohydrates should be given with much greater caution, as carelessness in diet is more frequent, and the glycosuria more often progressive. The limits of tolerance for carbohydrates should be closely watched. All carbohydrates must be forbidden excepting bread and potatoes, in addition to which the patient should have the following articles: 2 ounces of butter, with bread, cheese, and potatoes (480 calories); 2 eggs (150 calories); 3 to 4 ounces of olive oil with salad, cucumbers, etc. (90 calories); 1 ounce of fatty cheese (115 calories); 1 quart of milk (590 calories); and one ounce alcohol (210 calories); meats, *v. s.* (*b*) *Moderately severe forms* embrace cases in which the excretion of sugar is arrested or reduced to small amounts (1 to 2 per cent.) only by total abstinence from carbohydrates. The rise or fall of the tolerance limit should be watched every month, and every four or six months all carbohydrates must be withdrawn for three weeks. The diet must contain much fat; butter, 3 to 4 ounces (890 calories) must be weighed out every morning, and eaten on bread, vegetables, meat, fish, etc., either in solid form or melted. Olive oil, two-thirds of an ounce (186 calories) should be taken on salad (lettuce, chicory, cress, cucumbers, tomatoes, red cabbage), and the same amount as mayonnaise dressing for salad, cold meat, fish, lobster, etc. Bacon, two-thirds of an ounce (140 calories), 5 eggs (390 calories), and one ounce of brandy (280 calories) should be given. In addition the patient should have meats, and some of the articles mentioned in the conditional list under general diet. (*c*) In *severe forms*, despite a continued rigid diet, sugar is constantly excreted. These cases usually occur in early life with emaciation, frequent complications and a fatal issue after a few months or years. Careful dietetic treatment may retard the progress of the disease, prevent complications, and delay the fatal issue. The dietetic treatment is the same as in moderately severe cases. Carbohydrates aggravate the diabetes, but cannot be wholly interdicted. Less starch and more fats and alcohol are given than in other cases.

2. **General Treatment.**—Zimmer demonstrated the value of (*a*) *exercise* in lessening glycosuria, but as in obesity, we have to deal with an added factor, the improved appetite. The condition of the heart must govern the amount of exercise to be taken as Frerich early noted that fatal syncope and diabetic coma may occur after exhausting exertion. (*b*) The *mouth* and *skin* require special attention. Cleanliness averts in large part gingivitis, furunculosis and genital complications. Frequent warm (not cold) baths and change of underwear, gentle brushing of the teeth and soothing salves for eczema and similar eruptions are imperative. (*c*) Diabetics for the most part should be kept at home and at work, for they often become restless and desire to wander. A prolonged stay in a quiet place is better than travel. Sea bathing and exposure to cold and dampness are to be avoided. (*d*) *Suggestive* treatment may often be combined with the dietetic and medicinal therapy in depressed subjects, particularly in neurotics. (*e*) *Mineral springs* are suitable only for mild cases and those with obesity, gout and slight circulatory disturbance, in whom there is increased tolerance for carbohydrates. Carlsbad and other spas are contra-indicated in severe, youthful, very aged, reduced and nephritic cases. (*f*) The *mode of life* should be quiet, and avoidance of excesses is indicated.[1]

3. Treatment of Complications.—(*a*) *Tuberculosis* is the most serious complication. The liberal diet, necessitated by tuberculosis, is contraindicated by glycosuria. A low altitude and mild climate should be selected. (*b*) *Gastro-intestinal* catarrh often precipitates diabetic coma. As in infantile disorders, starvation for one or two days is more efficient than attempts at intestinal disinfection; only boiled water should be given; alcoholic stimulation is often indicated when there is exhaustion, red wine acting as a stimulant and astringent. (*c*) *Constipation* (*q.v.*) should be relieved by rhubarb and sodium bicarbonate; severe purges must be avoided. (*d*) *Nervous symptoms* of the neurasthenic order are observed in neurogenous diabetes; sodium bromide, opium, codeine or acetanilide may be indicated. Of all remedies, opium alone exerts an appreciable influence upon the glycosuria; it was recommended by Dobson (1774); its affects are observed more in the severe than in the light type, *i.e.*, it decreases the transformation of albumins into sugar, but does not control the transformation of carbohydrates into sugar; the average dose may be gradually increased without the patient exhibiting toxic symptoms, or suffering from its reduction afterward. Codeine may be given in doses of gr. j to x, though it is seemingly less effective than opium; it may be combined with sodium bromide ʒss. Inunctions of mercury are seldom valuable, but are indicated when cerebral or pancreatic syphilis is suspected. (*f*) *Neuralgia* and neuritis should be treated dietetically; the salicylates, salol and aspirin may benefit mild cases as well as somewhat modify coincident gout or intestinal fermentation. (*g*) *Acidosis*, acetonæmia, and *diabetic coma* are serious complications. Acidosis is said to be promoted by a rigid dietary (Naunyn, von Mehring); in pronounced acidosis even von Noorden, who opposed Naunyn's view, recommends an oatmeal diet, with butter āā ℥viij at frequent intervals; he adds eggs to this dietary which is preceded for a few days by a vegetable diet. Some writers would also exclude fats. Sodium bicarbonate and sodium citrate ʒij to ʒiij, t.i.d., and calcium carbonate ʒj once or twice a day may possibly postpone threatening coma. Much water should be given by mouth, by rectum or subcutaneously with sodium carbonate ℥iss to O ij. Developed coma is practicably incurable. Oxygen inhalations, alcoholic stimulation, strychnine, mild laxatives (not drastics) and subcutaneous or intravenous injections are given, but rarely with success. (*h*) *Gangrene* should be treated expectantly until there is a possible halt in the process, and definite demarcation; positive rules of treatment are laid down with difficulty. In personal experience, advanced diabetics stand operative shock and anæsthesia poorly. Most of them have died from alarmingly rapid syncope or from typical dyspnœic coma. Other operations may be performed, when indicated as absolutely necessary life-saving procedures, and only, if possible, after strict dieting. Boils, furuncles, etc., are treated expectantlv.

(B). DIABETES INSIPIDUS.

efinition.—A chronic malady characterized by large amounts of ne of low specific gravity. It was described by Willis (1674), who t differentiated between diabetes mellitus and insipidus (without te, *i.e.*, non-saccharine). It is uncommon; Eichhorst observed ases in 35,942 cases (0.02 per cent.), Futcher 7 in 403,535 (0.001 cent.), and 55 occurred in 113,600 in the Berlin Charité (0.048 cent.).

tiology.—Two groups of cases are observed: (*a*) the *idiopathic* rvous or vasomotor) and (*b*) the *symptomatic*, which occur in (i) *in disease*, especially basal syphilis and less often tumors of the lulla and the floor of the fourth ventricle, cerebral traumatism, lar meningitis, apoplexy, hydrocephalus, and lesions of the middle bellar lobe or corp. trapezoides of the pons. (It is possible that kidneys may be at fault, losing their power to concentrate the e.) (ii) It may result from *abdominal* diseases, aneurysms, tumors uberculous peritonitis. (iii) Insolation, diabetic ancestry (Trous- u), malnutrition (Ralfe) with tuberculous, syphilitic or gouty cedents, acute infections and alcoholism are numbered among ossible causes. Most cases occur in *males*. In Strauss' collection 5 cases, 10 per cent. were under five years old, 14 per cent. under and 45 per cent. were between ten and twenty-five years of age; rding to Jacobi, 25 per cent. occurs under ten years. Weil col- ed 23 cases among 91 individuals of four generations, all of whom yed good health. Congenital cases are recorded, but are rare.

ymptoms.—The onset is usually gradual, though abrupt symptoms e followed fright. (*a*) The *urine* is *greatly increased;* a secretion 0 to 40 pints daily is not rare, and even one of 90 pints is on record. re urine is passed at night than by day. It is said that less urine xcreted than water ingested. It is passed rapidly after ingestion water (tachyuria). Its specific gravity ranges between 1,000.5 ,001 and 1,005. It is pale and the total solids are usually normal, ugh sometimes the urea, chlorides and phosphates are increased, ng washed out from the tissues. Albumin, sugar and inosite are uncommon. (*b*) *Polydipsia* is secondary, though it is perhaps earliest symptom noticed. In one case, a physician with brain hilis, 2 gallons of water were drunk each night between 10 P.M. 7 A.M. (*c*) The *skin* is dry; carbuncles are rare. The saliva is reased. The appetite is usually not increased, though phenomenal yphagia marks some cases. Nervousness, headache, irritability, eased knee jerks, impotence and severe lumbar pain radiating the legs, are quite frequent. Nutrition is often good in the idio- hic cases, and no unusual metabolic findings are noted; in the mptomatic variety the condition of nutrition depends on the causal ease; in children growth may be retarded. The temperature is and chilling occurs readily. Aside from obvious causal lesions,

vascular, retinal and uræmic manifestations; (*c*) from
dipsia with secondary polyuria, which is exceedingly
from *transitory polyuria*, which is distinguished by it
as well as by its obvious etiology, as recent fever, hyst
of massive exudates, excessive medicinal diuresis, and
thral obstruction with sudden release of urine.

Prognosis.—The prognosis is obviously more favorab
forms, although cerebral syphilis doubtless causes many
Less than one-half of the patients recover, though de
frequently from the disease itself (save cerebral or abd
than from intercurrent infections, notably pulmonary t

Treatment.—Aside from antisyphilitic therapy, dru
use. Iodides benefit some cases in which there is no re
syphilis, other than the therapeutic results. Opium
as in the saccharine form, and large doses are often
Valerian (ʒj of the root, t.i.d.), gallic acid (gr. v to x
(fluid extract ♏xx, t.i.d.), sodium bromide (ʒss t.i.
(gr. v to x, t.i.d.), and electrotherapy are recommen
has clearly helped some cases. Talquist considers a
efficacious than a meat diet. Posner advocates chewi
apparently decreases the polyuria and polydipsia.

(C). GOUT.

Definition.—An obscure metabolic disease, characte
periodic or chronic inflammation of the joints with ura

Mentioned by Hippocrates, gout was clearly descr
Sydenham (1683), who suffered from the affection f
Murray Forbes (1792), and Wallaston (1797), recogn
nature of the joint deposits.

Etiology.—1. The *actual cause* is unknown. In
advanced the hypothesis that uric acid accumulat
(before the gouty seizure) in the blood. His, Vogel, an

idea is advanced that uric acid is not produced in excess, but is retained in the body because of periodic attacks of renal insufficiency. Whether the real metabolic change concerns the uric acid, the alloxuric or purin bodies is yet undetermined.

2. The *extrinsic, predisposing* factors are somewhat better defined. (*a*) *Heredity* is an apparent factor in 50 to 60 per cent.; transmission by the father is more common than by the mother, and the younger rather than the older children suffer from hereditary influences. (*b*) *Mode of life*; lack of exercise, over-eating, an almost exclusively meat diet, and alcoholism are potent factors. The old verse said that "Wine was the father, Eating the mother, and Venus the midwife" of gout. Though often true that gout affects the well-to-do (arthritis divitum), and arthritis deformans the poor (arthritis pauperum), gout is not uncommon among the lower classes. As alcoholism is a widespread scourge, it would seem that the heavy ales and beers of England and Germany are more injurious (in this respect) than whisky and light wine. Gout may occur in temperate individuals. Obesity, diabetes, arteriosclerosis, and calculous tendencies are concomitant or coördinate rather than causal conditions. (*c*) *Lead poisoning* is observed in 20 per cent. of cases or more, particularly in England, and its relation to gout is puzzling, though saturnine nephritis may explain it. (*d*) *Men* are particularly prone to gout (1 to 13 or 25). (*e*) *Age.* Gout begins between thirty and forty years in 38 per cent., and thirty and fifty in 60 per cent. Much less than 1 per cent. of cases begin before the seventeenth year, though Gairdner observed one case in a nursling. (*f*) England leads in frequency of its occurrence. In the United States it is rare, though Futcher shows that the cases in Baltimore and London hospitals stood as 2 to 3 (certainly not true for other American cities); again, relatively few cases seek hospital treatment.

Symptoms.—These may be considered under two captions, (1) acute, and (2) irregular, chronic and visceral gout.

1. *Acute* (regular, sthenic or) *typical gout* usually appears without prodromes, though an attack is sometimes heralded by digestive disorder (pyrosis, gastralgia, constipation), "cerebral congestion" (insomnia, vertigo, tinnitus, irritability, depression), paræsthesia, cramps or pain in the muscles, dark lateritious urine or other vague symptoms. The *paroxysm is remarkably characteristic.* (*a*) *Local findings:* The patient is awakened one to three hours after midnight, by an agonizing pain in the metatarsophalangeal joint of the large toe (*podagra*). The pain is described as crushing by a vise or like burning by a hot iron. Localization in the toe is so frequent (95 per cent., Garrod, or 73 per cent., Scudmore) as to be practically diagnostic of gout. The skin over it becomes hot, red, tense, slightly œdematous and exquisitely sensitive. Serum exudes into the joint. The pain abates toward morning, but the inflammation increases. Each early morning sees a repetition of the pain for three to five or less often for six to seven days; the paroxysmal pain and the local findings then gradually subside by lysis; in two-thirds of the cases,

the skin desquamates over the joint. Later attacks may involve
thumb, knee and other joints, though less commonly than in chro
irregular gout (*v.i.*). The local symptoms vary in intensity; as a r
the shorter the attack, the severer are the symptoms. The *patholog*
changes probably develop in this sequence: (i) local stasis (infla
mation), (ii) necrosis, and (iii) uratic deposit; the ordinary ura
(biurate of sodium in conjunction with sodium phosphate) do
cause such local reaction, but the biurate alone, even in most dil
solutions (0.004), produces inflammation (Pfeiffer); acute resid
swellings (acute gouty tophi) consist of this biurate which has b
precipitated by the carbonates of the blood (and tissues). Con
escence is complete in ten to fourteen days, often with greatly impro
spirits and health after the gouty attack. During the seizure (*b*)
stitutional symptoms are not absent. The temperature rises to 100
or 101°, rarely over 102°. The pulse is usually slow and hard, so
times arrhythmic and seldom exceeds 100. The heart may palpita
Furring of the tongue, slight leukocytosis, anorexia, nausea, vomiti
epigastric cramping and eructations are not uncommon. The ur
is dark, usually decreased, acid, and higher than normal in spec
gravity; the difference of opinion regarding uric acid has been m
tioned under etiology; Bain and Futcher find a close paralleli
between the uric and phosphoric acids, both being derived from d
integration of nuclein; they both fall below normal in the quiesc
period and increase during the attack; sometimes the indican a
urobilin are increased. Garrod's test for urates in the blood, exuda
or transudates, consists of allowing it to clot, adding 10 drops of
per cent. acetic acid to ʒij of the serum, and obtaining the cryst
after twenty-four hours.

Subsequent acute seizures may sometimes be averted by diet a
exercise, but they are likely to recur, notably in the spring and fa
Excesses in alcohol or in eating may precipitate acute gout; inde
in one case it would occur within fifteen minutes from drinking
glass of champagne; trauma, exertion or excitement may favor relap

Retrocedent or *suppressed gout* was once thought to develop wh
an acute paroxysm abated "too rapidly"; many symptoms attribut
to it are doubtless due to coincident arteriosclerosis or uræmia (*e.*
gastralgia, vomiting, cardiac pain, dyspnœa, delirium, convulsio
apoplexy, coma and death).

2. *Chronic, irregular, atypical gout* generally follows acute atta
which increase in frequency, decrease in acuity and show slight t
dency toward resolution. Pfeiffer claims that gout deposits (*to*
cannot occur without acute attacks. In some cases it appear
begin as chronic gout, particularly in the aged, the cachectic an
women with a gouty family history, all of whom lack reactive po
(atonic or asthenic gout). (*a*) *Local findings:* there is less pain, fe
and redness but greater and more permanent swelling than in
acute form, until the condition eventually becomes fixed and chro
the "attacks" may be most rudimentary, and remissions may altern

with exacerbations. The *lower extremities* and hands are most often affected; it may also develop in the thumb (*chiagra*), knee (*gonagra*), elbow (*pechiagra*), and spine (*rhachisagra*), but the shoulder is very seldom and the hip almost never invaded. In some cases its moving from joint to joint (*urarthritis multiplex seu vaga*) may cause confusion with acute rheumatism. Uratic deposits (*tophi arthritici*) form in the cartilage and later in the capsule and ligaments, and the swelling never recedes essentially. These prominences, and the attendant fibrosis, lead to deformation, subluxation, crackling on movement, ankylosis and contractures. Sometimes fluctuation is noted, especially in the big toe. The proximal joints of the fingers may point to the radial, and the others to the ulnar side. Tophi may rupture, evacuating yellow-white urates, though incompletely, as they are partly diffused through the tissues; these gouty ulcers heal tardily, if at all, for their basis is fibrous and but slightly granulating tissue. Tophi occur also on the ear (25 per cent.), bursæ, tendons, aponeuroses (which may explain some of the cases of Dupuytren's palmar contracture), and even in the skin, eyelids, nose, larynx, penis or scrotum. (*b*) *Visceral gout* includes a curious mixture of real gouty complications and incongruous doubtful symptoms ("gouty disposition," *arthritisme, herpétisme*, and the so-called "uric acid diathesis").

Complications.—*Renal complications* are the most important visceral complications, and sometimes dominate the clinical features. In 1,440 cases, Garrod observed albuminuria in 26.5 per cent., more often in advanced than in early cases. Its exact relation to gout is hard to define; it may follow the metabolic changes, or according to Ebstein may possibly constitute a primary gout. At first albuminuria may be considered toxic, marantic, congestive or arteriosclerotic but later the renal, cardiovascular and retinal findings of interstitial nephritis develop with all its accidents and complications; uratic deposits occur in the intertubular tissue and sometimes in the secreting tissue of the cortex and medulla, in 15 per cent. of cases, according to Moore. Uratic deposits (calculi) are not uncommon in the renal pelvis; hæmaturia, oxaluria, pyelitis, cystitis (and perhaps urethritis) may also occur.

Arteriosclerosis of the ordinary type is promoted by the individual's mode of life, by metabolic changes, alcoholism, plumbism and nephritis; it develops early and causes many (and, with nephritis, most) of the so-called visceral gout symptoms, as the *cerebrospinal* (headache, vertigo, brain congestion, epileptiform and apoplectiform attacks, encephalomalacia, myelitis), the *circulatory* (cardiac hypertrophy, myocarditis, aneurysm, angina pectoris, atheromatous valvular lesions, phlebosclerosis), and the *respiratory* symptoms (stasis, bronchitis, asthma), and retinitis.

Eczema (18 per cent.), scleritis and episcleritis (with small circumcribed points of inflammation, and sometimes semimicroscopic tophi), euralgia or less often neuritis, lumbago, sciatica and pain in the rachial plexus, burning and psoriasis-like patches on the tongue, digestive symptoms (*v.* PRODROMES), and burning in the eyeballs are probably often gouty. On the other hand, the "gouty disposition"—in some descriptions—consists of very vague symptoms; on analysis neurotic, paroxysmal (calculous colic, migraine or asthma), and other conditions which are common in persons not suffering from gout, account for many so-called visceral gouty manifestations.

Diagnosis.—(*a*) *Acute gout* is most typical in its etiology, history and localization, with acute, circumscribed redness. In a few cases (urarthritis multiplex), a diagnosis of acute articular rheumatism may be made; fibrous rheumatic nodes may be confused with tophi, but if the possibility of gout is recognized, there is usually no error. (*b*) *Chronic gout* often has a history of acute attacks, usually involves the extremities and distal joints of the hands and feet, is marked by unequal distention and thickening of the soft parts, and is often known by its association with early arteriosclerosis, contracted kidney, obesity, renal calculus and diabetes. The *x*-rays cannot determine the diagnosis (see ARTHRITIS DEFORMANS, DIAGNOSIS). Visceral gout is an entity only when it develops in a patient who is undoubtedly gouty.

Prognosis.—Gouty patients may live to an old age, but the usual recurrence every two months to two years gives a tendency to chronic gout, of which the issues are (*a*) uræmia, (*b*) arteriosclerotic complications, as coronary disease or apoplexy, (*c*) terminal infections as pericarditis, pneumonia, and pleurisy, which may be induced by the (*d*) gouty marasmus or cachexia. In general the outlook is better in frank gout than in irregular gout, and better in cases which develop after forty years of age.

Treatment.—**1. The Acute Paroxysm.**—(*a*) Absolute *rest* is indicated. (*b*) For *pain*, the use of *colchicum* is empirical; some practitioners believe that it increases the elimination of uric acid, though the claim that it enables the tissues to break down, and dispose of the uric acid seems more reasonable; nevertheless, it causes the disappearance of uric acid from the urine of those passing it in quantities, and in gout it relieves pain and inflammation and acts as a cholagogue; ʒiss of the vinum colchici radicis (not seminis) should be given daily. It must be administered with care in nephritic and marantic subjects. If long used, a tolerance to its action is acquired. It should be stopped when pain is alleviated and it does not act well when diarrhœa intervenes, which interferes with its diuretic effect. (Full therapeutic doses produce a slow pulse, nausea, vomiting, pain and purging; inversely to its action on the bowels there is diaphoresis and diuresis. It is eliminated by the kidneys, bowels and skin. After toxic doses, great nausea, retching, serous and finally hemorrhagic purging, great pain, headache, vertigo, prostration, paralysis of the peripheral sensory nerves, motor part of the cord and finally of the respiratory centre [the usual cause of death] develop; ecchymoses of the gastro-intestinal tract with swelling may be seen at the postmortem examination.)

If colchicum produces no effect, *sodium salicylate* should be given, gr. xv every two hours (ʒj to ij daily); it usually relieves the pain and swelling, and probably aids in solution of the uric acid; its early effect is greatest, and it usually fails after the second or third day. Finally, *phenacetin* (gr. viij to x) and morphine are indicated in severe paroxysms. In prolonged attacks, potassium iodide gr. v (which should be given with care in nephritis), and wine of colchicum ♏x are useful. (*c*) The *diet*, according to Pfeiffer, should consist of foods which "release uric acid in a free form," as meal soups, rice and other carbohydrates; meat and eggs should be withheld (though given in chronic forms, and in the intervals between attacks). Plain or acid water, as Seltzer (not alkaline water as Vichy) should be given freely; alcohol and lemonade must be forbidden. If laxatives are indicated, calomel, rhubarb or castor oil should be used. (*d*) *Local measures* should be largely avoided lest added necrosis and tophi develop; especially, vesicants, the use of the ice-bag and massage; warm applications of 50 per cent. alcohol or lead water (tinctura opii, liquor plumbi subacetatis, āā ʒij and water ℥j) may be used.

2. The Interval Between Attacks.—(*a*) Regulation of diet is the cardinal indication. Moderation in the quantity is more important than the quality of the food. *Quantity:* a full diet increases the uric acid, taxes resorption which is usually impaired, and favors acid fermentations which engage the diphosphates and carbonates of the food necessary for the formation of soluble uric acid salts; three medium-sized meals a day are allowed. *Quality:* (i) *alcohol* should be absolutely forbidden; (ii) *water* may be given freely, little with the meals but large amounts on an empty stomach. (iii) Moderate amounts of *fat* are allowed. (iv) *Fruits and vegetables* increase diuresis, and their malates and citrates help to dissolve the uric salts. (v) *Meats* are allowed (though there are various opinions on this subject). The urea they produce seems to dissolve uric acid which the meat, if given in moderation, does not increase. Meat is essential particularly in marantic cases. There is no difference between the effect of light and dark meats; those rich in nuclein (sweet-breads, liver, and kidneys) must be avoided. (vi) *Carbohydrates* should be largely *decreased;* this rule is empirical, though it appears that following their ingestion the uric acid (as compared with the urea) increases nearly two-fold. Milk should be avoided, for its lactic acid is injurious. (*b*) *Exercise* is indispensable, but prolonged or fatiguing effort, hurry and worry are to be avoided. (*c*) *Medicinal treatment* is rather secondary. Alkalies produce an increased output of uric acid, but they operate only by preventing decomposition of the neutral (bi-) sodium phosphate (the chief uric solvent). The kind of alkali used is not essential; lithium carbonate is inferior to *sodium carbonate* or *bicarbonate*, which act first by promoting the formation of bisodium phosphate, and second by neutralizing the acid formed by fermentation; it may be given (in doses of gr. x upward) for a long period of time, and has no effect on the blood; but the twenty-four-hour collection of urine should not become alkaline (lest alkaline deposits in the renal pelvis develop). Mineral waters merely operate in the same way, as

those of Saratoga, White Sulphur, and Bedford Springs in this coun
Bath and Buxton in England, Aix-les-Baines, and Contrexevill
France, and Fachingen, Carlsbad, Homburg, Wildbad, and Tep
Luff advises the use of guaiacum resin, gr. v to x, t.i.d. Pipera
lysidin, urotropin, urosin and sidonal are much inferior to the sim
sodium salt.

3. Chronic Gout.—This is difficult to treat. For general diete
hygienic and medicinal measures, see above. Colchicum wine,
and potassium iodide, gr. v to x, may relieve the condition of
joints and the gouty asthma, neuralgia, and eczema. Tonics, pr
cipally arsenic, are indicated in marantic forms. Tophi should not
operated on; local measures are useless, save gentle massage,
heat, moist applications or salicylate salves at night (ac. salicylic.
lanolin 40, and oleum terebinthinæ 1 part), though all treatme
generally fails.

(D). ARTHRITIS DEFORMANS AND CHRONIC RHEUMATISM.

After excluding gouty, tuberculous, syphilitic, gonorrhœal and hæm
philic affections, there remains a group of articular affections of whi
the chief are arthritis deformans and the so-called chronic rheumatis
It is almost certain that a number of totally different affections a
gathered under these names. Their classification is most unsati
factory.

Arthritis deformans, which has been noted in Egyptian mummie
is a progressive or at least chronic affection, marked by changes
the synovial sac, cartilages and bone, causing functional disturban
of, and usually deformity in, the articulations, usually symmetric
and polyarticular; also called rheumatoid arthritis, and chron
deforming rheumatism.

Etiology.—(*a*) The predisposing factors are (i) *heredity,* which
causative in from 2 (Charcot) to 13 per cent. (Garrod); (ii) *age;*
per cent. of cases occur between twenty and fifty years of age; 5
cent. under twenty years and 40 per cent. over fifty years of age (G
rod); (iii) *sex;* though Stewart and McCrae consider that the se
are equally involved, Garrod observed 82 per cent. in women (
the disease possibly sustains some relation to the genital function
(iv) fatigue, cold, dampness, poor hygiene, trauma or sorrow m
also be predisposing factors.

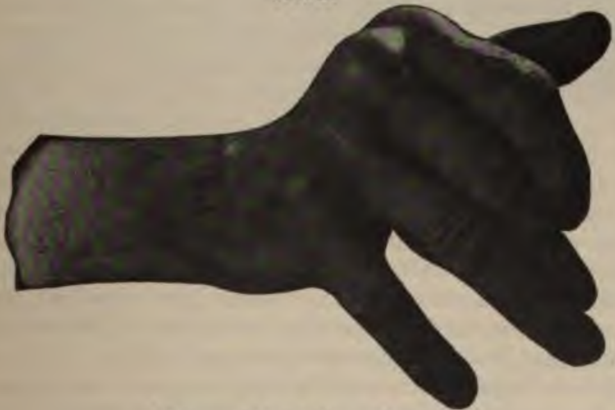
(*b*) *The immediate cause* is unknown. There are many theories
(*a*) the *neuropathic* theory (Mitchell), is that it results from a cor
lesion; this is now nearly abandoned; (*b*) the *infective* theory is base
upon finding bacteria in the joints, enlarged spleen and lymph glands,
and its occasional development after influenza, angina, and scarlatina;
Scheuller found a bacillus, Chauffart a diplococcus, and Bannatyne
and Blaxall have described microörganisms of inconstant types; (*c*)
the *rheumatic* theory, according to which it is a chronic rheumatism;

Garrod and Bannatyne find few heart lesions, though Pribrâm finds about one-fifth as many as in rheumatism.

Pathology.—Whether the synovia or cartilages are first involved is undetermined; the x-rays do not solve the question; practitioners, who see cases early, hold that the serous sac is first affected, and surgeons find the cartilage most often diseased. The synovial villi hypertrophy and the cartilage slightly fibrillates, cleaves, and finally atrophies. The bone becomes thin in some places and tuberous in others (Haygarth's nodosities) from periosteal proliferation; the joint surfaces become eroded and in part grow together.

1. Diffuse Progressive Type.—(*a*) The *acute form* is uncommon. There is some fever, rise in the pulse rate, and depression, and the joints become red and swollen, resembling in some ways acute articular rheumatism, but differing from it in several essential points; that is, arthritis deformans is less fugitive, involves perhaps the small joints more frequently, is attended by less sweating and very seldom by heart complications, does not react to salicylates and runs a much longer course. In some cases it has even resembled typhoid, because the fever and splenic enlargement lasted for some time before the joints became affected. (*b*) The more frequent *chronic form* develops insidiously, with uncomfortable sensations in the joints, pain or tearing nocturnal pains about the joints, and vasomotor skin symptoms. It begins most often in the small joints of the hands, with or without swelling, and remits and recurs. The hands often sweat. The fingers become fusiform from changes in the joints, capsular thickening and collateral œdema which separate the fingers from each other. The œdema often disappears, showing more clearly the bone and the capsule thickening. The cartilages become uneven and crackle on movement, loosened villi may become free bodies, and the adjacent bursæ and tendons may become swollen. The fingers deviate and become angular from flattening of the saddle-shaped epiphyses and muscular

Fig. 48.



Rheumatoid arthritis. (Musser.)

contractures (now extensor and again flexor in type). Extension to other joints occurs; the elbow becomes flexed, the shoulder often crackles and becomes fixed; the large toe is bent out, and lies under the other toes which are often extended at their first, and flexed at their two distal articulations; the ankle is often abducted; the knee is swollen, shapeless and flexed from fluid effusion or contracture; in a few cases the hip, jaw, and spine are involved.

Motion is limited by the swelling, muscular contractures, fixation of the joints by interlocking extracapsular nodosities, and capsular induration. The contiguous muscles atrophy from disuse, local inflammatory invasion or reflex influence on the cord. The skin may become puffy, glossy or even atrophic. The reflexes are chiefly increased, as the patellar (in 80 per cent.). Neuritis is not uncommon. In 64 cases in which the vertebræ were involved, Braun found nervous symptoms in 88 per cent., as weak muscles, occipital neuralgia, involvement of the brachial plexus and anæsthesia. The heart was not affected in any of Garrod's 500 cases. Anæmia, episcleritis and other complications are uncommon. The majority of patients do not improve or recover, and recurrence of the disease is very common. In some cases the process is arrested. Aside from atheroma and nephritis few intercurrent diseases develop.

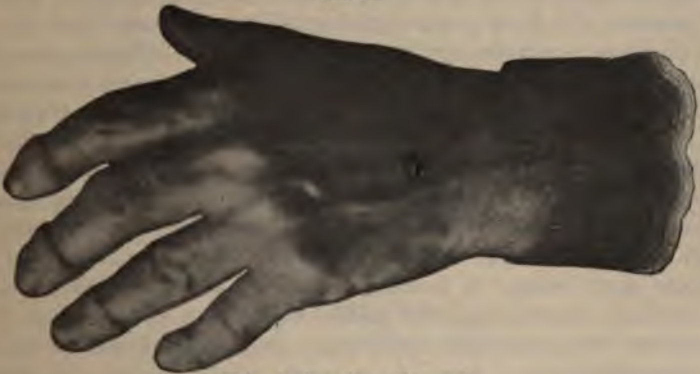
2. Partial or Monarticular Type.—Pathologically this type resembles the form described, but differs from it in that it presents more profound changes, and is limited to one or few articulations. There is the same capsular thickening and cartilaginous fibrillation; the cartilage often softens, ossifies or hypertrophies; the bone rarefies or becomes cystic and soft in some places, with flattening of the bone heads and widening of the sockets, particularly in the shoulder and hip; in other places bony over-growths form, known as Haygarth's nodes, or a new socket may develop; the bone may eburnate and the joint may be either smooth or ridged. As a rule the atrophic prevail over the hypertrophic changes. They resemble the extreme senile change (*morbus coxæ senilis*); trauma or a nervous basis are considered causal; it occurs in old persons and largely in males. Clinically, compared with the general progressive type, it is single or at least rarely symmetrical, occurs in the proximal large joints (hip and shoulder), and is less often attended by subluxation. The elbow, spine and smaller joints are less involved.

3. Vertebral Type.—Struempell (1885) named this type *chronic ankylosing spondylitis;* vertebral arthritis deformans occurs with ankylosis of the spine, shoulders and hips. Marie (1898) made it a separate type (*spondylose rhizomyélique*), characterized by complete vertebral ankylosis, scoliosis, ankylosis of shoulders and hips, exemption of all other joints, incidence almost exclusively in males, and its appearance in the young immediately after bony development has ceased. Von Bechterew (1892) described a similar type due to heredity, trauma or meningeal inflammation and characterized by paresis of the muscles of the neck, trunk and extremities, some atrophy of he back and shoulder, signs indicating involvement of the spinal erves (hyperæsthesia, pain and paræsthesia), and an ascending egeneration in the cord. These are probably but subtypes of arthritis eformans.

4. Juvenile Type of Still.—(*a*) The ordinary progressive (adult) orm may be observed, running an unfavorable course, and often ith almost universal ankylosis; in some cases the great toes are engthened. (*b*) "*Still's type*" occurs in poor children largely; 70 er cent. of cases are girls; 30 per cent. occurs between the second nd seventh years, 32 per cent. between the seventh and thirteenth ears, and 20 per cent. between the thirteenth and seventeenth years. The onset is often subacute. The multiple arthritis affects chiefly the xtracapsular tissues, and there is seldom crepitus. The spleen is lways enlarged, and the lymph glands near the affected joints are ard and large. Anæmia and retarded development are common.

5. Heberden's Nodes.—Bony nodes develop on the sides of the end halanges near the joints. They begin between thirty and forty ears, but are seen chiefly at about sixty years of age. Though at first soft, and sometimes painful and red, they become hard, indolent and as large or larger than a pea. Their growth is more often intermittent than continuous. Some disturbance in function is not uncommon, but only in extreme cases is deformity observed, as flexion of the end phalanges into the palm or deviation toward the radial side. Involvement of the large joints is rather uncommon, whence the prognosis is favorable. Some consider them gouty, though Heberden dissented strongly.

FIG. 49.



Heberden's nodes. (Musser.)

Diagnosis.—(*a*) In *gout*, the history, localization, fine (not coarse) crackling in the knee-joint and radial articulation of the elbow, greater spontaneous pain, infrequent generalization and symmetry, the uratic deposits and tophi in the ear are characteristic. (*b*) *Acute rheumatism* (*v.s.* ACUTE FORM). (*c*) *Multiple tuberculous arthritis* may cause diagnostic difficulty, particularly when it occurs in children, for pulmonary tuberculosis may complicate arthritis deformans; but the tuberculous hip and knee involvement, the fusiform fingers, the hectic fever, involvement of the glands and serous membranes are generally distinctive. Poncet has recently called attention to an ankylosing tuberculous pseudorheumatism, which may cause confusion (*v.* TUBERCULOSIS). (*d*) *Syphilis* (*q.v.*) of the joints occurs particularly in persons between four and fifteen years of age, and in about 40 per cent. of hereditary forms; the epiphyses are irregularly deformed. (*e*) *Hæmophilic* arthritis is distinguished by the unmistakable family history and its three stages (i) hæmarthros, (ii) panarthritis, and (iii) adhesions, erosions, deformity and ankylosis. (*f*) *Chronic articular infections* (chronic pseudorheumatism), especially subchronic and chronic gonorrhœal arthritis (*q.v.*), begin acutely, usually affect the knees first, then possibly after months the ankles, hands (in women particularly) and sternoclavicular and other joints; similar chronic arthritides may follow scarlatina, diphtheria, pneumonia, and grippe. (*g*) *Nervous arthropathies* present much the same pathological findings; (i) the arthropathies of *tabes* (*q.v.*), occur rapidly and early, without pain, tenderness or other inflammatory evidence; are attended by much effusion, are atrophic or flail-like rather than hypertrophic, in 80 per cent. involve the lower extremities and are accompanied by other signs of tabes. (ii) In *syringomyelia* (*q.v.*) the effusions are great, painless, in 80 per cent. involve the upper extremities, and are associated with the syringomyelic dissociated anæsthesia, and panaritia. (*h*) "*Chronic rheumatism*" follows acute rheumatism, in which the changes involve less the bone, cartilage and serous sacs than the capsule which becomes fibrous. The cartilage may become somewhat fibrillated and fibrous but is seldom hypertrophied or destroyed. The vascular bands become callous (Jaccoud's fibrous rheumatism), and ankylosis may result. It is maintained by some that this ankylosis of connective tissue, and the joint obliteration differentiate fibrous rheumatism from arthritis deformans, but clinical differentiation is often impossible.

Treatment.—Treatment is notoriously futile and arrest of the process results rather from spontaneous exhaustion of the disease than from therapeutic efforts. (*a*) "*Rheumatic*" *influences*, as exposure to cold and dampness, overheating while at work, and resting in damp garments are to be carefully avoided. Woolen underwear should be worn. General hydrotherapeutic measures are illy borne. A warm climate benefits especially those cases resembling (or including) chronic fibrous rheumatism, as that of Mexico, the Hot Springs of West Virginia and Arkansas. (*b*) *Diet* is not of such importance as in gout; indeed, liberal feeding is strongly indicated. (*c*) In *acute exacerbations* rest and coal-tar derivatives are indicated; salicylates are generally useless save in really secondary rheumatism. (*d*) *Passive movements* and *massage* relieve stiffness and muscular contractures. Massage reduces the œdema and adhesions. In some cases forcible pressure, as with the thumb, will cause some absorption of the (fresh) eoplastic nodes; it may be easily overdone, and once every day or o on the same area is sufficient. Cautious active exercise is also ential. (e) *Extension* by pulley weights sometimes produces wonder- results according to Hoffa, in lessening pain and exostosis, though e author has never observed any essential results. (*f*) *Local measures* ay be of some use. The *x-rays* and strong electric lights may afford mporary alleviation from pain but do not seem to affect the basic ange; cases of fibrous rheumatism are more benefited than arthritis formans. The same may be said of local applications of warm ater. *Hot air* causes hyperæmia of the parts and is sometimes of al (temporary) benefit; cages of wire, asbestos and felt are devised enclose the joint, leaving around it an air space; the temperature gradually raised by a lamp placed under a connecting elbow to 0 or 250° F. and is gradually lowered. Bier's method of *venous peræmia*, used by him in treatment of tuberculous joints, is also vocated; the Esmarch constrictor above the joint should produce velling but not pain. "*Firing*" the skin with the Paquelin is bene- ial in lighter grades of deforming arthritis and in fibrous rheumatism. ncouraged by the results in one case in which injections of iodoform d olive oil were given, the author used it in six other cases, and ther without benefit or with great increase of pain and swelling (the jections were made with absolute asepsis). *Operation* (resection) may e considered in nonprogressive, monarticular forms, but the general oor health and age of the patient rather contra-indicate it. (*g*) *In- rnal medication* as cod-liver oil, arsenic and syrup of the iodide of on may seemingly help a few cases. Luff uses guaiacol carbonate ith marked success; the initial dose of five grains, t.i.d., is increased vo grains every week till a single dose of twenty grains is given.

(E). RICKETS.

Definition.—A metabolic affection of early infancy, characterized iefly by hyperæmia and deficient calcification of the growing bones. he disease was first well described by Glisson (1650). The name derived from an old English word meaning "twisted," and Glisson ggested the Greek "rhachitis" (vertebral inflammation) because the pine was often involved, and the term sounded like "rickets."

Etiology.—Its etiology is obscure. All theories advanced, such as eficient absorption of lime salts or infection, and reduced activity of he thymus, are insufficient. (*a*) The *diet* seems to be of prime im- ortance; proprietary foods, condensed milk, cow's milk, excess of arbohydrates, prolonged lactation or nursing during pregnancy are robable factors. Cheadle considered that a deficiency in animal fats nd proteids was causative; after 20 litters of lion cubs had been lost rom rickets in the London Zoological Gardens, Bland Sutton found hat a diet of milk, powdered bone, meat, and cod-liver oil prevented ickets. (*b*) *Unsanitary surroundings* may be causal, as lack of fresh

air and sunlight, crowded quarters and kindred factors observ
largely among the poor classes in large cities, particularly in Euro
Among nurslings Kassowitz (Vienna), observed it in 89 per cen
and Joucousky (St. Petersburg) in 90 per cent.; in children's clin
abroad, Marfan (Paris) found rickets in 50 per cent.; in Hollan
London, Christiana, Naples, and Rio Janeiro about the same figu
(50 to 80 per cent.) prevail; in the United States it is most freque
among negroes and Italians. Hereditary syphilis is a predisposi
factor. (*c*) *Age* may be of etiological importance. In more than 6
cases, Baginsky did not observe it in children under three months
age; his figures show 7 per cent. in children between three and
months, 28 per cent. in those between six and twelve months, 35
cent. in those between one and one and a half years, and 18 per ce
in those between one and a half and two years (*e.g.*, 88 per cent.
cases occurred in children between three months and two years
age). The occurrence of congenital rickets (apart from achond
plasia, see DIAGNOSIS OF BARLOW'S DISEASE) is usually denied
there is some authoritative testimony for it. Rickets which develo
later, up to puberty, is called *rhachitis tarda*.

Symptoms.—An experienced observer may diagnosticate the gradu
onset from the prodromes, as the irregular or tardy dentition; restles
ness, irritability, insomnia and throwing off of the bedclothes; swea
ing, particularly about the head and neck; bronchitis; digesti
disturbances, coated tongue, anorexia, malnutrition, meteorism
diarrhœa; immobility, the child crying when moved or touched, an
general hyperæsthesia. However, a positive diagnosis is not justifiab
until the bone symptoms develop.

1. Bone Symptoms.—(*a*) The *skull* usually shows the earlie
changes, generally in the first year of life, and the bone changes (*v.i.*
are most marked in the skull and thorax. The *face appears sma*
and the *head large*. Nodes or bosses of bone appear on the fronta
and parietal regions, which with the occipital flattening produce th
caput quadratum (*tête carée*); the head may be dolichocephalic. Th
anterior *fontanelle increases* forward, and in some cases all the suture
open toward the end of the first and beginning of the second yea
The fontanelle and sutures (which normally close about the fifteent
or eighteenth month) remain open, even until the fourth year (*i.e.*
there is lack of normal involution). The occiput softens (*craniotabe*
in 33 to 50 per cent., near the lambdoid suture; the occipital protube
ance remains hard. Craniotabes may less frequently occur elsewher
as in the frontal bones; besides the actual bone changes (*v.i.*), t
pressure of the pillow and of the brain further its development.
extreme cases the peri- and endocranium actually touch, and
moderate degrees the skull feels "parchment-like." Though it
not absolutely pathognomonic of rickets, and though it occurs also
syphilis, craniotabes is rather characteristic when there are also
tender quadrate head, head sweating, thin occipital hair and b
scalp veins. Craniotabes may be an element in the general and loc

spasms which occur in rickets. The frequent *systolic murmur* heard over the greater fontanelle is also heard in other affections or in health; it seldom endures beyond the fifth year, and is said to occur only in conjunction with a similar bruit in the carotids. The *upper jaw* becomes long and narrow, and the lower jaw becomes hexagonal, probably from muscular traction on the soft bones. The eruption of the teeth is delayed; they do not appear until the second or even the third year; the second teeth are crowded, carious, curved and eroded. (*b*) In the *thorax*, the rhachitic "*rosary*" appears toward the end of the first or early in the second year; it is so-called from the bead-like succession of enlarged chondrocostal joints; they are enlarged epiphyses. The *chest* itself is altered toward the middle or end of the second year; this is due to the softness of its bones, the traction exerted by the diaphragm, atmospheric pressure and sometimes by external influences, as holding of the child with a hand on the chest, or coincident respiratory inflammation. Louis' angle is often prominent, and the sternum juts forward, causing "*chicken breast*" (pectus carinatum). There is also a depression outside of the costochondral junctions which extends outward and downward, and is accentuated by some eversion of the costal arch. The thoracic deformity promotes respiratory complications and dyspnœa, which in turn aggravate it. With the deformity of the chest, we may group lumbar *kyphosis* which is arcuate and not angular, the thickened or bent clavicle and the thickening of the spine or edges of the scapula. (*c*) In the *extremities*, the *epiphyses*, particularly of the ulna, radius, tibia and fibula, *are enlarged;* this occurs at the end of the first or beginning of the second year at the same time as the rosary, craniotabes and wide fontanelle. The epiphyses, like the ribs and skull, are tender and painful. It may look as though there were "*double joints.*" Deformities may develop from muscular traction or from the weight of the body; the arms may incline outward and forward; the forearms may become convex toward their extensor surfaces. The thighs sometimes become convex anteriorly and outwardly; the legs bow outward or less often forward, especially when early walking is allowed or encouraged. Spiral or other extreme deformities are less frequent. The gait is waddling. Greenstick or actual fractures sometimes occur. (*d*) The *flat rhachitic pelvis* develops by the weight of the body pushing the sacrum into the pelvis and the counter-pressure of the thighs narrowing the anterior pelvis, so that the pelvic aperture may become heart-shaped; the acetabula lie more anteriorly, and the iliac crests flare outward. (*e*) *Pathology* of the bone affection. Normal bone grows in length as follows: there are two zones in the epiphyseal surface facing the shaft; (*a*) the *first zone* is nearest the shaft, is faintly yellow, and measures ½ mm.; it is the zone of preliminary calcification, into which normally the bloodvessels grow, and form medullary canals by absorbing small areas of bone, and in which osteoblasts appear and calcification develops. (In rickets the calcification is imperfect and irregular in this zone, and the bloodvessels grow not merely into these calcified areas but beyond them, even into the cartilage area, and absorb what little new bone is formed.) (*b*) The *second zone* lies outside the one described, is sharply demarked from it, is normally bluish, measures 1 to 2 mm. in thickness, and is known as the hyperplastic zone, in which the cartilage cells divide and proliferate. (In rickets this zone becomes larger, proliferates irregularly and is invaded by bloodvessels, and irregular foci of abortive calcification; the demarkation between the first and second zones is ill-defined). Normal bones grow in thickness by bony increase beneath the periosteum. (In rickets this layer becomes several millimeters thicker, irregular and nodose). Normal bone develops in the skull and face in the connective tissue. (In rickets the lime salts are either not deposited, are insufficiently deposited [craniotabes] or overdeposited as bosses [osteosclerosis].) The essential element of these rhachitic changes is *undue hyperæmia* and *irregular, deficient calcification;* which one is primary remains a disputed point; the bones may contain but 20 or 30 per cent. of the normal amount of lime salts.

2. General Symptoms.—(*a*) *Muscular weakness* is pronounced, as evidenced by impaired breathing, the tardy efforts to hold up the head, and disinclination to walk. (*b*) *Nutrition* is usually *poor;* there is anæmia, sometimes with leukocytosis and often with emaciation; the irritability and sweats have been mentioned under prodromes. (*c*) The *nervous system* is affected. Mentality is unimpaired. Rickets patients frequently grind their teeth. Spasmodic conditions are not infrequent, as generalized *convulsions*, *tetany* and *spasm* of the *glottis*, all of which will receive separate consideration. (*d*) Temperature is not symptomatic of rickets and always denotes complications. (*e*) *Digestive disturbances* are common; there may be anorexia, diarrhœa without pain or tenesmus, more commonly constipation and *distention* of the *abdomen* which can occur without any digestive disorder, from enlargement of the liver and spleen, and possibly from the thoracic changes. The amount of chalk in the fæces may be increased (Baginsky). The *spleen* is *enlarged* in 70 per cent., and, less often and less markedly, the liver. (*f*) Rickety children *catch cold easily* and bronchitis frequently leads to atelectasis and bronchopneumonia. (*g*) The heart may be luxated from the altered thoracic conformation, which also frequently causes hypertrophy of the right ventricle. (*h*) The urine remains normal, the alleged increase in calcium and phosphorus salts being quite inconstant. (*i*) Eczema is common.

Course, Complications, and Prognosis.—(*a*) *The onset* is insidious and the clinical *course covers a year* or more. (*b*) *Complications* include but few of immediate danger, as bronchitis, pneumonia, and spasm of the glottis which may result fatally. Later complications include the contracted rhachitic pelvis, which is of obstetrical importance, kyphosis and pulmonary tuberculosis, which is found in 36 per cent. of the fatal cases, sometimes with abdominal complications (tabes mesaraica); its early diagnosis depends almost entirely upon auscultation, for percussion is peculiarly deceptive when the chest is eparate; it usually develops at a time (first to sixth month) rickets is most uncommon, and is usually stamped by other itic stigmata, as cutaneous and mucous syphilides; when it es the epiphyses, the femur is usually its seat, and there may be, en the cartilage and bone, a wall-like, very tender, painful and y doughy swelling which also is rather fusiform, compared with under rhachitic nodes; epiphyseolysis with great dislocation of agments, may occur in syphilis, but never in rickets; in doubt- ses an *ex juvantibus* diagnosis may be made. (*c*) *Vertebral ulosis* (*q.v.* and also compression of the cord) is easily dis- shed; the rhachitic kyphosis is arcuate, not angular; other oms of either disease are almost invariably present. (*d*) *Hydro- lus* (*q.v.*, for differentiation).

atment.—1. Hygienic.—Fresh air and sunlight produce wonder- ults particularly in the country, mountains or at the sea-side.

Dietetic.—In nurslings (who seldom develop the disease) or in young babies, the use of a wet-nurse may entirely cure the e. In other children it may be less a question of food than of ion, which may require pepsin, taka-diastase, nux vomica, or hosphites.

Medicinal.—No specific remedy is known, but the results pro- by *phosphorus* make it more important than any other drug. its use the child becomes easier and brighter; the nervous oms abate, as the insomnia, convulsions, tetany, or laryngospasm; seous symptoms are improved, bone is deposited and dentition , though less obviously in clinical experience than in animal ments in which it seems certain that small doses stimulate bone tion directly and rapidly. Phosphorus itself is given in doses $\frac{1}{200}$ to $\frac{1}{100}$ in pill form, or as the ol. phosphoratum (1 per solution) ♏j, t.i.d.; the oil or emulsion should be freshly pre- , for both degenerate readily on exposure to light. (Its toxic

Bretonneau in 1827; it carries out Cheadle's theory that animal and proteids are essential, and comforms with Sutton's experien (*v. s.*). If it is well tolerated by the stomach, the general level of hea may be elevated by its use; if not, cream and butter should be giv *Iron* (syr. ferri iodid.) is a good tonic. Calcium salts are now l used than formerly, as it has been proven that a sufficient quant is present in the food and is absorbed.

4. **Orthopedic.**—During the florid stage the child should be ke quiet upon a firm mattress. He should be kept off his feet. Lat corrective measures are indicated for the kyphosis and deformity the limbs. Mikulicz holds that the apparently monosymptomatic ge valgum adolescentium is only a rhachitis tarda, and that genu valgu and varum should always be treated with rest, cod-liver oil and ph phorus.

(F). OBESITY.

Etiology.—Obesity (adiposity, corpulence or polysarcia adipos is favored (*a*) by *heredity;* it frequently occurs in entire familie congenital cases are known; (*b*) by *mode of life;* lack of exercise, ove eating and alcoholic indulgence, are potent factors. (*c*) The *men pause* is frequently attended by increase in weight. (*d*) *Vario diseases* may cause it, as those of the nervous or osseous syste *making exercise impossible;* and gout, diabetes, various anæmia chronic valvular disease, and emphysema, which act rather by pre venting adequate exercise than by deficient oxygenation or from th condition of the blood. Adipose tissue may develop from tissu albumin.

Symptoms.—The general *habitus* requires no description. Fa develops in the subcutaneous tissues, in parts where it is normally found, as in the omentum and around the kidney, and it may cause fatty infiltration and degeneration of all internal organs excepting the brain and spleen; even the blood may contain fat (lipæmia).

1. **Cardiac Symptoms.**—The heart is embarrassed by increased resistance to the capillary flow, by the fatty infiltration (and degeneration) of the myocardium, and by fat accumulation in the chest wall, omentum and mediastinum. The pulse is more often frequent than slow, though cases of bradycardia are noticed (see Stokes' triad under FATTY HEART). Cardiac hypertrophy may occur. Œdema is not uncommon.

2. **Respiratory Symptoms.**—The breath sounds and heart tones are faint, and thoracic percussion is difficult because of the subcutaneous fat. *Bronchitis* is common and probably results from the ready sweats and consequent chilling. Dyspnœa may occur on exertion only, or may develop from pulmonary œdema.

3. **Digestive Symptoms.**—Digestive disturbances may develop. The large liver is less frequently fatty than congested and hypertrophic. Constipation and hemorrhoids are frequent.

4. The Urine.—The urine may be scant in comparison with the fluids ingested; coincident oxaluria, lipuria, lithuria, albuminuria or glycosuria may be noticed. Actual impotence is not uncommon, as well as mechanical obstacles to coitus.

Two other groups of symptoms mark extreme obesity, *viz.*: (5) *lessened physiological resistance* to infections, as typhoid or pneumonia and to surgical measures or phlebotomy, and (6) such complications or *associated affections* as diabetes, gout, calculous disease and less often contracted kidney, arteriosclerosis, and apoplexy. Somnolence may be marked.

Treatment.—Treatment is (*a*) that of the fundamental condition, as gout or diabetes, and (*b*) *dietetic*. Various "cures" have been formulated which are enumerated because they possess more than historical interest—(i) *Banting's cure.* Banting, a merchant, was treated so successfully by Wm. Harvey for obesity that he addressed a "Letter to the Public on Obesity" (1863). This diet was largely *albuminous*, and consisted of fish, meat, eggs, tea without sugar or milk, vegetables, fruit, some red wine, and toast without butter. This diet may cause indigestion, nervousness, insomnia, palpitation or gout. (ii) *Ebstein*, himself a sufferer, proposed (1872) a diet with larger proportions of *fat;* his diet consisted of meat, fish, eggs, tea, leguminous vegetables, fruit, fats and butter, but restricted the carbohydrates; digestive disorders may also occur under this system. (*c*) *Oertel* allowed *more carbohydrates*, limited the fats and fluids, and insisted upon sweats and graduated exercises. From the following it will be seen that all these methods are "starvation cures," with restriction of water in Oertel's method. The average man requires 118 gm. (4 ℥) of nitrogen, 56 gm. (2℥) of fat, and 500 gm. (17℥) of carbohydrates (Voigt and Pettenkofer), equaling 3,054 calories.

The diet of Banting contains 172 gm. (5.7℥) of nitrogen, 8 gm. (2 ℥) of fat, and 81 gm. (2.7℥) of carbohydrates, equaling 1,112 calories.

The diet of Ebstein contains 102 gm. (2.3℥) of nitrogen, 85 gm. (2 ℥) of fat, and 47 gm. (1.6℥) of carbohydrates, equaling 1,401 calories.

The diet of Oertel contains 183 gm. (6℥) of nitrogen, 38 gm. (1.3℥) of fat, and 143 gm. (4.8℥) of carbohydrates, equaling 1,690 calories. (A calorie is the temperature required to raise 1,000 gm. water, 1° C.; a gram of albumin equals 3.2 calories, of fat 8.4, and of carbohydrates 3.8 calories.)

Banting's method supplies to the body about 33 per cent. of the heat and energy normally required; Ebstein's less than 50 per cent. and Oertel's slightly more than 50 per cent. The lesson taught then is *moderation in eating* (*v.* GOUT). In any reduction-cure the patient may suffer from breaking down of the body proteids, which causes the weakness that is experienced. In all cases the total amount of nitrogen in the urine, which constitutes 90 per cent. of the nitrogen excreted, should be watched; if more is excreted than ingested the nitrogenous movements (*v.* FATTY HEART, MYOCARDITIS AND DILA massage are indicated when active exercise is contra-ind

(*d*) The patient should *sleep not more than seven hour* bathe daily to avoid skin complications and to promote Thyroid extract is not to be taken, except perhaps under s tions, for it breaks down the albumins rather than th excites the heart, sometimes induces glycosuria and is fluous (with proper dietetics and exercise).

(G). ADIPOSIS DOLOROSA OR DERCUM'S DIS

This affection was described by Dercum (1892), as characterized by unequal symmetrical deposits of fatt various parts of the body, preceded or attended by p were only 31 reported cases up to 1904, most of which It usually develops in middle life, chiefly after the fortieth the author has a case under treatment, aged thirty-five ye

Pathology.—Its pathology is not yet developed, as but have been made (1903). In the cases of Dercum and B stitial neuritis was found, which is likewise noted in the which have been removed during life. It is interesting symmetrical lipomatosis has been observed in multiple Dercum's and Burr's cases the thyroid gland was atroph carcinoma and gliosis of the hypophysis have been repor

Symptoms.—*Two cardinal clinical features* are obse term adiposis dolorosa indicates; (1) the *obesity* is eith may be localized fatty tumors, or sometimes both in lipomata occur on the trunk and proximal parts of the but never on the face, forearms, hands, legs, or feet. Ind ness of the wrists and legs contrasts sharply with the m thighs, and upper arms. The fatty tumors range from pea to large dimensions. The pendulous abdominal f myxœdema, save for its circumscribed distribution and facial exemption.

2. The *pains* may be neuralgic or paræsthetic. The trunk is often tender.

Subcutaneous hemorrhages, hæmatemesis, epistaxis and metrorrhagia are not uncommon. The patients are often nervous, perhaps hysterical. Asthenia is common, and subjects often lack initiative and readily tire on exertion. In the later stages epilepsy has developed, and death from asthenia, psychosis, nephritis or intercurrent affections is recorded. A number of cases, including one of the author, have improved under administration of thyroid extract.

SECTION IX.

DISEASES OF THE NERVOUS SYSTEM.

DISEASES OF THE BRAIN.

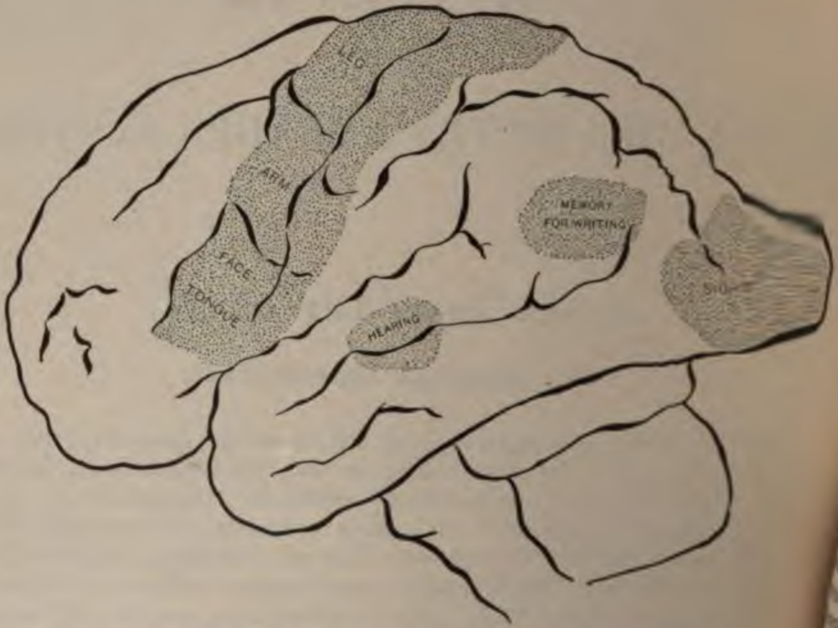
(A). CEREBRAL LOCALIZATION.

The anatomy and physiology of the brain are most important in topical diagnosis. Autopsy findings in carefully studied cases, animal experimentation, and the constantly increasing surgical intervention, have developed not merely the *nature* of lesions, but also important results regarding their *localization*. The old doctrine of Flourens that all parts of the brain were equally important, met its first contradiction in Broca's discovery (1861), that motor aphasia was due to disease of the third left frontal convolution. In 1870, Fritsch and Hitzig discovered that stimulation of certain areas, now known as the motor cortex, invariably resulted in contraction of definite muscles of the opposite side of the body. Since then, Bevor, Horsley, Meynert, Jackson, Flechsig, Ferrier, Munk, Glotz, Nothnagel, Charcot, Wernicke, and others, have contributed to the development of brain localization, although much still remains unclear and tentative.

I. The Motor Cortex.

This embraces chiefly the central convolutions on either side of the fissure of Rolando (the ascending frontal and ascending parietal lobes and anterior two-thirds of the superior parietal lobe), the foot of the three frontal convolutions, especially the lower, and on the inner surface of the hemisphere, the paracentral lobule. In this area lie the large pyramidal cells which Betz discovered were the origin of the motor or pyramidal tracts. The centres are not sharply marked, but run somewhat into adjacent areas, especially those with physiological associations. The *leg centre* occupies the upper quarter of the ascending frontal and ascending parietal convolutions, and part of the superior parietal lobe (all adjacent to the longitudinal fissure), and passes over to the inner aspect of the hemisphere to the paracentral lobule, which is the centre for the thigh, pelvis, and gluteal muscles (Fig. 51). A lesion in the falx cerebri, or dura near the longitudinal fissure may, in rare cases, involve both paracentral leg centres, and produce cerebral paraplegia, although most paraplegias are spinal in origin. The *arm centre* is the largest, covers the middle two quarters of the central convolutions, lies below and probably somewhat overlaps the leg centre. In Fig. 52, it is seen that there are certain general as well as separate centres for the thumb and fingers. The *head centre* is below that of the arm, covering the lowest quarter of the central convolutions (*i.e.*, the entire operculum), the upper part of the Sylvian fissure, and the posterior part of the lowest frontal convolution (and possibly the first and second frontal gyri). The *face centre* lies directly beneath the arm centre, and is mostly located on the anterior ascending convolution, the upper facial component lying higher than the lower facial centres. The eye centre of the facial is bilateral, and probably consists of several foci. The mouth apparently has several centres. The *motor speech*

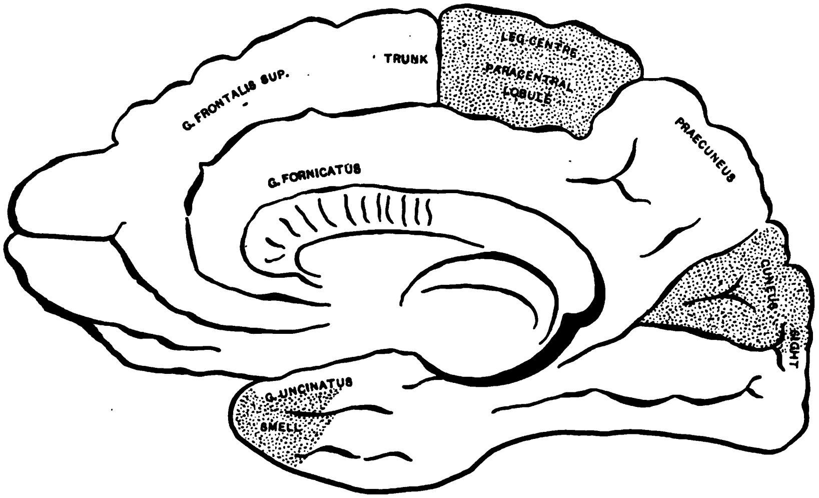
FIG. 50.



General distribution of centres in cerebral localization; outer surface of brain.

area is principally in the lower left frontal gyrus, and is probably the chief centre for chewing. The *trunk centre* is less clearly defined; it probably lies in the posterior part of the first frontal convolution, and also on its median aspect, just anterior to the paracentral lobule. The more complicated the function, the larger is the centre (as for the tongue, fingers, mouth), and the more unilateral the localization in the brain. Muscles used rhythmically, or in pairs, are supplied to some extent from both hemispheres, as the muscles of the forehead, and of respiration, swallowing, chewing, vocal cords, and to some degree, of the legs and trunk. Protrusion of the tongue to one side has a monolateral centre, though its protrusion in the median line and its withdrawal depend on both hemispheres.

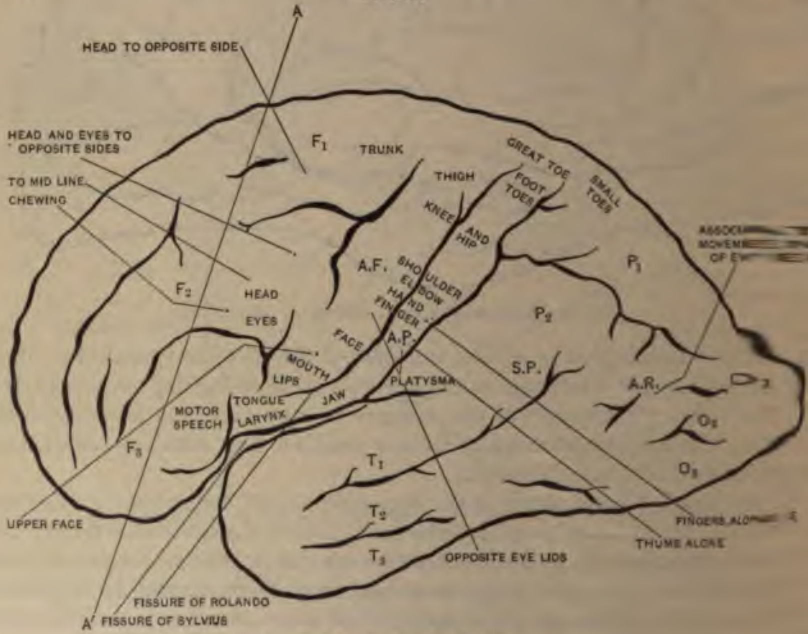
FIG. 51.



Cerebral localisation; inner surface of hemisphere.

Total lasting hemiplegia of the opposite side of the body, with sensory disturbance, aphasia, and intellectual disturbance, results from complete destruction of the entire motor cortex of one side. Cortical hemiplegia is less common than *monoplegia,* which results from partial cortical foci. Monoplegia (paralysis of one member, as of the arm alone), always suggests, but does not absolutely prove, the presence of a cortical lesion. The paralyzed limbs are flaccid, and later undergo contractures. *Pure monoplegias* of cortical origin are rare, although the arm or leg may be paralyzed alone from a small softening, hemorrhage, cyst, or tumor, or from traumatism. Crural (leg) monoplegia is rare (few instances being reported by French writers), and is due largely to vascular lesions in the paracentral lobule (anterior cerebral artery). Some sensory disturbance may be noted. Pure brachial (arm) monoplegia may result from vascular lesions, small tumors or trauma; complex movements as writing are most involved. The finest differentiation or selection, as of the thumb alone, fingers, or wrist, is noted in this form. Sensory disturbance is common (*v.i.* Pure facial monoplegia is unknown, although a faciolingual for with motor aphasia is observed. *Associated monoplegia* is more usu It consists most commonly of paralysis or paresis (partial paralysi of the arm and face (brachiofacial monoplegia, passing under th name, although not strictly a monoplegia). Next in frequency is t arm-and-leg type (brachiocrural) in which two contiguous cent are involved. A leg-and-face type never occurs, because their cent lie too far apart. A lesion involving one cortical area after anot speaks for cortical localization. Cortical paralyses are not attenc by the reaction of degeneration. In cortical lesions with paraly twitchings or convulsive movements are common, and, from t resemblance to epileptic convulsions, are called "cortical, partial *Jacksonian epilepsy"*; 50 per cent. of cases are small cortical tu

Fig. 52.



Cerebral localization in detail.

F_1, F_2, F_3, first, second and third frontal convolutions. AF, ascending frontal convolution and AP, ascending parietal convolution. SP, supramarginal and AR, angular convolution. O_1, O_2, O_3, first, second and third occipital convolutions. T_1, T_2, T_3, first, second and third tem-poral convolutions. The explanation of line AA is given under INTERNAL CAPSULE.

or cysts; the lesion partly destroys the motor cortex (paralysis), and at the same time causes irritation (convulsions) precisely as contractions are produced in animals by electrical stimulation of the cortex; they are mainly (*a*) mechanical, as irritation or increased intracranial tension (rapidly growing tumors), or (*b*) due to vascular affection (hemorrhage, embolism, or inflammation). Stationary lesions rarely produce Jacksonian epilepsy, unless they are located in the cortex. The spasms

lve but one part, as the arm (*monospasm*), which is exclu-
sign of cortical irritation, or they may pass from one centre
. They always commence in one centre *locally*, and extend
l order. If the arm-region is the seat of a tumor, the Jack-
ilepsy flexes the fingers, moves the thumb, then the forearm,
shoulder, and then passes down the cortex region (see Fig. 50
rm, face centres), so that it involves the face, mouth and
e leg. If the lesion is in the face area, the various facial
contract, and the eye deviates, then the next area above the
re participates, and finally the higher leg centre is implicated.
the spasm never skips from the face to the leg centre, but it
the arm first. If the leg area is involved, the spasms pass
ively down to the arm and then to the face, *i.e.*, they never
arm, nor involve the face before the arm is convulsed.

asms are often preceded by a tingling in the part. The con-
are mostly clonic, or clonic broken by tonic spasms, and
ness is very often maintained. Unconsciousness may intervene
fits reach the other side of the body, when the lesion is deep
w the cortex, or when they are very frequent, a hundred or
ly. The opposite side is involved through the commissural
the corpus callosum; "bilateral representation in both hemi-
which saves certain groups of muscles in hemiplegia, neces-
heir involvement in severe spasms" (Gowers). Jacksonian
om regular epilepsy in that no cry is observed, the commence-
ocal and more gradual, and consciousness, if lost, disappears
, not precipitately. The convulsions may precede or accom-
paralysis. The seizure lasts several seconds to a minute or
is followed by some vertigo, nausea, stupor, or disturbance of
if spasms occur without paralysis, paresis in the involved parts
due to transient exhaustion of the cortex. Jacksonian epi-
ts lightest forms may be sensory.

ling *sensory localization* in the cortex, opinions vary, and our
ge is far less exact. Munk holds that the sensory and motor
the same, while Charcot, Nothnagel, and Ferrier hold that
ory areas are separate (*v.i.*). Some *vasomotor disturbance*
n in cortical lesions, and Landois and Eulenberg have demon-
vasomotor centre here.

ces of disease in the motor centres depend on the nature and
the lesion. After total lesions of the central convolutions, the
prove very little in adults; other members improve, to some
least, through assumption of function by the sound side of
; in young individuals, the sound pyramidal tracts in the
hypertrophy to even double their original size. The motor
nning from the pyramidal cells of the cortex to the anterior
the spinal cord, constituting the *upper motor neuron*, undergo
ng degeneration, that is, degeneration in the direction of the
pulse.

II. Cortex of the Parietal Lobes.

Our knowledge of the function of this cortex is less definite than that of the motor cortex. (*a*) Lesions of the *first lobe* (P_1 Fig. 52, the Precuneus in Fig. 51), supplied by the anterior cerebral artery, cause disturbance in motility of the leg, with altered muscle sense, "mind blindness," inability to read (alexia), and according to some authorities, hemianopsia. (*b*) Disease of the *supramarginal convolution* (S.P. Fig. 52), also involving the motor cortex, produces greater disturbance of sensation than when the lesion is confined to the motor centres; muscular sense is disturbed, usually with some motor paralysis or paresis. Three cases are reported in which there was disturbance of muscle sense without paresis. Muscle sense is disturbed in 23 per cent. of all cases of cerebral hemiplegia, though in more than 50 per cent. of cerebral paralyses, no special sensory disturbance is noted. Disturbance in muscle sense probably indicates that a cerebral lesion is located in the cortex, if indirect pressure can be excluded. There may be loss of the stereognostic sense, whereby the patient fails, when his eyes are closed, to recognize the geometric form of solid objects, as a key or ball. Cortical incoördination (ataxia) sometimes results, probably on account of disturbance in the association fibers. It differs from other ataxias (the tabetic and cerebellar forms), in that the muscular strength is usually diminished, and the finer movements are impossible, though the coarser movements may sometimes be carried out. Cortical hemianæsthesia is not frequent. (*c*) Ptosis and paralysis of the orbicularis oculi are said to result from lesions of the *gyrus angularis* (A.R. Fig. 52), and according to Monakow, inability to read (alexia), optic aphasia and "mind blindness." Wernicke correctly diagnosticated softening in this lobe by conjugate deviation of the eyes and head as the only symptom. Disease of the posterior inferior part probably causes crossed amblyopia.

III. The Cortex of the Occipital Lobes.

This is undoubtedly the *visual centre.* Its exact seat is somewhat disputed, but the weight of evidence shows that it is in the cuneus (Fig. 52) and the first occipital lobe. A lesion of the occipital cortex produces (*a*) bilateral *homonymous hemiopia* (hemianopsia), (for details of which and the following see AFFECTIONS OF THE OPTIC NERVE). (*b*) *Hemichromatopsia*, an homonymous color blindness, especially for green and red, in which the color perceptions are confused rather than lost; the lesion in such cases is superficial and anterior; (*c*) *total cortical blindness*, if the lesion is bilateral, or both optic radiations are destroyed. (*d*) "*Mind blindness*" results from a lesion on the anterior outer surface of the *left* occipital region, especially the second occipital lobe or (according to others) from disease of the gyrus angularis.

The visual memory is lost; things actually seen are misinterpreted, and the patient confuses persons and things. (*e*) *Alexia*, inability to read, also called "word blindness," wherein the patient can read but fails to understand the letters or words he sees and reads. His memory for words is lost. It is due to destruction of the subcortical long and short association fibers in the second occipital lobe (von Monakow) and the gyrus angularis; it most commonly follows vascular disease. (*f*) *Optic aphasia* results from foci in the left occipital lobes (and also in the angular and supramarginal convolutions), and consists of misinterpretation of visual impressions, so that the patient miswrites and misnames objects seen, though able to name them when he feels them. (*g*) *Crossed amblyopia* sometimes occurs.

IV. The Cortex of the Temporal Lobes (T_1, T_2, T_3, Fig. 52).

The outer surface of the posterior half of the first (and second) temporal convolution is the *auditory centre;* it is said to be larger on the left side; disease in this cortex produces (*a*) *deafness* in the opposite ear; atrophy of this lobe is encountered in congenital deafness. Deafness is usually transitory, because fibers seemingly pass to both ears from each centre. If this lobe is destroyed on both sides, total deafness results. (*b*) *Conjugate deviation of the eyes* has been observed, and is said to be a reflex from auditory impressions. (*c*) Disease of the first temporal lobe produces "*word deafness,*" also known as "mind deafness"; it is a loss of the comprehension of speech. Somewhat in front of this lobe, it is supposed that musical memories are stored. In the *uncinate gyrus* (Fig. 51) the special sense of smell probably has its centre. Ferrier refers the general sensory centre to this lobe. The gyrus fornicatus (Fig. 51) is said to be the centre for *taste,* and possibly for the *sense of pain.*

V. The Frontal Cortex and Aphasia.

Regarding the frontal lobes anterior to their participation in the motor cortex, we have little definite knowledge. Profound disease may develop without clinical symptoms. They have long been thought to be the residence of (*a*) the *psychical functions,* especially for ethical and abstract thought (Meynert, and Flechsig). "Defect in character" followed the passage of a large crowbar, 1½ inches thick and 3½ feet long, through both frontal hemispheres, in the famous crowbar case of Harlow. Psychical functions probably concern the frontal lobes, although the relative integrity of the entire brain is necessary for them. (*b*) Flechsig considers the frontal lobe the anterior *association centre,* the middle one being the cortex of the island of Reil, and the posterior one the large area posterior to the motor cortex. (*c*) Bruns has observed ataxia similar to the cerebellar type, but this probably results either from pressure transmitted to the cerebellum, or from involvement the trunk centre. (*d*) *The motor speech area lies in the lower fronta convolution.* Its function and relations can be well considered in co nection with *motor* and other varieties of *aphasia*. The history aphasia marks the beginning of cerebral localization, and three Frenc writers have determined its motor centre. Bouillaud, in 1825, referre aphasia to the frontal lobes; Dax, in 1836, narrowed the localizatio to the left frontal region, and the *motor centre* was finally confined the posterior part of the *lower left frontal convolution*, in 1861, b Broca, whose name is given to this convolution. The sensory elemen necessary to all voluntary movements, was unknown until later, whe Meynert and Wernicke discovered the auditory centre in the first tem poral convolution; Wernicke localized "*word deafness*" in the sam convolution of the left side, and Naunyn, and others, emphasized th importance of the *gyrus angularis* and adjacent parts of the *occipit* lobes in cases of disturbance of writing, visual aphasia, etc.

Motor Aphasia.—The *motor speech* centre lies in Broca's lobe, the posterior part of the left third frontal convolution; it controls the motor centres next described and retains the impressions or memories necessary to their exercise. The corresponding lobe on the right side has some function, being the actual centre in left-handed persons. It often assumes the functions of the left speech centres, after disease of the latter, especially in children. Articulate speech also depends upon the integrity of the lower part of the ascending frontal convolution of the motor cortex, in which lie the centres for the mouth, lips, tongue, jaw, and larynx. (*a*) These constitute the *cerebral mechanism* of speech, whose disturbance is designated motor aphasia. Motor fibers run from the cortex through the centrum ovale, internal capsule (lying in front of the pyramidal tracts), crus (inside the pyramidal tracts) to their nuclei in the pons and medulla, whence the peripheral bulbar nerves to the tongue and larynx take their origin. (*b*) These lower tracts constitute the *bulbar mechanism* of speech, disease in the components of which causes abolition of articulation (anarthria), or more often imperfect articulation (dysarthria). The elements of speech are correct in dysarthria, but articulation is imperfect, for the labials, palatals, or linguals are imperfectly delivered, or consonants are ru together. To speak, the individual must have ideas, conceptions To illustrate, a cortical lesion in Broca's convolution, as small as hazel-nut, may produce motor aphasia, which is either absolute partial; the patient may pronounce correctly some few words, p sibly only under emotion or in certain relations, or he may pronou words wrongly formed, although he recognizes his mistakes. He c prehends what is said to him, for the lesion involves no special element. He can read writing and print, especially if educated, be "sight may stimulate images without intervention of the motor ce but he cannot speak voluntarily, nor repeat what is said to hi cannot read aloud; he cannot write (agraphia) as a rule, becau ses cannot pass to the hand centre. He may be able to copy (*v. i.* BLE). The cortical is the usual form of motor aphasia.

subcortical lesion in the centrum ovale may sever the fibers between ca's convolution, and the motor cortex for the tongue, lips, The patient cannot speak, but the speech centre is normal. It is re lesion, and is known as pure motor aphasia (Lichtheim). The ient can write. It involves the commisural fibers by way of the pus callosum to the right side, and usually produces permanent asia, because the possibility of compensation by the right hemiere is precluded, but if the lesion is in the internal capsule, the asia is transient, since compensation through the corpus callosum stablished.

ensory Aphasia.—In speech, as in every voluntary motion, sensa and sensory memories are necessary. The sensory relations of ech are *hearing, sight, taste,* and in the blind, *touch.*

uditory Aphasia.—Hearing is most important, and develops in child long before speech. When a child begins to talk, or when one has lost speech relearns it, the brain cortex, through an association entres, learns to connect certain sounds or words with definite objects onditions. The child hears through the auditory nerves which run m each side to both *auditory centres* in the *first temporal convolution* either side. He hears mechanically, just as we may hear without derstanding a foreign language, and learns to associate the sounds h objects, conditions or ideas, for which an *auditory speech centre* the *left first temporal convolution* (in right-handed persons) is develed. Here sounds coming from the two centres of hearing, are nembered, associated, and controlled. Remembering a sound he s heard, but perhaps has never spoken, the child calls into play oca's convolution, the cortical centres for the lips, tongue, etc., and aks. Hearing is necessary to articulate speech, unless, as in deaf ites, education replaces, by sight and touch, the associations usually quired by hearing. Destruction of the auditory speech centre proundly disturbs speech. The patient then hears by the auditory rves and centres, or reads by the visual apparatus, but does not derstand what he hears or reads; this is known as *sensory (auditory) hasia*, or *word deafness.* It is rarely absolute, and recovery is more mmon than in motor aphasia. The subject in speaking transposes rds (paraphasia) but does not realize his mistakes, an important tinction from motor aphasia. Often he later becomes speechless. cannot repeat words nor write from dictation. Voluntary revival words is lost (Gowers). Voluntary writing is impossible, but copyof words is sometimes maintained.

ubcortical auditory aphasia, or pure word deafness (the rarer form), ults from disease *between* the auditory speech centres and the two tres for hearing. It must therefore be below the cortex in the white tter, hence called subcortical. Auditory impressions cannot reach auditory speech centres of the patient, who cannot understand it is said, cannot repeat or write from dictation. As the auditory speech centre is normal, memory for words is retained, and the patient can read, speak, and write.

"*Amnestic*" *aphasia* may be a simple difficulty in recalling certain words in speaking or writing, or it may be very pronounced (word deafness). It is also poorly called conduction aphasia (Wernicke) and is due to interruption, probably in the island of Reil, of the fibers between the motor and sensory speech centres. Gowers states that it may result from partial recovery from motor aphasia, or from sensory aphasia.

Visual Aphasia.—*Sight* is very important in sensory speech. The child who has learned sounds (words) and is able to speak them, reads by associating the sounds heard and spoken, with words, letters, or symbols seen by him. Memory of these *visual concepts* is stored in the *supramarginal* and *angular gyri*. Reading music, etc., is learned in the same way. While in most individuals mental processes are carried on by the memory of words, in rare instances, visual memory may be more important. Destruction of the *angular* and *supramarginal* convolutions by causing a loss of the *visual memories*, produces *alexia*, word blindness, an inability to read. In some cases, the patient can read, but without understanding. Isolated alexia is always due to lesions in these convolutions. He usually cannot write (agraphia) and cannot copy, but can speak fairly well and understand what is spoken. If the optic centre in the occipital lobes is intact, he cannot name objects after seeing them, since the visual memory centre is destroyed, but may recognize a watch by hearing it tick or by feeling it. Lesions just below the visual memory centre, involving the afferent fibers, produce *pure subcortical word blindness, or alexia,* that is, inability to read, with hemianopsia and preservation of voluntary speech and writing. Word deafness and word blindness often co-exist. Sixteen cases of congenital word blindness were collected by Stephanson (1904).

Pure motor and sensory aphasia is less common than complicated forms. The diagrams often employed to illustrate the mechanism of aphasia are open to the objection that they are hypothetical, and do not cover all cases. The anatomical lesions, mostly arterial, are largely the result of softening, the entire sphere of speech or language being supplied by the artery of the fossa of Sylvius. Motor aphasia is likely to occur with disease of the motor cortex or cerebral pyramidal tracts, and the sensory form with disease of the optic fibers. Hemorrhage, trauma, and in children, tuberculosis of the brain or meninges, are also causative. Lasting aphasia in children is usually due to mental causes, since in early life the right frontal area usually takes up the function of Broca's convolution. Congenital aphasia may follow bilateral meningeal hemorrhage. Aphasia also occurs in idiocy in adults, in melancholia, migraine, convulsions, and is especially apt to occur from functional causes in children, as from fever, fright, worms, etc. In adults, the outlook is less favorable than in children. Partial recovery is possible in cases of hemiplegia, and sometimes with care, education may yield fair results. The following table (on page 52) is borrowed from Archibald Church's excellent work on *Nervous iseases.*

VI. Centrum Ovale.

Lesions are usually hard to diagnosticate, and focal symptoms may e entirely absent, if a lesion occur between the associative, commissural, motor, or sensory fibers (corona radiata). When present, sympoms are usually those of (*a*) a cortical, or (*b*) an internal capsule lesion. lesion must be (*a*) very near the cortex to produce either Jacksonian r general convulsions. A monoplegia and subcortical aphasia may ccur in this location. Sensory disturbances, as hemianopsia, may be noted in a subcortical disease. Aphasia from a lesion in the island of Reil may produce the so-called conduction aphasia. Psychical symptoms may result from interruption near the centre of the association fibers which run from one part to another of the same hemisphere. (Commissural fibers are those running from one hemisphere to the other, as through the corpus callosum, disease of which presents no pathognomonic sign, according to most neurologists.) (*b*) Internal capsule symptoms result in lesions low in the centrum ovale. Hemianæsthesia and sensory symptoms in general indicate lesions of the internal capsule.

VII. The Internal Capsule.

It is compacted and consists of nearly all the motor, sensory, and special sense fibers from the brain to the lower parts. It has an extensive vascular supply. The capsule is divided into an anterior limb, the knee and a posterior limb.

Anterior Limb.—Its function is not clear, although it contains fibers from the frontal cortex to the optic thalamus. Lesions here are very rare. It also contains fibers which, in lower horizontal sections, run into the knee and are found in basal median bundle of the crus, whence they run to the pons nuclei for the eye, head, neck, and probably also for the larynx, tongue, and mouth.

Knee.—The knee (see Fig. 53) contains from before backward fibers for opening of the eyes, associated movements of the eyes and head, the tongue and mouth; Horsley and Beevor have been able to stimulate them electrically, one by one. This segment contains the phonation fibers for speech, probably including those of the larynx.

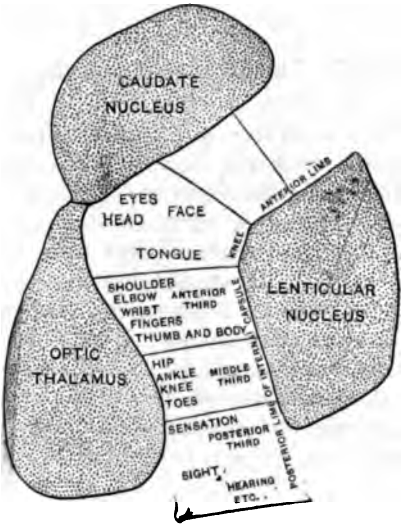
Posterior Limb.—The posterior limb contains in its anterior third, the fibers for the shoulder, elbow, wrist, fingers, thumb, and body; in its middle third those for the hip, ankle, knee, and toes; in its posterior third (Charcot's "sensory crossway"), those for sensation and the special senses. The number of fibers to a part depends less on the size of a muscle than on its development in fineness of movement; for instance, the fingers receive a relatively large number of fibers. If a

TABLE OF SIMPLE APHASIAS. (Church.)

VARIETY.	LESION LOCATED.	PRINCIPAL FEATURE.	UNDERSTANDS SPEECH.		EXPRESSES SPEECH.		REPEATS DICTATION.	COPIES WRITING.	WRITES FROM DICTATION.	INCIDENTAL CONDITIONS.
			Spoken.	Written.	Spoken.	Written.				
AUDITORY APHASIA.	Posterior thirds of the first and second left temporal gyri.	Word-deafness.	No.	Yes.	Paraphasia—jargon.	Paragraphia or agraphia.	No.	Yes.	No.	Amusia.
MOTOR APHASIA.	Foot of third left frontal gyre.	Dumbness.	Yes.	Partly.	Articulation impossible.	Exceptionally.	No.	Yes.	May do so.	Amimia or paramimia.
VISUAL APHASIA.	Angular gyre of left parietal lobe.	Word-blindness.	Yes.	No.	Yes.	No.	Yes.	Yes.	Partly, but can not then read it.	Homonymous hemianopsia, when the lesion is in the optic radiation; mind blindness, if the lesion is bilateral.
CONDUCTION APHASIA (Wernicke).	Island of Reil usually; must cut off connecting tract between auditory and motor word memory centres.	Paraphasia. Paragraphia.	Yes.	Yes.	Paraphasia.	Paragraphia.	Paraphasia.	Yes.	Paragraphia.	Mental confusion.

uler be placed anterior to the line (AA) in Fig. 52, and slowly moved o the right, preserving its parallelism, it will be seen that the cortical motor areas touched will approximately correspond with their order in the internal capsule. The most common sequence of lesions of the internal capsule is *cerebral hemiplegia* of the opposite side of the body. Though hemiplegia may result from lesions elsewhere in the motor tract (cortex, centrum ovale, crus, pons, or medulla), in the vast majority of cases, hemiplegia is the result of disease of the internal capsule. If the internal capsule is destroyed, *total hemiplegia* results, and paralysis of the arm, leg, and lower part of the face (the upper part of which, and the head escape, because they have a bilateral supply from both hemispheres). The hypoglossus is but moderately involved as a rule, and aphasia is usually temporary. With the hemiplegia are also associated hemianæsthesia, hemianopsia, and unilateral disturbance of smell, taste, and hearing, by lesion of the fibers ascending from the tegmentum; with the hemiplegia are observed contractures, descending degeneration, decrease or absence of the skin reflexes, especially the cremasteric, and increase of the tendon reflexes. Monoplegia is rare, because the tracts are so compact that the face, for example, is rarely alone involved, though Duplay and Diday record isolated cases. It is possible that a *linear* lesion may involve the outer part of the capsule's knee, that is, it may involve the face and also the leg, leaving the arm free (Wernicke, Charcot, Kolisko). Hemianæsthesia (skin sensation and muscle sense), rarely exist without some paresis of the leg. Some tingling or, in incomplete lesions, some pain may result, even when the leg-arm areas are alone involved, for some sen-

Fig. 53.



Localization in internal capsule.

sory fibers course with the motor fibers. Hemi-tremor, -athetosis and -chorea result from lesions of the sensory area, usually associated with disease in or just below the optic thalamus. (The course of the sensory paths will be considered under LOCALIZATION IN THE SPINAL CORD.) Back of the general sensory tracts, disease may injure the optic radiation (running between the occipital sight centre and the lateral geniculate body), or interrupt the auditory and other special sense fibers. Psychical disturbance, aphasia, alexia, agraphia, and permanent involvement of any cranial nerve are not symptomatic of disease of the internal capsule.

VIII. The Corpus Striatum.

This is composed of the caudate and lenticular nuclei. It is doubtful whether it is connected with the cortex. The lenticular nucleus is extensively connected with the superior peduncle of the cerebellum and the tegmentum of the crus. The caudate nucleus is connected with the internal capsule, and with the crus; these fibers seem to end in the pons and cerebellum. An apoplexy in the corpus striatum probably causes rise of temperature; Nothnagel's law still holds that hemiplegia is permanent only when the internal capsule is directly injured, although indirect transient hemiplegia, from simple pressure on the capsule, may result from disease of the corpus striatum. Landouzy reports a case of contralateral hemiathetosis from isolated disease of the lenticular nucleus.

IX. The Optic Thalamus.

This is surrounded by gray matter, continuous with that surrounding the third ventricle and central canal of the cord, and is connected by fibers with all parts of the cerebral cortex, the tegmentum, and superior cerebellar peduncle. Nothnagel (1879) was the first to describe its topical symptomatology. Sometimes disease in this locality is latent; again, the following findings are observed, alone or in combination: (*a*) *Contralateral hemianopsia* may develop from a lesion in the posterior part of the thalamus, the pulvinar (Gowers), or a lesion which also invades the lateral geniculate bodies (von Monakow). These areas are connected by the optic radiation with the optic centre. This hemianopsia is distinguished from that caused by a cortical lesion by the hemianopsic pupillary reaction and the absence of the marginal field of vision. (*b*) *Disturbance of the mimetic movements of expression* may develop (Nothnagel, Bechterew). The optic thalamus is the reflex centre of the involuntary movements for expression, as weeping or laughing. A patient with hemiplegia from destruction of the left *internal capsule* cannot voluntarily move the lower right part of the face, but the paralyzed portion of the face moves when the patient laughs, for the centre is in the posterior part of the thalamus. On the other hand, in isolated disease of the optic thalamus, the opposite side—indeed both sides of the face,—can be voluntarily moved, since the facial nerve runs in the internal capsule, but the patient cannot laugh or weep. (c) *Sensory disturbances* may occur, such as hemianæsthesia, hemichorea, hemitremor, pain in the opposite arm (Edinger), or hemiataxia, but it is not absolutely certain in some of these cases that the sensory portion of the internal capsule was not involved. There may be vasomotor changes, atrophy, and disturbance in the evacuation of the bladder and rectum. Von Monakow thinks that all the symptoms referred to the thalamus are probably referable to disease exterior to it.

X. The Corpora Quadrigemina.

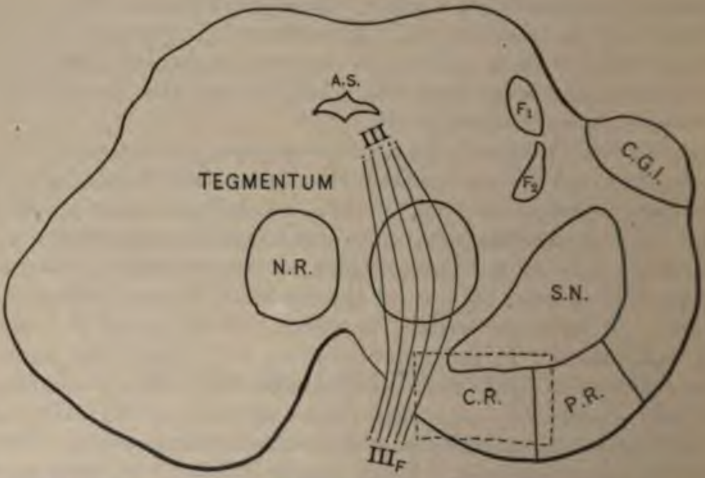
These are masses of gray matter, whose fibers lie over the aqueduct of Sylvius and the tegmentum. There is much variance of opinion regarding their function. They are actually connected with the third nerve nucleus, and some hold that they contain the centres for contraction of the heart and stomach.

Disease in the *anterior corpora quadrigemina* produces (*a*) *moderate involvement of sight.* Some writers claim that total blindness or hemianopsia may result, but most of these cases had tumors with optic neuritis; slight disturbance has a greater localizing significance if there is no coincident neuritis. These bodies are connected with the external geniculate bodies and, by way of the internal capsule, with the optic radiation. (*b*) In acute lesions, narrowing of the *pupil* is suggestive of this localization. In old lesions, one or both pupils are wide, and without reaction to light or accommodation. (*c*) *The eye muscles* may be partially paralyzed, especially those supplied by the third nerve; this results in ptosis, and upward movements of the eyes. The disturbance is not associated. Much doubt is felt in considering these symptoms indicative of this localization, because the lesion is always so near the nucleus of the third nerve. Disease in the *posterior corpora quadrigemina* may result (*a*) in *ataxia,* according to Nothnagel, though others refer it to actual cerebellar disease, to connections with the cerebellum through the nucleus ruber, or to disease in the fillet of the tegmentum; (*b*) in *trochlearis paralysis;* in which the eyes cannot be turned inward or outward; sometimes it results in abducens paralysis; (*c*) in *difficult mastication* from involvement of the descending branch of the quintus; (*d*) in contralateral *disturbance of hearing,* since the posterior corpora quadrigemina are connected with the fillet of tegmentum and, by means of the "sensory crossway" of the internal capsule, with the hearing centre in the first temporal lobe

XI. The Crus (Cerebral Peduncle).

Focal symptoms may be divided into motor and sensory symptoms. The *motor* fibers to the cranial nerves decussate higher than the pyramidal tracts which decussate in the medulla; the fibers to the third nerve cross in the upper crus, although some enter the nucleus of the same side. *Crossed paralysis* (hemiplegia alternans oculomotoria superior) is common in crus lesions. It consists of a *hemiplegia of the side of the body opposite to the lesion*, and a *paralysis of the third nerve on the same side* as the lesion, often known as Weber's paralysis, though it was first described by Gendrin. From Fig. 54, it is seen that a lesion involving the right pyramidal fibers may also catch those of the third nerve on the same side, giving a left-sided hemiplegia (face, arm, and leg), and right-sided oculomotor paralysis, which is peripheral and usually complete, with strabismus, ptosis, inability to look up or down, and a wide reactionless pupil. The crossed or alternating hemiplegia is called superior to designate its location, because inferior alternating hemiplegia may also occur in lesions of the pons. One must be sure that one lesion only exists. In tumors near the internal capsule which press on the third nerve, the same symptom complex may occur, and is recognized by noting whether the hemiplegia and paralysis of the third nerve occurred together (indicating a crus lesion) or whether the third nerve paralysis followed the hemiplegia (indicating a lesion near the internal capsule pressing secondarily on the oculomotorius).

Fig. 54.



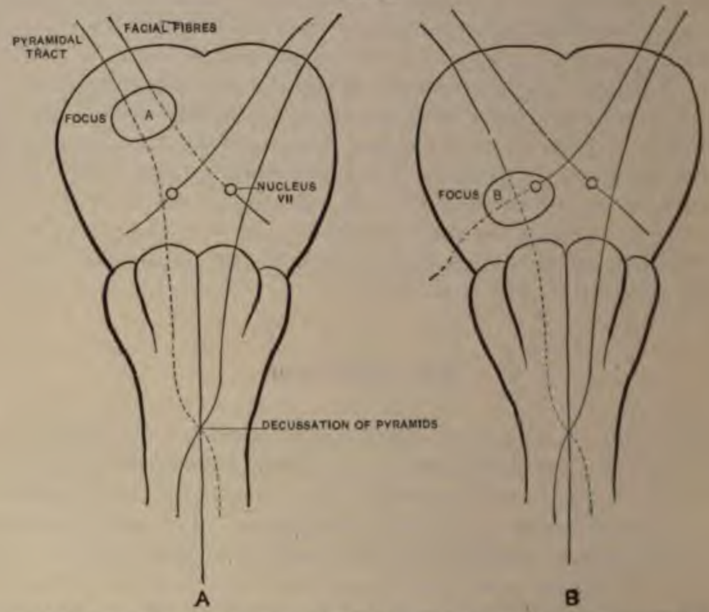
Lesion in crus (shown by dotted lines), producing a superior alternating or crossed hemiplegia; the lesion involves the pyramidal tract (PR), the cranial nerves (tongue and face, CR) and the ouclomotor nerve (III, F) which is paralyzed on the same side as the lesion while PR and CR are paralyzed on the opposite side. AS–Aqueduct of Sylvius; III–nucleus of third nerve; CGI–Corp. geniculatum int.; F_1–upper part and F_2–lower part of fillet; NR–nucleus ruber; SN–Substantia nigra.

A lesion in the tegmentum of the crus may cause (*a*) *sensory disturbnce*, as hemianæsthesia, which involves skin sensation and muscle ense, and of which over thirty cases are recorded; such a lesion is in he fillet; hypæsthesia (reduced sensation) is more common. (*b*) The *pecial senses* are sometimes involved, producing hemianopsia, or poor earing in the opposite ear. (*c*) *Ataxia* has been seen alone (6 cases), r together with other symptoms. It differs from the cerebellar type n that it is manifest only on motion. Involvement of the red nucleus which is connected with the cerebellum or with its arm), produces erebellar ataxia. (*d*) *Oculomotor paresis* on the side of lesion is charcteristic when combined with crossed hemiataxia, hemiplegia, heminæsthesia, or possibly with tremor, low temperature, and vasomotor isorder. Fig. 54 shows how a tegmental focus can rarely miss the iffuse nuclei of the third nerve. If the lesion is in the lower part of he crus, the oculomotor paralysis is usually complete, but if in the egmentum, it is always partial, selecting the pupil, the lid, or the uperior or internal rectus muscles.

XII. The Pons.

Lesions of the pons are most commonly tumors, hemorrhage, softeng, or abscess. Its structures are so closely arranged that sometimes th sides are implicated, and this may produce bilateral hemiplegia, ough a small lesion, as a long thin tumor, may give no localizing mptoms; most diseases of the pons produce a typical picture. From lesion of the *upper pons, i. e.*, the portion above the facial nucleus, a emiplegia results like that produced by lesions of the internal capsule, hich affects the face, arm, and leg, all on the same side of the body. ee Fig. 55, focus A.) The facial paralysis is supranuclear, partial, nd with no reaction of degeneration. *In a focus in the lower pons* ocus B, Fig. 55), where the facial fibers have already crossed, e tongue, arm, and leg are hemiplegic on the side opposite to the cus, and the facial paralysis is on the side of the lesion; this is e crossed or alternating paralysis (hemiplegia alternans facialis), hich was first described by Millard (1856), and then by Gubler 1859). The facial paralysis is peripheral and nuclear, involves all anches, and gives the reaction of degeneration. Sometimes there also some slight paralysis of the face on the hemiplegic side from nvolvement of some fibers from the cortex. Other alternating paralyses are also observed, as *hemiplegia with crossed paralysis of the hird, fourth, fifth, and sixth nerves*, the paralysis of which is usually oth central (nuclear) and peripheral. Hemiplegia may occur with onjugate paralysis or paresis of the lateral eye muscles, as the external rectus (abducens nerve) of one side, and the internal rectus (third nerve) of the other, due to a lesion just in front of the abducens centre, involving its fibers, or sometimes also including its nucleus. In the conjugate paralysis, the antagonist muscles pull the eyes to the other side, and the *eyes look away from the side of the lesion,* toward the paralyzed side. This is not to be confused with the "forced position" of the eyes due to irritation, known as conjugate deviation, in which the eyes look to the sound side of the body. In this form even the paralyzed internal rectus will functionate and converge when one eye is tested at a time. In some cases both internal recti and one abducens may be paralyzed, resulting in fixture of the eye in the median position; accommodation and convergence are preserved. Involvement of the *motor branch* of the fifth nerve produces difficult mastication. While alternating paralysis is the usual pons lesion, sometimes double hemiplegia occurs, or again, involvement of the third to seventh cranial nerves without an hemiplegia. The *auditory nerve* is rarely involved, save in acute or very extensive lesions affecting the lateral lemniscus. Implication of the cortical fibers of the hypoglossus produces impaired movement of the tongue (dysarthria), nasal speech, and poor enunciation of consonants, especially linguals and labials, though the vocal cords are normal. *Dysarthria and dysphasia,* due to disease of the cortical motor paths of the hypoglossus and vagus, are less common in lesions of the pons than are affections of their nuclei in diseases of the medulla oblongata. *Ataxia* may result from involvement of the fillet, and possibly of the longitudinal bundle of the formatio reticularis, and produces incoördination on voluntary movement only. *An alternating sensory*

Fig. 55.



A illustrates a lesion in the upper pons, producing the usual hemiplegia (face, arm and leg paralyzed on the opposite side). B illustrates a lesion in the lower pons, producing alternating paralysis; all branches of the face (peripheral paralysis) and tongue on the side of lesion are paralyzed with paralysis of the contralateral arm and leg.

ralysis may occur, as (*a*) hemianæsthesia involving the muscle sense, d cutaneous sensation of the extremities opposite the lesion, caused disease of the tegmentum, and (*b*) an anæsthesia of the fifth nerve the side of the lesion. *Irritative* symptoms sometimes develop, as in, paræsthesia, painful convulsions, which usually occur in acute ses, and which are peculiar in their involvement of both arms or th legs, tonic and clonic spasms in the opposite arm and leg, con-ctures, paroxysms of coughing, and trismus. The patient may ook toward the lesion" when the eye muscles are irritated. A dency to fall toward the side on which the lesion is located, is indic-ve of disease of the middle peduncle of the cerebellum. It is some-es difficult to decide whether the focus is located in the *pons or in medulla.* Though the topical symptoms of the medulla oblongata described under its individual diseases, it may be stated that *an rnating or crossed paralysis*, involving the third to seventh nerves lusive, *indicates disease in the pons*, and that *aphonia, dysarthria, l dysphagia* are symptomatic of *localization in the medulla*, especially en the hypoglossus is paralyzed, shows the reaction of degeneration, l produces atrophy of the tongue. Glycosuria, albuminuria, dis-bed respiration or heart action (by involvement of the vagus), and miting are other bulbar symptoms. Disease in the medulla may se paralysis of both arms and legs, or rarely of one arm and the posite leg (this latter is known as hemiplegia cruciata).

XIII. The Cerebellum.

Physiology—The functions of this organ are not fully known. rtain physiologists admit the possibility of some psychical function. s *sensory relations* are important, because of its reception of numer-s fibers from the spinal cord, including the direct cerebellar tract and e posterior median column of Goll. It is an "end organ" (Luciani) r these tracts, through which and its relations to the motor tracts nd the special senses, it is supposed to coördinate and *harmonize e movements of the body, to maintain equilibrium, conserve muscular rength* and *inhibit the contralateral cerebral motor cortex.* In other ords, it associates (*a*) the sensory impressions ascending from the spine ith (*b*) those gained by hearing (the auditory nerve having connections ith the middle cerebellar lobe), and (*c*) probably with those impressions resulting from the eye movements.

General Pathology.—Disease of a lateral lobe affects the same side the body, but lesions of the middle (vermiform) lobe influence both des. Results of extirpation in animals vary somewhat, and will be nsidered only from their clinical aspect. Krauss found, in 100 cases cerebellar disease, tumors in nearly 80 per cent., abscess in 10 per ent., cysts in 7 per cent., and softening and hemorrhage constituted he balance. Agenesis, sclerosis, and gliomatosis are also occasionally ound. Disease of one hemisphere, or indeed extensive lesions of both hemispheres may be fully compensated by the cerebrum. Acute disease of the cerebellum produces symptoms more frequently than does chronic disease, in which latter the cerebellum has opportunity for accommodative compensation. Rapidly growing tumors, abscess, and sometimes acute vascular disease often produce the following symptoms, in part typical, and in part secondary (general).

Typical Symptoms.—1. *Cerebellar ataxia* is almost always present, and occurs when the patient stands; equilibrium is disturbed, and the body sways; this is known as static ataxia. Ataxia also occurs when the patient moves (dynamic ataxia), and concerns especially the trunk and legs. The incoördination is due to disease of the middle lobe or vermis, and the consequent withdrawal from the cerebrum of the cerebellar coördination. The gait is that of an inebriate (*démarche d'ivresse*); the body sways and the individual stumbles a zig-zag course, and sometimes toward the side of the lesion. The arm and hand movements are usually normal. Ataxia may also result from lesions of the nerves, spine or brain (cerebellum, cerebral cortex, internal capsule and probably tegmentum). In the ataxia resulting from other cerebral localization, the incoördination affects the finer movements, is associated with disturbed muscle and stereognostic sense, and very often with paresis. Bouillaud first described cerebellar ataxia, and differentiated it from the tabetic type. The essential differences are (*a*) that in the cerebellar form the ataxia disappears when the patient is lying down, and (*b*) that closing the eyes does not increase it (indeed, it may often disappear).

2. *Vertigo* is very frequent, and usually occurs rather when the patient sits than when he lies down. He thinks that objects are turning around him, or he may feel himself being turned. The vertigo is either constant or paroxysmal, and is due to lesion of the vestibular nerve which connects the semilunar canals with the cerebellum by means of the corpus restiforme.

Secondary Symptoms.—The following secondary symptoms are important but less significant than ataxia and vertigo: (*a*) *Choked disk* is rarely absent in cerebellar tumors, and *amaurosis* often results from increased pressure on the third ventricle, leading to hydrocephalus internus, and then to pressure on the chiasm or optic nerves. (*b*) "*Forced*" *postures* and movements are generally considered due to disease of the middle peduncle (*crus cerebelli ad pontem*). Forced postures of the head and trunk are observed; one eye looks upward and inward, while the other is directed downward and outward. Forced movements consist of the turning of the patient constantly to one side, movement in a circle and a tendency to fall toward the side of lesion and sometimes backward. (*c*) *Muscular weakness* (asthenia) is frequent in animals (Luciani), and is occasional in man. A hemiparesis occurs on the side of lesion. The patient may not be able to rotate the trunk, to straighten the body after bending, or to get up after falling. (*d*) The *patellar reflexes* are usually normal or increased. Sometimes they are lost from pressure on distant structures, or from spinal diseas

Vomiting and *pain* in the head or nape of the neck are frequent of some localizing value. (*f*) Occasionally the following symptoms elop; nystagmus, rhythmical contractions of the head, convulsions, alysis of the cerebral nerves from pressure on the midbrain, dis- ›ed hearing, intention tremor, muscular rigidity, irritability, weak nory, and stupor.

XIV. Medulla (See Diseases of Medulla Oblongata).

(B). CIRCULATORY DISEASES OF THE BRAIN.

I. Anæmia of the Brain.

'his term has been much confused and abused. The symptoma- gy of anæmia and hyperæmia is so similar that differentiation is ıetimes impossible.

tiology.—(*a*) General systemic anæmia may result from chronic :mia, cachexia, and hemorrhages of medical, surgical or obstetrical ıre. (*b*) *Decreased blood supply,* due to cardiac insufficiency, valvular ıosis, stenosis of the vessels by aneurysm or tumor, and puncture ›leurisy or ascites leading to paresis of the thoracic or abdominal ıels may be causal. It may also follow the evacuation of an enor- ısly distended bladder or colon. Sometimes fasting seems to be a se in individuals who faint when the stomach is long empty. (*c*) *pillary compression* by hydrocephalus or tumors, but especially by ebral hemorrhage, may produce anæmia. Sometimes venous hyper- ia may act in the same way. (*d*) *Local causes* in the vessels will considered under softening. Ligature or compression of one carotid duces symptoms only when the other vessel is atheromatous. rhardt induced convulsions by pressure on one carotid, and con- ered it indicative of cerebral atheroma of the other cerebral vessels.

'athology.—The meninges, the cortex, and the white substance are e, the brain is often firm and dry, and fewer *puncta vasculosa* are ı. The fluid in the ventricles is often increased. Geigel terms the v capillary flow, "diämorrhysis."

ymptoms.—Local anæmia of the brain leads to imperfect function, ı loss of function, and finally, to necrosis, as in embolism or throm- is (*q.v.*). General anæmia may be sudden or gradual in onset. *sudden anæmia,* as after hemorrhage or in fainting, the subject omes drowsy, syncope occurs, and after return to consciousness, ıt is impaired. Amaurosis is also frequent without syncope. There inging in the ears, and the pupils at first contract from irritation of third nerve, and later dilate from its paralysis; this is a sign of ıger. The skin is pale and clammy, the muscles are lax, and nausea requent. Epileptiform convulsions often occur when much blood ›st, and are probably due to irritative anæmia of the cortex, or possibly of the pons or medulla. Anæmia of the medulla causes the slow, sighing respiration, and the frequent yawning. Death may follow syncope, and was called nervous apoplexy by the old writers. In *gradually developing anæmia,* as pernicious anæmia, the brain power is lessened, and irritability, delirium, insomnia, headache, vertigo, weak muscles, spots before the eyes, impaired sight, ringing in the ears, and sometimes deafness, may result. These symptoms are aggravated by sitting or standing, and are relieved on lying down. Palpitation and thoracic oppression are common symptoms. The pulse varies in rate and rhythm. Increased reflexes, paræsthesia, and jerking of the muscles are frequent. Some mental symptoms usually remain after recovery.

Marshall Hall has described *hydrencephaloid,* a variety of cerebral anæmia, which is seen in children suffering with acute diarrhœa, and is characterized by contracted and later dilated pupils, depressed fontanelles, sunken face, pallor, early irritation followed by stupor or even coma. It may resemble meningitis from the strabismus, rigidity of the neck, and convulsions. It is usually amenable to treatment.

Treatment.—Treatment, as well as diagnosis and prognosis, is chiefly that of the cause. The *anæmia* is treated by putting the patient in the recumbent posture, and instituting rest in bed, iron, arsenic, a full diet, and sometimes small doses of opiates. Acute anæmia necessitates absolute rest, free stimulation, coffee by rectum, faradization of the chest and abdomen, and enemata or subcutaneous infusions of salt solution. Firm bandaging of the extremities, sometimes called autotransfusion, is valuable. Rest and cardiac stimulation are indicated in cardiac cases. For *hydrencephaloid,* warmth, stimulation and fluids are necessary. Nitroglycerine is valuable in certain cases, which are possibly due to vasomotor spasm.

II. Hyperæmia of the Brain.

Congestion of the brain is an obscure lesion. Andral (1836) described 8 varieties; and when Trousseau, in 1861, attempted to refer some varieties to epilepsy and Ménière's disease, he drew down upon him the wrath of the entire Academy. The hyperæmia which is described in nephritis is usually uræmic, and the collateral arterial fluxion once attributed to erysipelas is entirely toxæmic. Geigel has proposed the term "endiämorrhysis" for the normal flushing of the brain by its capillaries, which implies a normal metabolism and exchange of gases. The essential element is the rapidity of the capillary flow. When the vessels dilate from paralysis of the sympathetic system, the rapidity decreases, whence there is not hyperæmia of the brain, but rather a lessened capillary flow, *adiämorrhesis.* On the other hand, when the vessels contract from sympathetic irritation, the brain is not anæmic, but on the contrary, the capillary flow is accelerated, *hyperdiämorrhysis.* General arterial tension is not important, but the tension of the vessel wall is the essential element (Geigel).

Etiology.—Hyperæmia may result from active or passive congestion, though many doubts are entertained in regard to the older conception. The prevailing description may be adhered to, provisionally.

Active congestion results (*a*) from cardiac overaction; (*b*) from overfilling of the brain vessels, when arterial blood cannot perfectly reach the lower parts, as in stenosis of the isthmus of the aorta; (*c*) from contraction of the arterioles in other parts; (*d*) from dilatation of the brain vessels by alcohol, nitroglycerine, coffee, exophthalmic goitre, excessive brain work, neurotic states, and insolation. (*e*) It may occur as the first stage of inflammation. (*f*) In plethoric men, with florid faces, thick neck, and short thorax, active congestion may cause certain passing brain symptoms.

Passive congestion results (*a*) from general mechanical venous stasis, as in valvular heart lesions; and (*b*) from local venous stasis, as sinus thrombosis; according to Geigel's conception, it is held that venous stasis leads to capillary anæmia.

Pathology.—The pathological findings are few, for congestion disappears after death; the brain may appear anæmic after death by strangulation (Ackermann and Jolly), and the head may become congested after death, because of its posture alone. The sinuses and veins are usually turgid and sometimes varicose, and the color of the brain is darker than normal. Microscopic examination shows distention of the capillaries, at times increased blood pigment, or actual rupture into the perivascular lymphatic sheaths, or into the brain substance.

Symptoms.—No symptoms are characteristic. In *active congestion*, the symptoms vary with the cause; chronic cause fewer symptoms than acute factors. Whether toxæmic symptoms of the acute infections are due to hyperæmia, is an open question; the headache in early typhoid has often responded to ergot in personal experience, and this may possibly be used as an argument. The retina has seldom been examined in such cases. Nothnagel and Gowers report cases with paroxysmal throbbing of the vessels, flushing of the face, headache, delirium, sometimes nausea and fever, which are relieved by nosebleed and venesection; some describe similar symptoms from suppression of the menses, or of bleeding piles. Irritability, insomnia, spots before the eyes, ringing in the ears, and increased pulse are also said to occur. In progressive paralysis, the transient pyrexia, heat in the head, or coma, are referred to congestion of the brain. In middle and advanced life, the so-called congestive apoplexy has been noted, which consists of coma, sometimes with transient hemiplegia, though many cases are probably due to small hemorrhages, softening or disease of the vessels.

Passive congestion, if gradual in onset, may be well tolerated, or may produce stupor, dulness, or even delirium. If intermittent, as from coughing, the symptoms are a sense of fulness in the head, headache, and sometimes convulsions; these are increased by exertion, by the patient lying in the dorsal decubitus, and by constipation.

Diagnosis.—Focal symptoms, as hemiplegia, indicate orga
ease. The mere existence of such symptoms, as flushing, l
pressure in the head, cannot be used for diagnosis, because t
often due to neurasthenia, or to hypochondriasis. Marie and
have never made the diagnosis of brain hyperæmia. The grea
arity between the symptoms of anæmia and hyperæmia of th
has been noted.

Treatment.—Active congestion is treated by slight elevatio
head; by *venesection* in extreme or very plethoric cases in ad
in children by application of *leeches* over the mastoid bone, tl
of which connect with those of the brain; by *free purgation,* by
of croton oil, followed by mercurials and concentrated salines,
the blood to the large abdominal vessels; by *hot baths,* to di
blood to the periphery. Some practitioners prefer to give coo
with active friction; by *vasoconstricting* measures, especially d
alcohol and opiates (save in small doses) should be avoided, a
mides should be used; ice may be applied to the head and c
a light—especially a *fluid diet* should be given.

III. Œdema of the Brain.

Etiology.—Œdema of the brain results from the same
causes as general œdema: (*a*) circulatory disturbances, as veno
gestion in cardiac disease, emphysema, and the acute infection
(*b*) the *marantic group,* including cachexia and nephritis. Co
and inflammatory œdema may develop around brain tumor, a
or hemorrhage. In some instances brain œdema occurs in the
agony. It may also develop when the brain shrinks, *hydrops ex*

Pathology.—The brain is pale, moist, and glistening on
the so-called "wet brain." The affection may be general or
the ventricular and subarachnoid fluid is usually increased.
volume of the brain may be somewhat increased in the gene
form.

Symptoms.—The symptomatology is indefinite, and a certai
nosis cannot be made between œdema and lessening of the ca
velocity (adiämorrhysis). When the œdema is localized, tr
focal symptoms may develop, as unilateral convulsions, hemi
or even crossed paralysis; the latter was observed personally i
of nephritis. Great variability in the symptomatology of organi
disease may be due to associated œdema. Traube held that
œdema causes the uræmic manifestations of nephritis, a view
ously opposed by Cohnheim.

IV. Cerebral Hemorrhage.

This is the most common and important of all brain lesions, and its symptoms involve an enormous part of brain pathology. It is usually hemorrhage into the brain substance, and is spontaneous, *i.e.*, it does not result from trauma. Although apoplexy strictly refers to a "stroke," which is symptomatic of various other lesions, it is used to indicate hemorrhage into the brain (Rochoux).

Etiology.—(*a*) *Miliary aneurysms* are the chief, and according to some, the sole cause of hemorrhage of the brain; the leading issue at present relates to their pathogenesis. According to many authors, the causes of hemorrhage are (*b*) those of *arteriosclerosis* (alcoholism, plumbism, syphilis, hard physical work, contracted kidney, gout, diabetes, and over-eating), but others maintain that atheroma does not *per se* predispose to hemorrhage. Eighty per cent. of hemorrhages occur *after the fortieth year* of life. It may occur in early life from glioma, whooping-cough, or trauma. More cases occur *in men* (80 per cent.) than in women. *Heredity* is not an uncommon factor, but relates rather to renal and arterial lesions than to an actual heredity of hemorrhage; this is especially emphasized by English writers, and by Dieulafoy. Hemorrhage is most common among civilized races and in temperate zones, because of the strenuous habits of life. For years the importance of the *apoplectic habitus* (plethora, ruddy face, short, thick neck, wide shoulders and chest, round, small body and large muscles) has been emphasized, although Gowers finds most of his cases in thin individuals. Contracted kidneys are found in 33 per cent. of cases of cerebral hemorrhage, and are among the chief causes of atheroma and miliary aneurysm. (*c*) *Increased arterial tension* is usually considered an important etiological factor. Hypertension alone is less important than its relation to arteriosclerosis and miliary aneurysms, which occur where the blood pressure is greatest, as where the vessels branch. Rupture is also seen in hypertrophy from stenosis at the isthmus of the aorta. Syphilis is usually considered a factor, but it more frequently produces softening than hemorrhage; it is a causal factor of aneurysms of the large vessels of the brain. Violent exertion, excess in liquor, excitement, coitus, straining, overeating, vomiting, coughing, or convulsions, may cause rupture of an already-diseased vessel, or a miliary aneurysm. A healthy vessel almost never bursts, however high the blood pressure. Embolism from heart disease may produce the "embolic aneurysm." Sometimes trauma is a factor, usually, however, with the causes mentioned. (*d*) *Permeability of the blood vessels* causes a few cases. In nephritis, purpura, various anæmias, leukæmia, septic infection and specific fevers, hemorrhage by diapedesis through the arterial wall is sometimes seen, and embolism is occasionally causative.

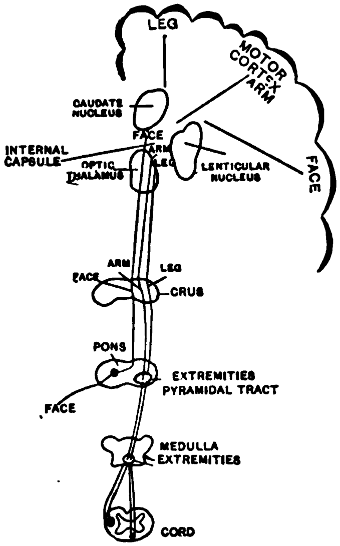
Pathology.—Hemorrhages most often occur in the branches of the arteria fossæ Sylvii. Among its branches the lenticulostriate artery of Duret, called, by Charcot, *the artery of hemorrhage*, is the seat of

may be seen with the naked eye, but are best observed
ing or macerating in water, and examination on a sl
power. They are found mostly in persons over forty y
in the locations where hemorrhages are most comm
central ganglia. Their pathogenesis is obscure. Char
ard described a change in the outer tunic (periarteri
and Zeigler held that intimal changes were primary.
miliary aneurysm is due to atrophy of the muscular
present the generally accepted theory. They are the ch
the sole, cause of spontaneous hemorrhage, although
from simple arterial degeneration. Large aneurysms w
separately.

Localization.—Hemorrhage is most common about t
cle, *i.e.*, in the caudate and lenticular nuclei, the inter
the adjacent centrum ovale, extending to the optic thal
beginning in it. Hemorrhage in the white matter has
gated form. It is much less common in the cortex, pons,
or medulla. Though it occurs mostly in the substan
hemorrhage may either rupture on the surface, or bre
tricles, causing sudden death; this is the usual autopsy
apoplexy. Hemorrhage is usually single. The multi
served in anæmia. When the skull is removed in cases
rhages, the dura is tense, the convolutions are flatten
falx is pushed toward the other side, and the fissures
Fluctuation may be felt in rare cases, and on section t
it sometimes involves the entire hemisphere, or may be
pea, but usually the size of a hazel-nut or walnut. T
the focus is softened from pressure, œdematous, an
torn and pulpy. The dark clot later becomes ch
then yellow-red, and finally yellow-white. The wall

is much less common. *Traumatic hemorrhage* may occur, though infrequently, near the point of injury or *contre coup* on the opposite side; it sometimes takes place after a week or more; this is the late traumatic hemorrhage of Bollinger, which occurs near the fourth ventricle and aqueduct of Sylvius, and probably, in areas of traumatic softening. Brain hemorrhage may indirectly press on other parts, may press on their arteries and veins, or directly sever the nerve fibers of the internal capsule.

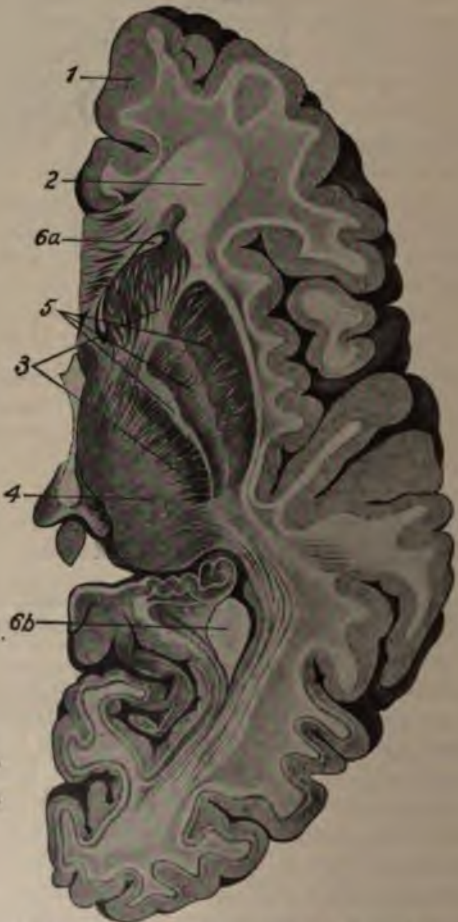
Fig. 56.



Showing course of individual fibers of motor tracts.

Secondary degeneration occurs in the *pyramidal tracts*, mostly as a result of lesions of the internal capsule. It is usually partial, and sound and diseased fibers intermingle; it descends through the crus, pons, medulla, and cord, in the latter of which it involves the inner part of the anterior columns of the same side (the uncrossed or direct anterior pyramidal tract), and part of the lateral columns of the opposite side (the crossed lateral tract). These areas become progressively smaller, and cease just above the conus medullaris. In children a cerebral lesion may cause degeneration in the anterior horns of the cord and their fibers. An ascending degeneration occurs only when the large pyramidal tracts of the cortex are diseased, except in the young, in whom degeneration of all neurones is prone to follow the destruction of one neurone. (See figures under LOCALIZATION IN THE SPINAL CORD.) Other degenerations may occur. (*a*) The *optic radiation* degenerates when a lesion occurs in the cortex of the cuneus, and also degenerative changes in the external geniculate body and pulvinar. After years it may reach the optic nerve. (*b*) Hemorrhage in the second and third frontal lobes leads to degeneration in the *anterior limb of the capsule,* median basal fibers of the crus, and the fibers connecting the optic thalamus with the cortex. (*c*) Lesions in the temporal lobes cause degeneration in the *lateral part of the crus.* (*d*) Degeneration in the median lemniscus follows diseases of the cortex, the subthalamic region and the upper part of the pons; it crosses to the opposite nuclei of the medulla, and after years destroys the ganglion cells in the posterior columns of the cord. Cerebellar hemorrhage may (rarely) cause degeneration of the arm of the pons and of the corpus restiforme.

Fig. 57.



Horizontal section through the right hemisphere; 1, Cortex; 2, white substance; 3, internal capsule; 4, optic thalamus; 5, lenticular nucleus; 6*a*, anterior horn; and 6*b*, posterior horn of the lateral ventricle. (After Déjérine.)

Symptoms.—*Prodromes* are most rare. Miliary aneurysms produce no symptoms until they rupture, and premonitory symptoms are due to *atheroma.* None of them are pathognomonic. They may be

) general, as described under congestion and softening (*v.i.*); cardiorenal, as hypertrophied heart, high arterial tension, and urinary findings of contracted kidney; or (*c*) sometimes focal, as lopia, alexia, facial paresis, dysarthria or preparalytic chorea. ssibly in some cases prodromal symptoms are due to an initial small norrhage, or to hemorrhage developing very slowly.

The Insult or Stroke and Its Primary Symptoms.—The "stroke," apoplexy is usually *sudden;* its most common general symptom is *a*, which may be absent in small lesions, and its most common focal nptom is *hemiplegia*. It may develop during effort or excitement, again without cause, as during sleep, when the head is low. In *ravescent apoplexy* the onset may be gradual, with delirium, con- sions, and finally coma; it is due to hemorrhage external to the icular nucleus, and later involves the internal capsule (hemiplegia). ath may, in rare instances, occur in five minutes (Abercrombie) n rupture into the ventricles, or from direct pressure on the medulla *lminating apoplexy*).

The insult, or stroke, is due chiefly to *anæmia of the cortex*, and to ne extent, to direct brain trauma, pressure by the hemorrhage on ıer vessels, serous infiltration and alterations in the cerebrospinal id (in which lumbar puncture sometimes shows blood). Its *cardinal ical symptoms* are *coma*, which is greater the nearer the lesion is the optic thalamus and third ventricle; *flushed face*, or some- es pallor or cyanosis; usually a full, tense, regular and *slow pulse;* g, deep, *stertorous breathing* (due in part to palate paralysis); the *eks puff out*, but are drawn in on inspiration, as a result of paralysis the buccinator muscle; inspiration is less irregular than it is usually d to be; yawning is frequent (Todd); the *temperature* usually suffers *initial fall*, but rises when reaction sets in, though lesions in the ıs, medulla or central ganglia may cause an initial rise. The *pupils* variable, usually wide, from paralysis of the third nuclei, or from npathetic stimulation; they are sometimes unequal, and in deep na, *reactionless*. They may be narrow from irritation of the oculo- tor nucleus in lesions of the pons, or when the ventricles are involved. nvulsions are infrequent. The head and eyes look toward the side the brain lesion ("conjugate deviation"), *a flaccid hemiplegia* curs, in which the limbs fall limp when they are lifted; the paralyzed ıscles appear semi-fluid and lax (Heilbröner); the *reflexes* are *gone*, nich symptoms will be described in detail below. There are *invol- tary evacuations* of urine and fæces. Ollivier described an increase the urine with a low specific gravity (1,004), and transitory *albu- nuria* and *glycosuria;* these are usually due to a large lesion pro- cing indirect or distant pressure on the medulla, or to lesion in the dulla itself.

The permanent damage can only be estimated when the reaction is ssed. The *direct focal symptoms* are those of the hemorrhage itself, d the subsequent cyst or scar; they are permanent if essential structures e involved, as the internal capsule. *Indirect focal symptoms* are those of other structures disturbed by pressure, œdema or inflammatory reaction. Time is required to separate these two classes of symptoms. The pulse and respiratory symptoms, conjugate deviation, albuminuria and temperature are usually indirect symptoms.

Details of improtant symptoms:

1. *Convulsions* are not common; they indicate involvement of the pons, ventricles, cortex or corpus striatum.

2. *Conjugate deviation,* described by Vulpian,. but especially by Prévost, is a transitory phenomenon in which the *lateral muscles of the head and eyes are paralyzed;* their fellows on the sound side contract, so that "the patient *looks at his lesion*" (Prévost); that is, in left hemiplegia, the eyes and head look toward the right side of the brain, where the lesion is located. The centres paralyzed are in the lower parietal lobe (gyrus angularis and gyrus supramarginalis, Landouzy). It disappears in a few days, because the sound side of the brain compensates or because it is a distant pressure symptom, or at the most, only a moderate paresis remains, shown by nystagmus. Conjugate deviation may also be due to *irritation* or convulsions (Jacksonian epilepsy), when the patient "looks away from his lesion" (Landouzy), *i.e.,* toward the side affected with convulsions. These differ from the deviation in localization in the pons (*q.v.*).

3. The *hemiplegia* (see LOCALIZATION, INTERNAL CAPSULE) is due to lesion of the internal capsule (66 to 75 per cent. of cases). During deep coma it is often difficult to determine which side is paralyzed. The affected side is usually flaccid, and the mouth shows greater puffing on the hemiplegic half. If the coma is not profound, the sound arm and leg may move. Sometimes, especially with hemorrhage into the lateral ventricle, the paralyzed side is rigid (early rigidity or contracture). The temperature is at first lower on the hemiplegic side from paralysis of a cortical centre. Later, during reaction, it is higher on the side of paralysis and may occur with œdema in this arm and leg. After consciousness returns, the contralateral hemiplegia is more clearly seen. The lower two-thirds of the face (nose, mouth, cheek) are paralyzed, but the upper third (eye and forehead) is usually quite free; in very rare cases only, a total facial paralysis is seen. The lids can be moved, but the nasolabial fold is obliterated, the lips cannot be puckered as though to whistle, the mouth is oblique, or a little triangular when the teeth are shown. The upper part is so often unaffected because it is innervated from both hemispheres, or because it has two distinct centres and paths, whereas in peripheral facial paralysis all branches are involved, and atrophy and the reaction of degeneration occur. If the optic thalamus (*q.v.*) is intact, the paralysis is not apparent on emotional movements, as laughing. The *tongue* is slightly involved; when protruded it deviates toward the diseased side (Schiff, Heidenhain), because the sound genioglossus muscle pulls the tongue forward and over toward the hemiplegic side; the base of the tongue is higher on the paralyzed side; it may lead to some difficulty in the external speech mechanism (dysarthria),

vhile internal speech (*i.e.*, aphasia) is not often disturbed unless here is psychical alteration. The *palate* may hang lower on the aralyzed side, but this is of little importance because it varies physiogically. Deglutition, mastication and phonation are but little or nly transitorily involved; marked difficulty in mastication is usually ssociated with total hemianæsthesia. In the *trunk*, paralysis of the apezius and levator anguli scapulæ causes sagging of the shoulder, hile other muscles of the neck are relatively free. (Wernicke hinks certain cerebral fibers of the spinal accessory are involved, hich is analogous to the partial facial paralysis.) The *accessory uscles of respiration*, as the scaleni, are involved on deep inspiration, nd ordinary respiration is somewhat less complete, to which some vould refer certain lung complications as atelectasis. Hughlings-ackson finds respiratory movement greater on the paralyzed side n automatic breathing and greater on the sound side in forced breathng. Early, the paralysis of the *arm and leg* is often absolute, someimes partial (hemiparesis). The arm is usually more involved than he leg. In rare cases the arm may be free in linear lesions; again he leg may be unaffected, or the face remains free (see INTERNAL CAPSULE LOCALIZATION); monoplegia is much less common than in cortical and subcortical disease, and when one member is more affected than the others, there is still partial or "dissociated" hemiplegia. In the *typical case* the arm is most involved, though all parts on one side usually are somewhat paralyzed. The arm abductors, hand flexors, and finger extensors are most affected. In the leg, the extensors of the leg, dorsal foot flexors and knee flexors are most implicated. The *paralysis is flaccid*. Considerable improvement may follow (see SECONDARY SYMPTOMS). The hemiplegia is almost invariably contralateral, *i.e.*, opposite to the side of the lesion. (In the older literature hemiplegia on the same side, or collateral hemiplegia was described; it was referred to the rare lack of decussation of the pyramidal tracts; however, if the motor tracts do not cross in the brain, they usually cross in the cord. Collateral hemiplegia, of which Lederhose collected 45 cases, is due (*a*) to more than one lesion, (*b*) to a focus pressing on the other side of the brain, or (*c*) to mistaking the early rigidity of the paralyzed side for the normal side, Pineles and Ortner.)

4. *Sensory disturbance* has been considered in the topography of the internal capsule. It is observed mostly during the insult or reaction, is less important than hemiplegia, less frequent, less distinct, less complete and less stable. Some hemianæsthesia is frequently found in early cases after careful examination, as disturbed tactile sensation, stereognostic sense and sometimes muscle sense, pains and hyperæsthesia or paræsthesia. A large lesion only, can cause a complete hemianæsthesia, which very seldom occurs without some paresis. It affects the skin and mucosa of the nose, mouth, palate and eyes, but leaves the cornea intact (Grasset), which is supplied by the ophthalmic ganglion (see DIAGNOSIS). Cortical hemianæsthesia is usually partial and most often united with a monoplegia; lesions a little

above the capsule, may produce hemianæsthesia, involving the fibers from the thalamus to the cortex. Hemianæsthesia also occurs in lesions of the crus, pons, medulla and spine (*q. v.*).

5. The *special senses* are not often involved, or are affected but temporarily. Hemianopsia is frequent (Gowers and Kines), usually occurs with conjugate deviation, is temporary, and is often overlooked; it probably is due to indirect pressure on the optic thalamus.

6. The *reflexes* of the skin, cornea, pupils, mucosæ and tendons are suspended when there is complete coma. Theoretically they should be increased by withdrawal of cerebral inhibition, but the central nervous system is almost wholly paretic during severe coma. After the stroke they return slowly, first on the sound side; the corneal reflex, the reaction of the pupils to light, the epigastric and other skin, and tendon reflexes return in the order named; the patellar reflex is often exaggerated. On the *hemiplegic side*, they return slowly, the periosteal tendon reflexes and patellars often become exaggerated; this is due to interrupted inhibition. The patellar reflex on the diseased side may return only after a considerable time. The skin reflexes return slowly, especially the epigastric, cremasteric, scrotal and mamillary (Jastrowitz); this is a point of early diagnostic value; they probably have a higher centre than have the tendon reflexes, although the explanation of their conduct is not clear; it has also been suggested that there is a cerebral centre controlling Setchenow's inhibitory reflex centre in the medulla; when this higher centre is affected, the medulla centre wholly inhibits the reflexes.

The *Babinski sign* (toe phenomenon) indicates organic pyramidal disease and may be found very early; when a sharp object is slowly drawn over the outer part of the sound sole, there is plantar flexion of the large, and sometimes of the other toes; when drawn over the diseased sole there is great dorsal flexion (extension) of the great toe. After the prodromes and the initial primary insult, coma and reaction are over, the chronic symptoms can be studied to the best advantage.

The Chronic or Secondary Symptoms.—The indirect focal symptoms (conjugate deviation, coma, etc.), the inflammatory reaction and collateral œdema disappear; this is followed usually by considerable improvement.

1. If the *hemiplegia* is indirect, from lesion without the capsule, marked improvement or rarely, total recovery ensues. Complete lasting hemiplegia follows total division of the pyramidal tracts, due to hemorrhage from the anterior choroid artery (Kolisko). The face may recover rapidly; certain muscles are entirely unaffected or are involved for a short time only; this is explained by *Broadbent's theory* that *muscles used in pairs* are little involved or escape entirely; with some modification of his theory, we find that: the *intercostals, masseters* and trunk muscles used together, innervated from both hemispheres and having little differentiation in function, escape permanent paralysis. The muscles of the *arm* and *hand* have especially complicated functions, are more often used alone, are innervated almost

holly from one hemisphere, and therefore suffer the greatest per-
anent damage. In adults the hand is usually disabled for life.
he *legs* are much used together, have some innervation from both
emispheres, and often show some improvement after paralysis. Just
ow compensation occurs is undetermined. Broadbent thinks that
e bilateral spinal and bulbar nuclei enable "paired muscles" to
e innervated from the sound hemisphere. Possibly uncrossed fibers
the anterior (and lateral) columns may explain the phenomenon.
ompensation is not an absolute advantage, for Brown-Séquard found
e sound side reduced in strength in 50 per cent. of his cases. The
ce often improves after an attack. In the *arm* the residual paralysis
fects functionally associated muscle groups rather than individual
uscles, as the muscles supinating the forearm, those abducting and
ing the arm and shoulder, those moving the elbow, opening the
nd, and apposing the thumb (Wernicke and Mann). The shoulder
proves a little, the elbows considerably, the hands very little. In
leg, the flexors, dorsal flexors of the foot which "shorten" the
in walking, abductors and internal rotators of the hip are most
olved. The hemiplegic gait is awkward, the foot is swung out
d around in a half circle and scrapes the ground. The power of
paralyzed leg is greater when it is used with the sound leg, as in
lking, than when it is used by itself.

2. The *contracture*, the important posthemiplegic manifestation, is
transitory or permanent fixation of a joint in an abnormal posture
m nutritive or functional factors. *Passive contractures* are mechan-
al, as from muscular shortening, while *active* contractures result
om disease of the central nervous system. Early contractures are
ten regarded as tonic spasm (myotatic irritability). The late con-
acture develops in two to four weeks and is quite typical; the shoulder
lifted, the arm adducted to the chest, the elbow rigid, the forearm
ronated, the wrist flexed, and the fingers flexed even violently into
e palm. When the wrist is passively flexed the fingers can some-
mes be extended. In general the contracture involves both extensors
nd flexors, but more often the latter. The contractures occur in the
uscles which are least paralyzed. In the lower extremity, the con-
acture is not marked (in the thigh there is often only a trace), the knee
extended, plantar flexion of the foot occurs (*pes equino-varus*), the
eat toe is dorsally flexed. Locomotion is difficult, for there is
ending of the trunk to the sound side, lifting of the pelvis because
the adduction paralysis, and swinging of the foot over the sound
ot. There may be pain in walking, and the sound leg also suffers
me slight contracture. Contracture may occur in the face, platysma
nd sternomastoid, but the trunk is rarely affected. Contractures are
ncreased by cold, irritation, and movement, and decreased by massage,
warmth and sleep. They may be caused by irritation in the periph-
eral nerves, inflammation in joints or muscles, interruption of inhibition
in the pyramidal tract or irritation acting on lower centres as the pons
or medulla. Hitzig regards them as involuntary associated movements.

In rare cases the paralyzed side remains lax (*hémiplégie flasque* o Bouchard, occurring largely in children).

3. *Associated movements* are variously explained; by Westphal as due to lessening of reflex inhibition, by Hitzig as movements attempted in paralyzed muscles but reflected to their associates, and by Leube as due to bilateral representation in the hemispheres; examples: an effort to grasp with the sound hand produces a similar movement in the one paralyzed; yawning or coughing produces a movement of the paralyzed arm; movement of the diseased side occurs after passive movement of the sound one; movement of the paralyzed foot results when the patient attempts to flex the hip.

4. *Hemitremor* is uncommon. The muscles are stimulated from the brain but not enough vibrations occur for actual involuntary movement.

5. *Atrophy* is less marked than in peripheral and spinal lesions, but may occur in both spastic and flaccid cerebral forms, though more in the latter; Steinert recently concluded that atrophy is as frequent in cerebral as in peripheral paralysis. Charcot referred it to degeneration of the anterior horns of the cord, while Quincke and Senator assume that it results from injury to some trophic paths. It is distinct from lack of development in the young. It develops in a month or two, has no relation to the degree of paralysis, is often associated with disturbed sensation and muscle sense, affects principally the arm, occurs in large lesions which are either (*a*) cortical in the region of the third branch of the artery of Sylvius, or (*b*) central in the distribution of lenticulothalamic artery. The electrical irritability of the muscles is either normal or somewhat depressed. The skin also may atrophy.

6. *Posthemiplegic chorea* (Wier Mitchell, Charcot, Raymond) results in 80 per cent. of cases from lesions in the internal capsule, or near it, in the optic thalamus, the lenticular or caudate nuclei, from which irritation is reflected to the brain cortex. It sometimes is preparalytic (Grasset). It develops months or years after *small lesions*, when the hemiplegia and rigidity are largely gone. It occurs especially in the arm, causes movements similar to but coarser than those of chorea, sometimes resembles the tremor of multiple sclerosis or paralysis agitans, and is often associated with pain and sensory disturbance (see LOCALIZATION, INTERNAL CAPSULE). In *posthemiplegic athetosis* (Hammond), the fingers and toes are incessantly moved, athetosis meaning "without fixed position." It is a "mobile spasm" consisting of somewhat spasmodic, slow, coördinated movements in paralyzed members; it is frequent in partial paralysis acquired early in life is slow in development, appears after the paralysis has regressed, an has the same localization as hemichorea. The following differentiati by Greidenberg and v. Monakow gives the main characteristics the two affections:

Hemichorea.—*vs.*—Hemiathetosis.

1. Face, trunk, whole extremity involved; may pass to all extremities.	1. Chiefly or alone in forearm and hand (fingers) and in leg and foot (toes); simultaneous innervation of antagonists; other parts paretic and stiff.
2. *Excursion* active, manifold, tremor-like, purposeless, increased by exertion, tension lost by irritation.	2. Slow rhythmic hyperextension, elastic resistance, seems purposive, joints fixed.
3. Contracture little or none; limbs relaxed; no great deformity.	3. Contracture frequent, but spasmus mobilis often great.
4. Hemianæsthesia frequent.	4. Hemianæsthesia rare (almost never in children).
5. Quiet in sleep.	5. Not always quiet in sleep.
6. Increased by movements and attempts to suppress them.	6. Lessened to some extent by movement.

7. *In trophic and vasomotor disturbance* (*v. s.*, Muscular Atrophy), the paralyzed members are usually cool, moist and sometimes œdematous. The skin is glossy, even livid and fissured. Nothnagel observed in some cases paralysis of the cervical sympathetic system causing increased temperature, redness, narrowing of the eye fissure, drooping of the upper lid, retraction of the eyeball, lachrymation and narrow pupils. Sometimes there is overgrowth of the hair. Bed-sores were considered trophic by Charcot, but can usually be avoided by care regarding pressure or heat, and by cleanliness. Swelling of the joints and hypertrophic neuritis (Charcot) on the hemiplegic side are rare.

8. *Psychic disturbance* is not symptomatic of brain hemorrhage, and is chiefly referable to arteriosclerosis. It is evidenced by self-concentration, weak memory, insomnia and irritability. Carpani found more mental alteration when the left half of the brain was diseased. Laughing and weeping are referable to disease of the optic thalamus (*q. v.*), or to disease of the median fibers of the pons (Brissaud).

Diagnosis of Brain Hemorrhage.—Two cardinal features, *coma* and *hemiplegia*, together with the *typical insult* facilitate diagnosis. In cases with slow onset, differentiation from thrombosis (*q. v.*) is difficult.

Coma.—In *syncope* the pulse is fast, weak and often irregular; in "cardiac apoplexy," an insult follows heart rupture, myocarditis, and various forms of heart failure, and differentiation from rapidly fatal brain hemorrhage may be impossible. It is always dangerous to make a diagnosis of *alcoholic coma;* an intoxicated individual may suffer injury, basal fracture or actual apoplexy; an opinion should be withheld until such complications can be excluded, even though the alcoholic coma is usually incomplete, the vomitus and breath smell of alcohol, violent struggling is frequent, and early convulsions and focal symptoms are rare. *Opium coma* is characterized at first by slow onset, slow respiration, slow, hard pulse, flushed skin, great contraction of the pupils, opium in the urine or stomach washings, and absence of convulsions and focal symptoms; later there are Cheyne-Stokes respiration, rapid pulse, damp cyanotic skin and dilated pupils. Hemorrhage in the pons produces narrow pupils, which are often unequal. In this and similar intoxications, the focal symptoms, and reflexes of apoplexy are lacking. While in general the diagnosis of apoplexy during the coma is precarious, *uræmic coma* rarely occurs without premonitory symptoms, as *headache*, *drowsiness*, *delirium*, *restlessness*, *vomiting*, *uremic retinitis*, or less often convulsions; the coma is often less profound than in cerebral hemorrhage. The renal, cardiac and arterial findings of nephritis may also occur in apoplexy, of which nephritis is a frequent cause (33 per cent.), and it must be remembered that local symptoms in uremia sometimes occur; the author has repeatedly observed hemiplegia and even crossed paralyses. In deep coma rigidity may be observed in both uremia and apoplexy, but in uremia it is often variable. In *diabetic coma*, the fruity, acetone breath, acetone in the urine, headache, unrest, Kussmaul's breathing and absence of focal symptoms are characteristic. The coma may be sudden, granular casts are frequent and the sugar may disappear; the diagnosis is then only suggested by a clear history of glycosuria. *Symptomatic apoplexy* is of more theoretical than practical importance; transient coma and hemiplegia sometimes occur in brain œdema, congestion, progressive paralysis, multiple sclerosis, and more rarely in epilepsy and tabes. *Hemiplegia*, not resulting from any *lesion* may occur when the condition of the blood is altered (alcoholism, plumbism, gout, tuberculosis, cancer, uremia, diabetes, pneumonia).

Hemiplegia.—This is a prominent finding in embolism and thrombosis (*q.v.*, for DIFFERENTIATION); an exact diagnosis is often impossible. Kolisko states that most cases with the clinical diagnosis of embolism prove to be hemorrhage, and *vice versa*. Hemiplegia may occur in brain abscess, and meningitis. When it occurs in meningitis with coma, there is headache, more generalized convulsions, rigidity, hyperæsthesia, optic neuritis, eye-paralyses and fever. In extradural hæmatoma and pachymeningitis, the Jacksonian epilepsy followed by cortical paralysis is distinctive, *i.e.*, the focal symptoms are prominent compared with the general symptoms of early apoplexy. Hemiplegia also occurs in hysteria (*q.v.*), syphilis, brain tumor, trauma, and in some cases of chorea and paralysis agitans.

Other Locations of Hemorrhage.—(*a*) Hemorrhage may occur in the meninges (see DISEASES OF MENINGES). (*b*) It may occur, though rarely, in the *cortex;* Jacksonian epilepsy in subcortical lesions, convulsions and rigidity may be severe and lasting (Mills). (*c*) *Ventricular hemorrhage* is usually secondary to hemorrhage in the corpus striatum or optic thalamus and usually reaches all the ventricles. It may in rare cases be primary from aneurysms or ruptured veins in the choroid plexus (Dana). One-third of all cases are in persons under forty years of age. It occurs usually with violent symptoms, nvulsions (33 per cent.), early and sometimes bilateral rigidity. ie conjugate deviation changes and turns to the other side, the lse is slowed, the temperature falls, the reflexes are suspended, eyne-Stokes breathing, glycosuria and polyuria (Pascheles) develop, d death usually results within a day; the onset, however, is some- es slow (Broadbent). (*d*) Hemorrhage in the *crus* (*q.v.*, LOCALIZA- N) may be primary but is usually descending, from bleeding near internal capsule. Crossed hemiplegia oculomotoria (Weber's e), crossed ptosis (Richards) or diplegia (Paquet) may be observed. . Apoplexy in the *pons* (*q.v.*) occurs in 10 per cent. of cases, often the median line, and frequently with convulsions. Paralysis and vulsions may occur in the arms or legs only; the trunk musculature involved more frequently than in higher hemorrhages. The eyes k away from the side of the lesion. The pupils may be large from ralysis or small from stimulation of the third nerve; in the latter stance opium poisoning must be differentiated. Respiration is fected early, vomiting is frequent, there is often early high fever d death is usual within twenty-four hours. It has occurred in seven inutes. (*f*) Hemorrhage in the *medulla* will be considered later. arly or instantaneous death is frequent, but a gradual onset is known. is rarely diagnosticated. It may very rarely induce gastro-intestinal morrhage. (*g*) *Cerebellar apoplexy* occurs in 5 to 8 per cent. of ses. Vomiting is very frequent (50 per cent.). The insult is some- mes less marked than in the usual type, and "the absence of hemi- egia is more significant than its presence," (Gowers). Hemiplegia d symptoms from the fourth ventricle are due to indirect pressure secondary ventricular hemorrhage. Cerebellar symptoms, as ataxia, ay result (see LOCALIZATION).

Prognosis.—The prognosis concerns (*a*) the immediate danger and b) the ultimate outcome. (*a*) The *immediate prognosis* is always erious. In hospital cases 75 per cent. die but in general practice 6 per cent. survive the first stroke, and 33 per cent. the second, and very few the third. When death occurs, it is usual in one-half to two days, but the outcome is uncertain for a week, because light cases, even the rare forms without coma, may rupture into the ventricle with rapidly fatal result. Coma lasting more than twenty-four to thirty hours is dangerous. Early low fall of temperature or its rapid early rise is unfavorable, indicating a large focus. For the same reason, much conjugate deviation, relaxed sphincters, impaired breathing, trachial rattling, bilateral or ventricular symptoms, marked glycosuria or albuminuria, decubitus in the first week, convulsions and rapid pulse are unfavorable. The mode of death is as follows: the breathing becomes more rapid, irregular, of the ominous Cheyne-Stokes type, and rattling from accumulated secretion in the air-passages; the slow and regular pulse becomes rapid, small, irregular, often with a rise of temperature, and finally ceases, but respiration or a time continues; death occurs in total coma.

(*b*) *Ultimate outcome*—the symptoms of shock disappear and the patient becomes partly conscious or delirious; the *period of reaction* begins, usually within forty-eight hours after the onset. The pulse becomes faster, the temperature rises, pain develops in the head and limbs and sometimes, though rarely, the paralyzed limbs become rigid. Life still hangs in the balance, and the patient may die from the reaction, from hypostatic pneumonia, called trophic by some writers, from pulmonary œdema or hemorrhage, acute decubitus, or from a second brain hemorrhage. Reaction is over in one to six weeks. If the patient survives the insult and reaction, there is always some improvement, depending on the extent and localization of the injury which can be determined only after weeks or months. If movement appears in three or four weeks, the outlook is good; if it does not return in three or four months, it is not likely to develop later. Disappearance of indirect pressure leads to bettering (i) of damage to the sensory nerves and those of special sense, (ii) of motility, first of the trunk and face, then of the leg, and less of the arm. Absolute recovery is possible but improbable. Hemichorea and hemiathetosis are incurable. Broken neurones never unite, and secondary degeneration, contractures and increased reflexes result. Exaggerated reflexes developing a few weeks after the injury, are forerunners of contractures and ankle clonus, which preclude full regression. The outlook is fairly good in children when the hemorrhage is not due to blood disease, because compensation is likely to occur. Recurrence is possible in decrepid and aged subjects, although less common than usually believed; the average duration of life is five years (Dana).

Treatment.—**1. Prophylaxis.**—Prophylaxis regarding worry, hard work, syphilis, the use of alcohol, eating and excesses is necessary when there is hereditary tendency toward hemorrhage, or when one hemorrhage has already occurred.

2. The Insult.—(*a*) Absolute *quiet* is indicated, and any movement, transportation or extended examination should be avoided. (*b*) The head should be slightly elevated, but flexion of the neck, tight clothes or anything hindering the return venous flow should be avoided. Bowles thinks that stertorous breathing is due to the position of the paralyzed palate, and that turning the patient on his side relieves it more effectively than does venesection. (*c*) *Venesection* is seldom used, since being discountenanced by Trousseau and Traube. It is certainly indicated only in robust individuals with strong hearts and tense vessels; about 12 ounces of blood should be withdrawn; the chief objection to venesection is the difficulty in diagnosis of cerebral hemorrhage. Any physician who has used it frequently will admit that he has occasionally bled patients with thrombosis, embolism, syphilis or tumor. In thrombosis and embolism venesection increases the anæmia of the brain, which at present holds a prominent place in the pathology of hemorrhage. Cushing has shown that the high tension is a vicarious effort to keep the medulla supplied with blood. The same objection holds to compression of the common carotid.

Leeches and sinapisms are harmless but useless. (*e*) The *ice-cap*
be used, but cannot influence the deep circulation in the brain.
Evacuation of the *bladder* and *bowels* is indicated. Two drops
oton oil will act in an hour or two, and in case of failure, the dose
be repeated. Croton oil was Hughlings Jackson's only remedy.
The *diet* should be fluid and should be administered by the nasal
ter or by rectum. (*h*) The *skin* should receive attention: de-
us is prevented in practically all cases by care regarding posture
cleanliness. Heat and sinapisms must be used with greatest
mspection because the hemiplegic side is prone to necrosis and
mmation. (*i*) *Symptomatic* treatment of cardiac and of respira-
failure is by champagne or ammonia, but this should not be
until necessary; treatment of restlessness or convulsions is by
al in enema, or by morphine which theoretically congests the
, but really quiets it without detriment.

The Reaction.—During the *inflammatory reaction* the treatment
holly expectant, and if possible should be conducted without
s.

The Chronic Stage.—(*a*) Gentle *massage* of the muscles, move-
t of the joints and alcoholic rubs are instituted as soon as the
a subsides. (*b*) *Potassium iodide* is indicated when the reaction
ides. (*c*) As soon as the leg improves, that is, in four to eight
ks, the patient should be encouraged to sit up and walk. (*d*)
lication of the *faradic current* to the limbs is certainly valuable
r two to four weeks, and, supplemented with massage of the
lyzed groups and their antagonists, minimizes the contractures.
Hydrotherapy is valuable; cool baths or rubs are efficacious
pt in anæmic or poorly nourished individuals. Warm baths are
e avoided in old people and strychnia in all cases.

V. Cerebral Embolism.

mbolism and thrombosis of a brain artery result in *brain softening*
ephalomalacia), which is next after hemorrhage in importance
frequency, and is one of the best known subjects in brain pathology.
n softening from arterial occlusion is not the "softening of the
n" known to the laity, who give that name to progressive paralysis
he insane. *Encephalomalacia* must not be confused with inflam-
ory softening (*encephalitis*). Softening was first recognized by
rcrombie in 1818, and by Rostan in 1820, while those coming
them usually considered it inflammatory. Virchow's researches
mbolism confirmed the views of Rostan and Abercrombie, while
nheim elaborated the vascular relations, and Panum, Piltz, Pré-
, Cotard and Cohn worked out Virchow's views.

iology.—In embolism, Spierling found the kidneys involved in
er cent., the spleen in 54 per cent., and the brain in 22 per cent.
ses. (*a*) About 90 per cent. is due to *heart disease.* Most com-

mon is *acute endocarditis* or acute exacerbations of chronic valvular lesions, in which the embolism is benign (mechanical) or malign (mechanical and septic). Calcarious fragments from the valves, or parts of torn valves, may be thrown into the brain. Of valvular diseases, mitral stenosis produces embolism most frequently because of stasis in the left auricle. Less frequent are cardiac clots formed in the weak hearts of decrepid, cachectic subjects, in myocarditis or in such acute diseases as typhoid. Weakened heart action and slow circulation are never the only cause. (*b*) *Atheroma of the arch and aneurysm* are far less frequent causes, as are (*c*) *lung lesions*, as tuberculous cavities, abscess, gangrene or empyema (after irrigation) tumors, echinococcus, gumma or abscess of the heart. (*d*) In the rarest instances, lung tissue (Böttcher), fat embolism from fractures pigment embolism (Frerichs) in malaria or liver cells in acute yellow atrophy, may produce cerebral embolism. (*e*) With a patent foramen ovale, thrombi may reach the brain from the venous system; it has been observed in manual expression of the placenta. (*f*) Most cases occur *in youth* (and middle age) when acute infections are most frequent, as chorea, scarlatina, or endocarditis.

Pathology.—(*a*) The *embolus* in fresh cases is reddish, or gray-red and later becomes paler, drier and more friable. There are often "riding emboli," which straddle the point of arterial branching; they may in rare cases soften and disappear, or by slipping produce another embolism further on (Laborde). Secondary thrombosis may occur at the infarction. (*b*) The *artery* beyond, save where the embolus retracts or softens, becomes a thin, hard, impervious cord. Behind the plug an aneurysm may form. If the embolus is septic, inflammation intervenes in and around the vessel. (*c*) The fate of the *brain tissue* depends wholly on (i) the degree of occlusion, and (ii) the collateral circulation. (i) If the stoppage is partial, only stasis, œdema and swelling occur; if total, necrosis results. (ii) The brain arteries are "end arteries," as Cohnheim pointed out, whence complete collateral circulation is unlikely, although the *necrosis* is *rarely as extensive* as the *distribution of the artery occluded*, even where, as in the basal ganglia, the anastomoses are few. There is more chance of collateral compensation at the cortex than at the base, and the least collateral compensation occurs in the deep parts of the centrum ovale (Duret and Heubner). The brain tissue is *ischæmic*, or its blood is venous (Marchand). The collateral vessels (arteries or venules) allow regurgitation around the focus, the vessels become more pervious, and some white and red cells often escape; this collateral fluxion saves the tissue more often than is usually admitted, especially if the heart is strong. With a weak heart or diseased collateral arteries, ischæmic necrosis must follow. Some pathologists hold that shutting off of a large bloodvessel raises the tension in the adjacent (collateral) vessels, and thus causes the small capillary hemorrhages often observed; but a marked hemorrhagic infarct is not common, for it is prevented by swelling of the nervous tissue

Weigert). Much extravasation follows occlusion of a large vessel, as he internal carotid. Ischæmia lasting a few hours probably produces necrosis (Brieger and Ehrlich) but microscopic changes are seen only after one or two days; the brain substance becomes œdematous, gelatinous, grayish, marbled, fusing with the slightly œdematous brain tissue surrounding the focus, without clear demarkation; necrosis develops rapidly, the brain becomes fluid and pulpy, although its remarkable cohesion is a matter of frequent comment. The *gray matter* contains yellow plaques (*v.i.*); certain convolutions may waste or disappear; the gray and white substance, especially after plugging of the posterior cerebral artery, may wholly disappear, as the hippocampus, lingual and fusiform lobules. The *white matter* often disappears, and is replaced by lax cellular tissue, in whose meshes are cystic formations with turbid fluid, and flocculi of brain tissue. The meninges are often thickened. Durand-Fardel described three forms of softening: (*a*) The *red softening* is usually fresh and is found most often in embolism; there is capillary extravasation, which lends it the red color; it is irregularly oval, or wedge-shaped in basilar lesions; it usually measures but a centimeter or two; it is prominent on section from blood and serum; it is soft, but not fluid in the centre; is mostly located in the cortex or central ganglia, lasts one or two weeks, and then passes into the second form. (*b*) *Yellow softening* follows the red; the yellow color is due to old blood pigment or to fatty change; it is sunken on section, from atrophy of the brain substance; is fairly demarked and may contain fluid, *hydrops ex vacuo;* it may last years and is found especially in the cortex. (*c*) *White softening* may represent (i) a *fresh lesion* about tumors (Bouveret) or abscesses, or in various cachexias, is sunken below the cut surface and occurs where few vessels are involved and little regurgitation from adjacent vessels occurs; (ii) *lesions* which are several years old, occurring when the blood and fat are absorbed, whence their snow-white appearance; demarkation is often slight, and if diffuse, gives rise to the spongy *état criblé*.

Demarkation may occur after weeks, producing a "cyst" of spongy structure and milky contents. More rarely neurogliar increase may lead to shrinking, and a *scar* may result. *Microscopically* myelin and fat drops, leukocytes laden with granules and fat, collapsed vessels, relics of necrotic nervous tissue and some increase in neuroglia are observed. Location of embolism will be considered under SYMPTOMS. *Secondary degeneration* occurs when the pyramidal tracts are involved (see HEMORRHAGE).

Symptoms.—As in apoplexy, we consider (1) the *insult*, and (2) the *permanent symptoms*, to which might be added the symptoms of the causal disease, *e.g.*, those of mitral stenosis, etc.

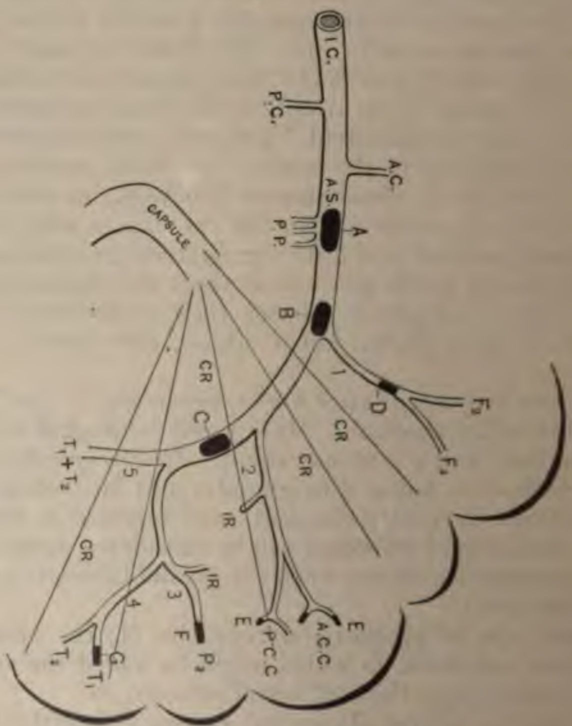
1. The *insult* is almost always (*a*) *sudden* and *without prodromes* (save when the embolism does not wholly occlude the vessel, and symptoms appear only when secondary thrombosis develops). Embolism in small vessels may occur without symptoms. The patient

may be stricken *without coma,* and sit "astonished" at the stroke, without a cry or even vertigo. The insult is caused by *brain anæmia, negative pressure and shock* (the *étonnement cérébrale* of Trousseau and Jaccoud); Geigel's idea that the rest of the brain is collaterally congested is less plausible. In some cases (*b*) *coma* occurs (generally *less profound* and *less protracted* than in hemorrhage); irritative symptoms, as (*c*) *convulsions, tonic spasms* and conjugate deviation are *more common;* convulsions are often unilateral, corresponding with the *hemiplegia.* (*d*) All possible *intermediate types* of *focal* disease are observed; for instance, cases with vertigo; tendency to fall to one side; hemianopsia, alexia, hemianæsthesia, motor or sensory aphasia, or only confusion, delirium and aphasia (Fagge). (*e*) The *pulse is full but not slow.* (*f*) The *temperature* rarely suffers initial depression, and later may rise.

2. The *permanent symptoms* and *secondary degeneration* are the same as in hemorrhage.

Localization of Embolism.—1. **The artery of the fossa of Sylvius** is most frequently involved (80 per cent.) because it is the largest and

FIG. 58.



Embolism of artery of the fossa of Sylvius

IC, internal carotid; PC, post. communicating; AC, anterior cerebral artery; AS, art. fossæ Sylvii and 1, 2, 3, 4 and 5, its main branches. CR, corona radiata from cortex to internal capsule. (See text for explanation of lesions at A, B, C, D, E, F or G.)

t direct branch of the middle cerebral which is practically a con-
ation of the internal carotid. Convulsions occur in 33 per cent.,
or without loss of consciousness, and are most frequent when the
ller branches are embolized. High temperature is common. The
cal picture varies with the site of infarction: embolism at A
. 58) where the perforating branches (P. P.) are given off to
nternal capusle, corpus striatum and anterior part of the thalamus,
t involve (*a*) these structures, and (*b*) the motor and sensory
ex, with *hemiplegia*, fairly complete *hemianæsthesia*, *motor aphasia*
ia), *deafness* and *word deafness* (if it occurs on the left side).
vulsions are absent, because the injured internal capsule cannot
ey the cortical irritation to the extremities; the large areas at
base and cortex thus rendered anæmic, may cause *marked reaction*
guenin), sometimes resulting in death. In rare cases only the
ral perforating branches are involved and the cortex obtains the
d by anastomoses with the posterior and anterior cerebrals. The
Sylvian artery is more frequently involved, because the left carotid
es directly from the arch; 59 per cent. are left-sided (Gelpkel), or
er cent. (Thénévet). If the lesion is at (B), (Fig. 58), the motor
ch area, F3, which produces motor aphasia, the anterior and
erior motor convolutions (A.C.C. and A.P.C.) which produce
iplegia or hemiparesis, the parietal convolutions, gyrus angularis
gyrus supramarginalis (P2) which produce alexia, and the tem-
l convolutions (T1 and T2) which produce deafness or word
ness, are involved. Somnolence and delirium are common; con-
ions almost invariably occur, because the internal capsule escapes.
vulsions and pain are most frequent in embolism of the smaller
els. Little hemianæsthesia is observed (see CORTICAL LOCALIZA-
r). A lesion at (C) is the most frequent site. It affects the island
eil (I.R.) in part; cortical convulsions, hemiplegia, monoplegia,
ory aphasia (T1, T2), alexia (P2) and, sometimes sensory dis-
ance occur. It is easily seen how an isolated lesion at (D) could
luce motor aphasia; at (E) a monoplegia; at (F) alexia; or at
word deafness.

Embolism of the posterior cerebral (in 2 per cent.) comes from
vertebral artery, and often occurs with embolism of the Sylvian
ry. It supplies (*a*) all the ventricles, the thalamus, ant. corp.
drigemina, the geniculate body and the peduncle by its central
iches, and (*b*) the mesial aspect of the occipital, part of the parietal,
most of the temporosphenoidal lobes. The most common general
ptoms are convulsions, coma, vertigo, conjugate deviation and
agmus; while the most important focal symptoms are *sensory*
ianæsthesia, *sensory aphasia* and bilateral homonymous *hemi-*
sia. The lesions are sometimes symmetrical.

Embolism of the anterior cerebral artery: Few clear cases are
rded, since the artery originates at a right angle to the carotid.

In embolism of the internal carotid (in 4 per cent.), the plug must
irge to produce much damage, for if the vessels of the circle of

Willis are normal, only transient hemiplegia results. Disease of these vessels, or abnormalities in size, may produce permanent, alarming or fatal results. Secondary thrombosis is especially dangerous. Sepsis is a frequent factor.

5. **Embolism of the basilar artery** occurs in 6 per cent.; the clot never fills the vessel, because it first passes the smaller vertebral artery; symptoms occur less frequently at the time of embolism than later when secondary thrombosis occurs. They are convulsions, trismus, paralysis of the third, fifth, and seventh nerves, crossed hemiplegia, diplegia, narrow pupils and conjugate deviation. The temperature often falls low and then rapidly rises. Death may follow in two to five days from respiratory failure.

6. **In embolism of the vertebral artery,** involvement of the tongue, larynx, lips, pharynx, spinal branch of the fifth nerve, result in anæsthesia, dysphagia or other signs of acute bulbar palsy. Hemianæsthesia, hemiplegia, and hemiataxia may develop. It is often fatal.

7. **Embolism of the cerebellar arteries** is the rarest of all embolisms.

Diagnosis.—The main points are (*a*) recognition of the *cause*, which is usually *cardiac;* (*b*) absence of *prodromes;* (*c*) *sudden* insult, frequent *convulsions*, and infrequent *coma*. (For details and differentiation, see table under THROMBOSIS.)

Prognosis.—The outlook depends (*a*) on the *primary* disease, being most favorable after acute infections, and in youth; (*b*) on the *intensity of the onset* itself, during which death may occur; (*c*) on the *localization;* the initial symptoms may improve; the outlook is more favorable than in hemorrhage. Psychical symptoms are cortical in origin, while the vegetative or trophic are due to lesion in the basal ganglia; basilar and vertebral embolism are most unfavorable prognostics. (*d*) Recurrence is possible, although it is less likely than is usually stated. (*e*) The condition of *other organs*, as the vessels of the circle of Willis, the strength of the heart muscle, etc., determine the prognosis.

Treatment.—(*a*) The *insult* is treated as in hemorrhage, in regard to diet, keeping the patient quiet, and general measures. (*b*) All derivative measures, as *drastics* and venesection, are *absolutely to be avoided.* (*c*) The heart must be *steadied* and *stimulated*, if hemorrhage is clearly excluded, with a view first, of relieving the brain ischæmia by a strong collateral circulation, and second, of strengthening the heart to avoid recurrence. Digitalis acts too slowly, and camphor, strychnine and ammonia are preferable. (*d*) *Convulsions* should be treated by chloral given by rectum, avoiding alcohol and supporting the heart. The administration of more than a dram of chloral in three hours is very dangerous; gr. x to xv should be given forty-five to sixty minutes apart. (*e*) The *after treatment* is unsatisfactory, but should be followed out as in the corresponding stage of apoplexy.

VI. Cerebral (Arterial) Thrombosis.

Thrombosis is more common than embolism. Three decades ago it was thought that fibrin was thrown out of the circulating blood by a ferment. Virchow held that slowing of the current was the chief cause, while Zahn, Weigert and others attributed great importance to coagulation necrosis of the leukocytes. Bizzozero, later also Ebert, and Schimmelbusch held that the blood plaques played a large part in coagulation. Brücke taught that the living healthy vessel prevented coagulation.

Etiology and Pathology.—**1. Changes in the Vessel Wall.**—(*a*) *Arteriosclerosis* (*q.v.*) is the chief cause of this most important group. It affects the basal arteries especially and usually several vessels. Blood plaques are deposited on the rough intima (Aschoff, Sahu), and in the tortuous, inelastic, calcarious, fatty vessels, with their unequal calibre; layer after layer of fibrin is deposited, aided by local slowing of the blood stream, and by weakening of the heart. Thrombosis may be local or extensive. Tumors, abscesses, and traumatic meningitis may initiate thrombosis. Brain atheroma usually occurs with general atheroma (see ARTERIOSCLEROSIS). The vessels most involved are, in order of frequency, the carotid, middle cerebral and its Sylvian branch, basilar, vertebral and posterior cerebral. (*b*) *Syphilitic endarteritis* occurs in young subjects, 84 per cent. being found in persons between the twentieth and fortieth years. The initial proliferation may of itself, without thrombosis, obliterate the vessel lumen (see BRAIN SYPHILIS). The pathological sequence is the same as in embolism; ischæmia, necrosis (softening) and loss of function occur, save that in gradual thrombosis the focus is necessarily less homogeneous than in the sudden lesion of embolism. The foci are more often multiple, for example, in the Sylvian artery and in the posterior cerebral vessel of the opposite side.

2. Changes in the Heart.—Sudden cardiac insufficiency in arteriosclerosis may precipitate thrombosis, as brought out especially by Kolisko. An acute infection in the aged, low arterial tension from exhaustion, grief and similar causes may produce it when the arteries are ripe for encephalomalacia.

3. Changes of the Blood.—This constitutes the smallest class of cases. Burns, poisoning by carbon monoxide and illuminating gas, gout, chlorosis, pregnancy, leukæmia, metallic poisons, jaundice and marasmus in very young children or in extreme old age may cause thrombosis. Mechanical factors, acute infections and heart weakness are frequent factors in this class. Malarial thrombosis might possibly be included. Thrombosis affects the smaller vessels and is usually multiple.

Symptoms.—In some cases thrombosis is found at necropsy without previous symptoms ("latency").

1. **Prodromes.**—These are frequent, and may appear hours, days, and even weeks or months before focal symptoms develop. The most common are headache, which is severe in the syphilitic variety, vertigo, tingling, numbness and weakness in a limb or in one-half of the body, disturbance in sight, mental changes, defective memory, poor articulation, irritability, loss of appetite for work, change in character, and sometimes a tense pulse with weak heart.

2. **The onset varies**; (*a*) an *acute onset* with insult is not frequen [illegible] If consciousness is lost, the coma is less severe and protracted tha [illegible] in hemorrhage; a "stroke" indicates thrombosis in one large or i [illegible] several vessels. Conjugate deviation, flaccidity and paralysis ar [illegible] frequent during the coma. Consciousness is usually preserved i [illegible] the syphilitic type. With acute onset the later course is usually slow [illegible] (*b*) The onset may be *gradual*. Layer after layer is added to th [illegible] clot in this most frequent type, and strengthened heart action may fo [illegible] a time help the collateral circulation. When the heart weakens th [illegible] clot increases and the collateral blood supply decreases, so that, afte [illegible] a step-like course of advance and regression, the final thrombosi [illegible] with irreparable ischæmia and softening results. The cardiac strength [illegible] the number and size of the vessels involved determine the acuity o [illegible] chronicity of the course. *Reaction* may be marked, especially in th [illegible] aged. (*c*) In the *chronic* or long *latent* variety, prodromes are absen [illegible] psychic alteration is apparent and focal symptoms are usually d [illegible] veloped.

3. **In the chronic stage,** whatever the onset, focal symptoms [illegible] monoplegia, hemiplegia, hemianopsia or aphasia are common. Ap [illegible] plectiform seizures mark the advance of the process in new vascul [illegible] areas. The motor or sensory paralysis, at first transitory, is prone [illegible] recur, and presents much the same picture as hemorrhage or emboli [illegible] in their chronic stage.

Localization.—It is impossible to determine the localization wh [illegible] multiple foci exist, as in the Sylvian artery of one side, and the poste [illegible] cerebral artery of the other; to the few reported cases, Senator ad [illegible] one in which left-sided hemiplegia was associated with aphasia. T [illegible] favorite sites are the cortical branches of the Sylvian artery and [illegible] branches of the posterior cerebral vessels.

1. **Sylvian Artery.**—The most common cause of this localizatio [illegible] syphilis or atheroma. When the *main trunk* is involved, the sa [illegible] symptoms appear as in embolism, *viz.*: hemiplegia, aphasia, he [illegible] anæsthesia, and hemianopsia with this modification; the *local sym* [illegible] *toms are more severe* in thrombosis, because other vessels are somewh [illegible] diseased; the *general symptoms* are less severe. Prodromes appe [illegible] first as vasomotor changes and tingling, then hemiplegia possib [illegible] with delirium or loss of consciousness; the symptoms may improv [illegible] then there may be recurrence with aphasia, and coma. The cours [illegible] is a "step-like" progression and regression. *Monoplegia* is more common than in embolism, because thrombosis seeks the cortical vessels, whence the face and arm may be affected, aphasia may exis [illegible]

alone, or alexia, mind blindness or deafness. When the basal arteries are diseased, the vegetative functions are involved, that is, trophic changes with involuntary evacuations occur (*gâtisme*).

2. **Anterior Cerebral Artery.**—This artery is rarely totally occluded. A clot beyond the anterior communicating vessel often produces crural monoplegia with some paresis of the arm. Very *gradual anæmia* produces *wasting* without softening; sudden anæmia produces softening. The brain may atrophy three to seven ounces, with senile dementia, mania or hypochondriasis and though not strictly softening, its close arterial connection is obvious. The frontal lobes, especially below, present the "worm eaten" atrophy, with abundant *hydrops ex vacuo*.

3. **Posterior Cerebral Artery.**—Thrombosis in this artery is fairly common and may result in hemiplegia alternans oculomotoria, hemihypæsthesia, and hemianopsia with irregular defects in the crossed fields; optic changes vary accordingly as the optic radiation, cuneus or other parts are affected.

4. **Basilar Artery.**—If the thrombosis is acute, the symptoms are like those of embolism. If slow, ophthalmoplegia may develop which is rarely lasting, or other motor paralyses, hemiplegia with alternating palsy of the fifth, sixth, and seventh nerves.

5. **Vertebral Artery.**—(*a*) *Acute bulbar palsy*, with stormy symptoms, which cannot be distinguished from embolism; or (*b*) *subacute bulbar palsy*, with respiratory difficulty, *dysphagia*, paralysis of the larynx, disturbed articulation, hemianæsthesia or hemiplegia with crossed paralysis of the tongue may result from this localization.

Diagnosis and Differentiation.—The *prodromes*, *gradual onset* and *cortical location* are most distinctive, although the analysis may be greatly confused and differentiation of complicating conditions in the heart, and kidneys may be wholly theoretical or impossible. (See TABLE, page 988.) Thrombosis may be confused with *brain tumor*, in which the course is slower, stabile focal symptoms less frequent, irritative symptoms (headache, vomiting and convulsions) more frequent than permanent paralysis; and choked disk, Jacksonian epilepsy and periodic psychical manifestations are frequent. In this connection, we may consider *hemiplegia in the aged* and Marie's *les lacunes de désintegration cérébrale; degeneration clefts* occur, chiefly in the large basal ganglia, sometimes in the internal capsule or pons (25 per cent.), and but seldom in the centrum ovale (13 per cent.); there are usually several foci. Marie holds that the lesion is a rarefication (cavity formation) of the brain tissue due to chronic sclerosing *encephalitis*, *without vessel occlusion*. It is said to cause 90 per cent. of "insults" in the aged, which occur usually with *partial paralysis;* sometimes with aphasia, dysarthria or dysphagia; rarely with loss of consciousness; and never with sensory disturbance or contractures. The gait is the *démarche à petits pas*. The prognosis is fairly good; great improvement may occur and the patient may live eight to ten years though hemorrhage may intervene.

HEMORRHAGE.	THROMBOSIS.	EMBOLISM.
(1). *Predisposing etiology.* Before third year (glioma); 80 per cent. after fortieth year.	After eightieth year more frequent than hemorrhage. Twentieth to fortieth year syphilis (of which 84 per cent. occurs between these years).	Youth: hemorrhage practically never in youth.
Miliary aneurysms; atheroma, and cardiac hypertrophy.	Weak irregular heart; fevers, cachexia. Atheroma, especially syphilis, alcoholism, plumbism.	Endocarditis (rheumatism, sepsis) especially mitral. Weak, fatty dilated heart. In heart disease Eichhorst found in 60 per cent. hemorrhage and in 40 per cent. embolism.
Heredity.	Rare.	Rare.
Nephritis: cause in 33 per cent. "granular kidney."	Rare.	Rare.
(2). *Attack precipitated* by heavy meal, acute alcoholism, high art. tension (excitement, effort, shock).	Low arterial tension; by sleep, grief, exhaustion, fatigue.	By exertion, dislodging clot.
(3). *Prodromes* unusual, unless from causal arteriosclerosis.	Very common, mental irritability, weak memory, headache, vertigo, numbness, tingling, weakness in extremities.	Absolutely none.
(4). *Insult:* Usually sudden; without prodromata; long duration (fatal if more than two days); Cause: positive pressure producing brain anæmia.	Rarely acute onset, (unless in a large vessel); usually gradual, especially in senile softening. If an insult, focal and general symptoms usually appear during the coma.	Sudden, shorter and less severe than in hemorrhage. Cause: negative pressure. Slow insult only with secondary thrombosis.
(5). *Intra-cranial pressure:* "Pressure symptoms" present, polyuria, albuminuria, glycosuria, red face, beating arteries, Cheyne-Stokes breathing; slow, hard pulse is the fundamental difference (Brissaud); bruit de gallop speaks strongly for hemorrhage. Irregularity, if combined with strength not against hemorrhage. Pulse may be fast (Brissaud).	None. Pulse soft, weak.	None (pallor rather than congestion).
(6). *Temperature:* Rectal temperature usually depressed at first, may fall till death. May rise later (inflammatory reaction). Early rise (pons, medulla, ganglia). Sudden rise of bad omen; rises before death.	Initial depression, usually lasting. Seldom temperature unless lesion in pons. More probably early reaction. Especially in the aged.	Temperature may rise to high at once without bad omen. Temperature no absolute guide; occurs especially when large trunk embolised.
(7). *Coma:* More frequent, greater in degree, longer in duration, usually complete. If lesion seems small, coma speaks for hemorrhage. Deep and prolonged speaks for hemorrhage and usually fatal if over twenty-four hours.	Less frequent and shorter. Recovery possible after several days of coma. None in syphilitic thrombosis. If lesion seems large consciousness speaks for softening. If coma, usually focal symptoms are then seen. Coma in large bilateral lesions.	No coma usually.
(8). *Paralysis,* sudden, complete, hemiplegic, non-progressive after first seizure. Foot gains more rapidly than hand.	Gradual, incomplete, monoplegia, hemianopsia; repeated, often symmetrical paralysis. Converse.	More like hemorrhage. Monoplegia rare. In 63 per cent. it is in the left side of the brain.
Persistent aphasia exceptional. Spasmodic laughing and weeping common.	Aphasia common. Absence of focal symptoms rather more common (central ganglia). Laughing and weeping exceptional.	Fairly common.
(9). *Convulsions:* Usually generalized; very rare from cortical irritation or in corpus striatum; post hemiplegic are rare.	Less than in embolism. More localized, Jacksonian epilepsy speaks for softening. May recur. Postplegic frequent.	More common (33 per cent.) than in hemorrhage or thrombosis (lesion cortical).
(10). *Sensation:* Anæsthesia usually transient.	Paræsthesia persists and more marked. Cerebral pains (hyperæsthesia dolorosa).	Ditto.
Postplegic chorea, trembling, athetosis, common.	Less common.	Less common.
(11). Atheroma retinal arteries, hence retinal hemorrhage (not significant unless large). Aneurysms very rare but speak strongly for hemorrhage. Retinitis albuminurica.	Arteriosclerosis.	Optic retinitis sometimes from endocarditis (Broadbent and Mackenzie). Occasionally embolism in retinæ.
(12). *Secondary reaction;* not much.	Greater secondary reaction.	
Psychical symptoms; far less.	Often greater: intelligence, memory aphasia, word deafness, etc.	Very seldom; after attack may be some delirium.

sis.—The prognosis depends on (*a*) the *cause*. It is unfavor- *eriosclerosis,* because collateral vessels are probably diseased use is progressive. If the other vessels are sound, enormous ay be compensated, as in Kussmaul's case of occlusion of ubclavian and carotid. In cases of *recent syphilis* the prog- ore favorable, though old specific thrombosis is incurable. ger the patient, the better is the outlook. (*b*) It also depends *et.* A brusque beginning is unfavorable, because most cases ass die (Durand-Fardel). Coma indicates thrombosis in els or in both hemispheres, and is therefore ominous. If o insult, the focal symptoms are usually irreparable, *i.e.,* no indirect symptoms. The first seizure does not usually ife. (*c*) The *location and extent of the lesion.* The number involved is obviously important. In Sylvian thrombosis the branches to the central ganglia, cachexia and more sturbance occur and the patient dies in four to six weeks, untary evacuations and bed-sores (Marie). Cortical lesions, her hand, are more prone to produce psychical alteration. vertebral thrombosis are directly dangerous. (*d*) The *focal* are likely to *persist* and *recur.* Ophthalmoplegia may regress. ondition of other organs, above all that of the *heart,* is of import.

ent.—(*a*) The *general treatment* is the same as in hemor- embolism. (*b*) The *causal treatment* relates to the iodides, of value in atheroma (lessening the viscosity of the blood) pecific in early syphilis. (*e*) The *heart* must be stimulated hnine, digitalis and strophanthus; absolute quiet in the ition, and free administration of food and fluid by rectum asal catheter, are indicated. Drastics and all depleting must be avoided. On the heart devolves the entire pos- collateral compensation. (*d*) In the *period of reaction,* ould be avoided, for it accentuates the inflammatory reac- r delirium and headache, bromides are most useful; c depressants, chloral or coal-tar products may be used. *hronic stage* is treated as in hemorrhage. At all times ation must be conducted antiseptically and great care exer- nst bed-sores. In the use of baths, massage and electricity, s short of producing fatigue are indicated.

VII. Intracranial Aneurysms.

y.—Aside from miliary aneurysms, aneurysms may occur in rain vessels. They are seen in 0.2 to 1 per cent. of autopsies. more frequent in males (60 per cent.) and in middle age, they may visit either extreme of life and a few more (53 per ur before than after the fortieth year. The causes are those sm elsewhere (*q.v.*); they are (*a*) *atheroma* in whose causation alcohol is particularly important; (*b*) *trauma*, initiating arteritis, especially in the internal carotid, as after basal fracture (Killian); (*c*) *syphilis* especially productive of basilar aneurysm, which often occurs within a year after infection; (*d*) *embolism*, the most frequent cause in early life. *Endocarditis* is the usual cause when trauma and syphilis are excluded (Lebert and Church). The embolus produces arteritis from which the vessel dilates. The clot may later disappear. The Sylvian vessel is most frequently involved.

Pathology.—*Location.*—The statistics of Lebert, Durand, Bartholow (collected by Gowers), and Osler combined show the following frequency: middle cerebral, 29 per cent.; basilar, 26 per cent.; internal carotid, 14 per cent.; anterior cerebral, 8 per cent.; anterior communicating, 6 per cent. They are somewhat more frequent on the left side, in 20 per cent. are multiple, are usually sacculated, of the size of a pea or walnut (rarely large as an egg), and are genuine aneurysms involving all coats.

Symptoms.—Symptoms may be absent until rupture occurs. They are (*a*) *general*, as *headache*, which is the most common sign, or *convulsions*, which result from aneurysm of the Sylvian branch; optic neuritis is rare; a systolic murmur, neuralgia or epileptiform seizures may be noted. (*b*) They may be *local;* according to their location, hemianopsia, paralysis of the ocular nerves, impairment of smell, hemiplegia, optic neuritis (extension of inflammation from the sac), aphasia, and symptoms referable to the pons or medulla may be noted. *Rupture* occurs in 75 per cent. of cases; (i) into the meninges or (ii) into the brain substance or ventricles, from which death results in a few hours or days; healing has been observed.

Diagnosis.—*Rupture* is often the first symptom, and differentiation from hemorrhage is therefore necessary. A general cerebral hemorrhage in a young individual, with a history of trauma, heart disease or syphilis, is rather suggestive of aneurysm, especially when the location is basilar. If symptoms have been present, they are often those of a basal tumor; syphilis (*q.v.*, CEREBRAL SYPHILIS) and trauma are causes of tumor and aneurysm, but iodides and mercury usually are not beneficial in aneurysm. The murmur is suggestive but is rare and may be found in tumors which compress vessels or in very vascular brain tumors. The location of the aneurysm is important; they are most often embolic in the Sylvian, and specific in the basilar artery. Loss of sight in an eye, sometimes with impairment of smell, rather indicates aneurysm of the anterior cerebral or, if the eye muscles are paralyzed, of the internal carotid artery. Oculomotor paralysis without loss of vision suggests aneurysm of the posterior communicating artery. Some cases closely resemble arterial thrombosis.

Treatment.—The indications are the same as in aortic aneurysm (*q.v.*) *viz.*, rest and iodides. Surgical intervention is occasionally opportune, as ligation of the carotid.

VIII. Sinus Thrombosis.

The marantic form was known to the oldest authors, while Aber-mbie, von Dusch and Bright described the pyæmic form. Lebert 54) diagnosticated the first case and with Tonnelé built up its ical recognition.

Etiology and Pathology.—There are two varieties, in which prac-ally all cases can be placed, (1) the marantic and (2) the inflam-tory types.

1. *Marantic thrombosis of the sinuses and veins* is the *primary non-lammatory* type, due to altered blood states and weakened circu-ion. It occurs (*a*) in *children* mostly, especially in the first six nths of life, and after acute infections, but particularly after infective *rrhœas;* (*b*) less frequently in the *aged* from weak heart and venous sis; (*c*) in *cachexia*, from cancer, gout and tuberculosis; (*d*) in anæmia of *pregnancy* and in *chlorosis* (Sollier, Bollinger, König, ckler and Kochel); (*e*) *compression* of veins or sinuses as by tumor, rare. Its most frequent *site* is the superior longitudinal sinus, ecially in children, because the sinus is fixed, its lumen is triangular irregular, crossed by trabeculæ and compressed by the Pac-onian granulations; the tributary veins empty against the current he sinus, the veins ascend and their stream is very weak. Gowers ieves that thrombosis in veins entering the superior longitudinal us may cause hemiplegia in children. Experimentation seems to w that simple slowing of the current is not sufficient for coagulation, that the vessel wall must be altered; Bollinger holds that in chlo-is there is fatty degeneration of the endothelium. In the aged, rantic thrombosis rather elects the cavernous or transverse sinus. e older the antemortem clot, the lighter is its color, and the more minated and adherent it is to the sinus wall, wherein it differs from e reddish, loose and separable postmortem clot. The marantic ot may be total, occluding the vessel, or may be partial. It may be ort, or may fill the whole sinus and enter the tributary veins which e then seen as firm cords over the hemispheres.

2. The *inflammatory thrombosis, thrombophlebitis*, is *secondary* to sease near the sinus; as (*a*) *ear disease*, chiefly chronic, sometimes ute, suppurative otitis media, *caries* of the bone (Fuchs), sometimes philis, tuberculosis or cholesteatoma. Infection from the ear reaches e sinus by (i) infecting the mastoid cells, which infect the transverse nus; (ii) by the passage of the virus from the middle ear to the nus by infecting small communicating veins (Politzer) or (iii) its ssage along the petromastoid canal (Voltolini). In 81,648 ear ses reported by Hessler, 116 fatalities occurred, of which 34 per nt. was meningitis. It may be secondary; (*b*) to *acute infections*, ith otitis media; (*c*) to *sepsis*, puerperal fever, pyæmia; (*d*) to *trauma*, berculous *meningitis*, erysipelas, disease of the orbit, nose, gums, nd face. The most common *sites* are the transverse, petrosal and

cavernous sinuses, whence it may extend to their branches. The c
is puriform, fœtid, soft, discolored and contains pyogenic organism
the process is frequently associated with meningitis and brain absc

In both types, *brain changes* may result, as œdema, swelling
softening of the cortex. Most frequently the thrombosis is bilate
suggillations in the pia, bloody imbibition of the brain or redden
of the cerebrospinal or ventricular fluid may occur. Pulmon
infarcts occur in 50 per cent., and are simple or septic according
the type of thrombosis.

Symptoms and Diagnosis.—Clinical signs may be absent or
scured by the primary affection. Diagnosis depends upon signs
(1) extra- and (2) intracranial venous stasis.

FIG. 59.

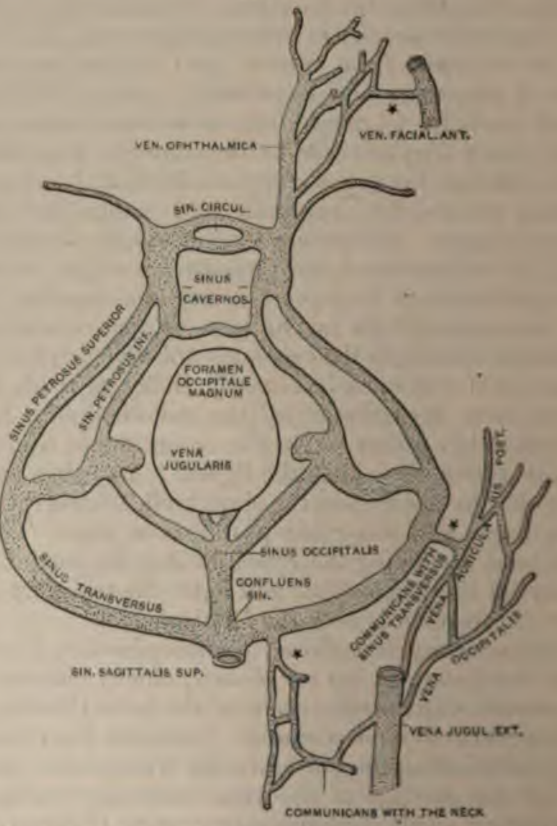
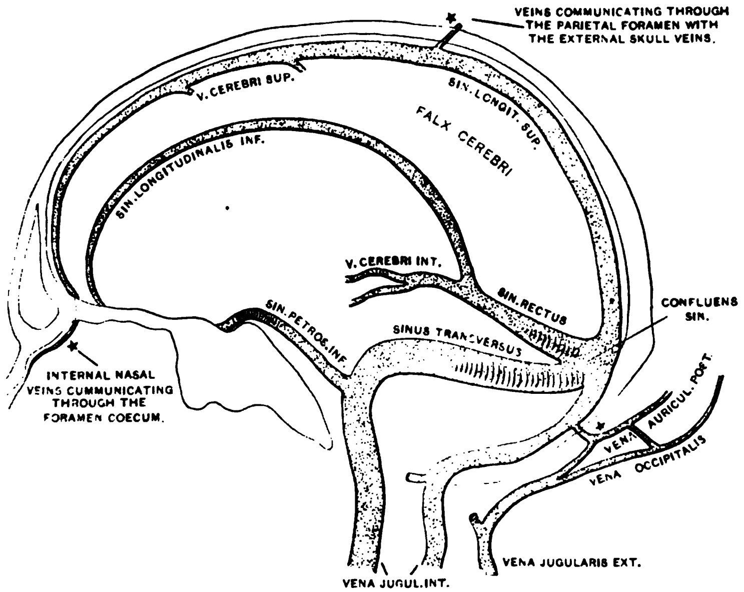


Diagram showing the connections * of the transverse and cavernous sinuses (Leube).

1. Extracranial Stasis.—(*a*) In thrombosis of the *superior long
tudinal sinus,* stasis in the nasal veins is often shown by *epistax*
(connection through the foramen cæcum): this was first observed
von Dusch. It is an effort of nature to deplete the cerebral congestio
Gerhardt observed *cyanosis* in the anterior frontal veins and *œdem*

ecially in children, in the temples between the great fontanelle l ear (by way of the emissaria Santorini through the foramen ietale). Œdema is often absent in chlorotic thrombosis. Sweats r the forehead, head and neck (Fritz) are of uncertain diagnostic ue. (*b*) In thrombosis of the *cavernous sinus*, stasis develops in its ıutaries, producing *exophthalmos*, *œdema of the lids* and conjunctiva na ophthalmica); *retinal stasis*, more rarely choked disk (Milligan) retinal thrombosis (Bouchut), results from congestion of the vena ıtralis retinæ. Sometimes œdema of the forehead results from sis in the vena facialis ant. Epistaxis is rare (Stäger). (*c*) In ombosis of the *lateral sinus*, a hard, tender *œdema behind the* (Griesinger) and *in the neck* (communicating with the posterior ·icular vein) occurs in over 50 per cent. of cases. Occasionally *swollen thrombotic internal jugular vein* may be felt. The *external*

FIG. 60.



Leube's diagram showing the connections of the superior longitudinal sinus with the external veins.

ular frequently collapses, or if the internal jugular is thrombosed, ›ecomes overfilled (Gerhardt). In 33 per cent. the disk is hyper- ıic, and in another 33 per cent. there is choked disk (Jansen) m complicating abscess or meningitis. (*d*) In thrombosis of the *us petrosus*, the signs are conflicting and Gowers questions whether ' are distinctive.

. **Intracranial Stasis.**—Progressive clouding of the mind, delirium ıdults, convulsions in children, conjugate deviation of the eyes and d, stiff neck, vomiting, headache, irregular pupils, nystagmus or strabismus are noted. Hemiplegia and sensory symptoms are occasional. Bilateral symptoms, as paraplegia, are due to the involvement of the veins in both hemispheres or more often to meningitis. Bouchut thought that antemortem convulsions in children were often thrombotic in origin. It will be seen that many of these symptoms resemble the *hydrencephaloid* (Marshall Hall) of brain anæmia. The fontanelles, sunken at first in infantile diarrhœa, may later become prominent (increased cerebrospinal fluid). Sometimes in chlorotic thrombosis the process may extend to the vena Galeni magna, producing hydrocephalus internus and resulting in early stupor, coma and death. Irritation or paralysis of the third, fourth, and sixth nerves may occur because of their intimate relation to the cavernous sinus; involvement of the first branch of the fifth nerve may result in ophthalmia neuroparalytica. Rarely is there paralysis of the hypoglossus and spinal accessory.

Other symptoms are those of the causal affection. The temperature is often normal in the simple variety, or high in the death agony. The pulse is slow at first and rapid later. The thrombophlebitic variety is usually attended by fever, chills, sweats, diarrhœa and other septic manifestations.

Differentiation from meningitis (*q.v.*) and brain abscess (*q.v.*) is often impossible; the cause, the extracranial stasis, and evidences of sepsis are of diagnostic importance. Körner held that metastases occur in the lungs from sinus thrombosis and in the bones and joints from mastoid caries.

Prognosis.—The prognosis is almost always unfavorable; death occurs in the second week, or possibly later. A few recoveries from both types are on record. Local atrophy of the brain has resulted after recovery from the marantic type.

Treatment.—*Prophylaxis* relates chiefly to the otitic variety, because discharges from the ear are much too lightly regarded by the laity. In *marantic thrombosis* stimulation and avoidance of constriction of the neck are the chief indications. In *septic types* operation is indicated; recovery is most likely when there is early diagnosis and early operation. Opening and packing the lateral sinus was first performed by Zaufal in 1884. He also recommended ligature of the jugular vein, which was first performed by Thersley and Lane in 1888. Recovery occurs in 50 per cent. of operated cases and in 72 per cent. of Macewen's series. Recovery has occurred even with complicating lung abscess (Ballance). Gravitating pharyngeal abscess and extradural foci also necessitate surgical interference.

IX. Infantile Cerebral Paralysis.

Cerebral infantile palsy is classed under circulatory diseases because of the frequent etiological hemorrhage, embolism and thrombosis. Pathologically it is sometimes described under encephalitis, sclerosis

orencephalia. Clinically it is variously classed according to the
inant syndrome, hemiplegia, diplegia, Little's disease, paraplegia,
iathetosis, and athetosis. The disease is a clinical picture, but
varying etiology and pathology. Osler, Sachs, Strümpell, Freud
Rie have contributed most valuable monographs.

he **Hemiplegic Form.**—Etiology.—It is equally frequent in either
Most cases occur in the *first year*. Gowers finds 60 per cent.
e first two years, 75 per cent. in the first three and 88 per cent. in
first five years of life. *Infections*, as scarlatina, measles, less fre-
ntly variola, pertussis, or diphtheria are apparent, and heredity,
ernal syphilis or trauma are doubtful, causes. The causes may
ate before, during, or after birth. The pathology is considered
er the next type.

mptoms.—(*a*) Initial symptoms, present in 66 per cent., are
re in character, consist of fever, vomiting, convulsions, delirium
coma, and last from a few hours to several days, sometimes longer.
vulsions, present in 50 per cent., are often unilateral and are usually
wed by coma. In 33 per cent. of cases the onset is insidious. (*b*)
paralysis (hemiplegia) usually develops when consciousness re-
s. It is more frequently right- then left-sided. The lower face
volved, but usually soon improves, the leg improves more than
arm and hand, and the paralysis, at *first flaccid*, soon becomes
tic and is usually permanent. In general it resembles adult hemi-
ia (See Next Stage). Sometimes the eye muscles are involved.
hemianopsia may be noted. Although the tactile and stereognostic
e may be somewhat reduced, sensation is but slightly implicated,
use the other hemisphere compensates. (*c*) The *residual symp-*
are the same as in adults, with some exceptions; the paralysis
astic (spastic infantile hemiplegia, Heine) but also *atactic* and
totic. Because is occurs in the young growing body and brain,
ked *trophic changes or, at puberty, retarded development* appear,
lection is retarded or actual idiocy results, and *epileptiform seizures*
common. *Contractures* are frequent, the *reflexes* are increased,
the gait is somewhat impaired, the foot dragging. In rudimentary
nes frustes) or benign cases, the paralysis may almost disappear
after months or years may be so replaced by epileptic seizures,
iathetosis, hemichorea, hemitremor (described under hemorrhage)
emihypertonia (rigidity with but little paralysis) that the paresis
ily escapes recognition. *Aphasia* is rarely lasting. It must be
lled (*a*) that *reflex aphasia* is very common in childhood from
r or digestive difficulties; (*b*) that the brain readily compensates
ildhood; (*c*) that permanent aphasia indicates great or total loss
telligence. *Trophic changes* are in part a moderate atrophy and
rt lack of later development, though seldom so marked as in the
l type of paralysis. They are noted in the extremities, body,
times the face and in the skull which often thickens; they are
common in porencephalia (*v. i.*). *Epilepsy* increases in fre-
cy after the paralysis, and is rated at from 13 per cent. to even

66 per cent.; it occurs (*a*) at the onset, (*b*) with the paralysis, or (*c*) a year or two later; it is not commensurate with the degree of paralysis but is more marked in rudimentary palsy; it may be slight like a vertigo; it is most often like the Jacksonian type, but may end in total loss of consciousness. In infantile cerebral paralysis there is less violence, initial cry, trauma, foaming at the mouth, and fewer involuntary evacuations than in ordinary epilepsy. After a decade or more it often becomes very marked, although it has been known to disappear between the fortieth and fiftieth years.

DIAGNOSIS.—The chief differential interest centres in its separation from the spinal type (poliomyelitis):

	CEREBRAL TYPE.	SPINAL TYPE.
Tendon reflexes.	Increased.	Decreased or abolished.
Muscular atrophy.	Only after a long time.	Rapid.
Electrical reaction.	Normal, or little quantitative decrease.	Partial or total reaction of degeneration.
Distribution of paralysis.	Hemiplegic—diplegic.	Chiefly monoplegic.
Intelligence.	Often involved.	Usually not.
Epilepsy and hemichorea.	Common.	Rare.

The Double Hemiplegic or Diplegic Form.—ETIOLOGY.—(*a*) *Pre-natal causes* are apparent in 7 to 31 per cent. and include fright, trauma, malnutrition or syphilis of the mother. These causes seem most common in cases which develop athetosis. (*b*) *Anomalies of labor*, suggested by Little and proven to be as a factor by Sarah McNutt, include difficult or long labor, malpresentations, resistant maternal tissues, twin pregnancy, version, cord prolapse and instrumentation, although delivery by forceps is less important than the causes calling for their use. *Asphyxia* is an important etiological factor. Premature delivery is also causal, possibly because the soft skull is injured (Gowers) or because venous stasis occurs from precipitate delivery. (*c*) *Extra-uterine causes* are indefinite, and include trauma and acute infections.

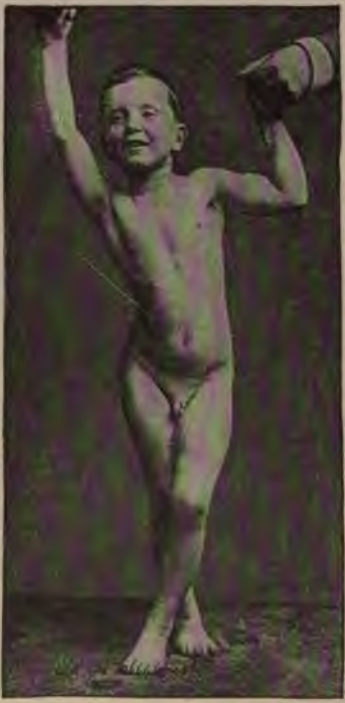
PATHOLOGY.—The pathology presents many obscure points. The *initial lesion* cannot always be determined. (*a*) In the *traumatic class, meningeal hemorrhage* causes 66 per cent. *of the diplegic cases*; Sarah McNutt found hemorrhage spreading over the motor cortex on both sides. It is promoted by *asphyxia* and the sliding of the skull bones of one side under or over those of the other side; hemorrhage is often basal in head presentations, and on the convexity in the after-coming head. The blood is absorbed slowly, especially if the brain is lacerated, resulting in atrophy of the motor areas of both sides hemorrhage rarely results from hereditary syphilis. (*b*) *Vessel disease* (18 per cent.) may be arterial (thrombosis, embolism or hemorrhage) or venous (in the cortical veins, Gowers). (*c*) Strümpell advanced the views of Vizioli and Marie that *acute encephalitis* in the motor cortex (poliencephalitis) causes the acute onset, an argument largely based on analogy (poliomyelitis in children), though seldom verified.

Chronic Lesions.—The most important are diffuse lobar sclerosis, and porencephalia, which are peculiar to infantile brain palsies: (*a*) *Diffuse lobar sclerosis* and atrophy of the brain occur in 55 per cent. (Osler). The meninges are normal or somewhat thickened. About the atrophic areas and in the ventricles the cerebrospinal fluid is increased. One or both hemispheres may be involved but usually unequally. The lobe affected is shrunken, hardened, leathery and the white matter is decreased, while the gray seems increased. The convolutions are much wasted and are smooth or "worm eaten." The nervous tissue is wasted, the neuroglia increased, and the blood-vessels tortuous, somewhat thickened and their perivascular lymph spaces widened five- or six-fold. The nature of the initial lesion is disputed; it is thought to be encephalitis (Bourneville), poliencephalitis (Strümpell) and *embolism.* (*b*) *Porencephalia* (Heschl, 1859) consists of loss of brain substance, and cavity formation. It occurred in 26 per cent. of Osler's cases. The nature of the initial lesion causing porencephaly is a matter of doubt; it is ascribed to anomaly of development (agenesis), inflammation (encephalitis or meningo-encephalitis), trauma (during labor or to the uterus during pregnancy) or *anæmic necrosis* (Kundrat); the latter is the most widely accepted view. It is most frequent in the distribution of the Sylvian or occipital vessels. Most often it is congenital and then the convolutions radiate from the defect, while in those cases resulting *post natum,* the convolutions stop short of the defect "as though cut off." Bourneville and Sollier hold that the congenital type communicates with the lateral ventricle, while the acquired form does not. Sixty-six per cent. is bilateral and somewhat symmetrical. (*c*) The *hypertrophic nodular sclerosis* described by Bourneville consists of multiple discrete prominent, often umbilicated nodes, the size of a quarter or half-dollar. They are overgrowths of the neuroglia.

Symptoms.—Diplegic cases are half as frequent as the hemiplegic. Freud describes the following forms: (*a*) *General rigidity,* or Little's disease, 33 per cent. of which is caused by asphyxia. The main characteristics are (i) *predominance* of the *rigidity* over the paresis, and (ii) *predominance* of the *leg paresis* over that of the arms, the converse of adult cerebral paralysis (hemorrhage occurs most often over the leg centres). Convulsions often usher in the symptoms, which appear after birth or a few months later. The rigidity is general but most conspicuous in the *flexors and adductors,* leading to unusual postures. The face is slightly involved, the maximum manifestation being spasm of the mouth; in rare cases the child cannot nurse or swallow. Speech is slow in development and stuttering is common.

In 33 per cent. the head cannot be lifted or the trunk flexed; this is due to involvement also of the uncrossed pyramidal tracts. The arms may be but slightly paretic, but the legs suffer extensor spasm on movement, and the toes turn down (talipes equinus or equinovarus). The child learns to walk late, if at all, because of the adductor rigidity, which may even cause crossing of the legs. The reflexes are greatly increased. The intellect is somewhat affected in 66 per cent., but nutrition in the paretic members is usually intact. Later epilepsy is uncommon. The paresis may regress and the mind improve, but the legs remain spastic. (*b*) *Paraplegic rigidity* resembles type (*a*) except that the arms are but little or not at all affected and the legs alone are rigid. The name Little's disease is also given to this form. Strabismus is common (40 per cent.). Premature delivery is a factor in 50 per cent. of cases. (*c*) *Paraplegia* is rare and is referred to extra-uterine causes or to infantile infections. The legs are rigid and paralyzed. In severe types there may be strabismus and imbecility. (*d*) While *bilateral spastic hemiplegia* (spastic diplegia) exists, the symptoms may vary on the two sides. Mental degeneration is frequent; nystagmus, convergent strabismus (25 per cent.), bulbar symptoms (Oppenheim) and speech involvement (60 per cent.) are frequent. (*e*) *General infantile chorea* is also called "choreatic diplegia"; asphyxia is a factor in 25 per cent. Its clinical characters are (i) paralysis, (ii) rigidity, (iii) chorea-like movements which are irregular, usually slight in degree and wide in distribution. (*f*) In *bilateral athetosis*, the mind is less involved than in the preceding types. The paralysis may precede it or it may be primary.

Fig. 61.



Spastic paraplegia, cross-legged progression. (Dercum.)

Forms resulting from premature delivery are less subject to convulsions, athetosis or mental changes, and are more regressive than those due to birth trauma or later acquired lesions. (See Diseases of the Spinal Cord for the allied Family or Hereditary Forms, as Hereditary [Friedreich's] Ataxia). In all forms epilepsy may (i) replace the regressing paralysis; (ii) remain so prominent that the paralysis is overlooked; (iii) paralysis may appear only after years of epilepsy, or (iv) may not appear at all, "cerebral paralysis without paralysis."

DIAGNOSIS.—The diagnosis cannot always be made at once, although ter, characteristic symptoms develop and leave little diagnostic fficulty. Separation from the family spastic paralysis (*q. v.*) may be fficult or impossible. As a rule, aside from disease of the spine hich compresses the cord, confusing spinal cord disease is very re in children.

TREATMENT.—Therapy is most unsatisfactory. In the initial stage, nvulsions may be treated by chloral or inhalation of chloroform. me regression in the paralysis is frequent, but the hemiplegia, mental anges, and epilepsy are incurable. Marked improvement can be pected in the syphilitic cases alone. Orthopedic devices are valuable. he resulting epilepsy is usually intractable, and bromides are of tle avail. Flechsig advises their use, in combination with opium. perative interference has been suggested. Twenty-five per cent. die om the operation, but the surviving cases seem to improve, especially regard to the convulsions.

(C). BRAIN TUMORS.

Etiology and Pathology.—Eichhorst found brain tumors in $\frac{1}{8}$ of 1 per nt. of his cases. Sixty-six per cent. of tumors occurs in the *male sex*, pecially glioma and tubercle, while sarcoma occurs equally in the two xes. *Age.* In the first decade, 18.5 per cent. of cases is observed; in the cond, 14 per cent.; third, 20 per cent.; fourth, 18.5 per cent., and the fifth, 14 per cent. (Gowers). The causation is that of neoplasms general, as developmental errors, granulomata, metastatic growths, d parasites. Trauma is a certain but infrequent cause; it may clearly use growths, especially glioma, but may act as the "*agent provocateur*" gumma, sarcoma, osteoma, tubercle or aneurysm. The author has en endothelioma of the dura develop years after and directly at the at of an unhealed skull fracture. Syphilis may induce sarcoma.

Classification.—*Classification* is difficult.

1. **The Infectious Granulomata.**—(*a*) *Tubercle* constitutes 50 per nt. of cerebral tumors, 50 per cent. being found in the first, and 75 er cent. in the first and second decades (Gowers). The subject is sually poorly nourished, and often exhibits lung changes; 33 per nt. occurs in the cerebellum, 33 per cent. in the cerebrum and the mainder in the pons, central ganglia, crus, medulla and corpora uadrigemina. With equal frequency they are single or multiple, and ay number even 20 or 80. They originate from the meninges or essels of the brain substance, and are firm, rather rounded tumors, ith grayish-red periphery and yellow caseated centre. Tubercles nge from the size of a pea or walnut to that of an egg or of the fist, om fusion of many small growths. They grow along the lymph essels, cause vascular thrombosis, present the degeneration of tubercle f other tissues, and may be shelled out of the brain tissue, which is mpressed and atrophied, but neither infiltrated nor softened. (*b*)

Gummata (see BRAIN SYPHILIS) occur far more frequently than statistics indicate, for they respond to treatment and often disappear spontaneously. In the adult they are the most common type of tumor. They are most frequent in the cerebral hemispheres and pons, and are rare in the cerebellum and central ganglia. Though they are most often the size of a pea or hazelnut, larger dimensions are observed. Their centre is yellow-gray, and their periphery is reddish-gray, gelatinous, rich in cells and ultimately cicatricial. Gummata are usually multiple. The adjacent brain tissue is not infiltrated, but commonly softened and compressed. Microscopically they closely resemble tubercle, but Virchow described them as more nodose, angular, irregular, more distinctly connected with the dura even when deeply located, more gelatinous and less caseated. Tubercle bacilli are not always found in unmistakable tubercles, and gummata are sometimes miliary in size. Both forms have been confused with actinomycosis.

2. Neuro-epithelial Growths.—(*a*) *Glioma* constitutes 25 per cent. of brain growths; 50 per cent. occurs in the hemispheres, especially the cortex; 25 per cent. in the cerebellum, and the balance in the central ganglia, pons, medulla, crus, and corpora quadrigemina. They occur more frequently in the brain than in the cord or retina, to which three structures they are peculiar. Gliomata are usually (90 per cent.) single, and are seen most frequently in adults (first to twentieth year, 20 per cent.; twentieth to fortieth year, 50 per cent.; fortieth to sixtieth year, 25 per cent., Gowers). Some classify glioma with sarcoma. Gliomata originate largely in the gray substance, which they so closely resemble in tint that detection is difficult at operation, or indeed at autopsy, where secondary hemorrhage or softening in them may be confused with primary hemorrhage. Cases formerly called brain hypertrophy were probably gliomata. Their size varies from that of a pea almost to that of a hemisphere. Glioma infiltrates without sharp demarkation rather than compresses the brain tissue, and does not form adhesions. The cells resemble the neurogliar structure and usually have round or oval nuclei, although Klebs found ganglionic cells. Their processes are fine; numerous and branched delicate band-like lines result from fiber degeneration. (*b*) The rare *cerebroma* of Hayem was held by Virchow to be heterotopic gray matter in the white substance. (*c*) *Adenoma* of the pineal gland and hypophysis may be brought under this heading as also may (*d*) growths from the plexuses, and (*e*) neuromata.

3. Connective Tissue Tumors.—(*a*) *Sarcoma* is the most frequent, and constitutes 20 per cent. of brain tumors; it may develop in the meninges, periosteum or vessels. It is most common in adults. Though it infiltrates more than tubercles or gummata, it does so less than would be expected. Many are well demarked, and cause more softening than infiltration. Its structure and degenerations are those of sarcoma elsewhere. Though it is most commonly single, diffuse or multiple sarcomatosis is sometimes encountered. Endothelioma (Zieg-

, cylindroma (Billroth), angiosarcoma and melanosarcoma are but
eties. The sand tumors (psammomata) of the pineal gland, Pac-
onian bodies and choroid plexus are sometimes described as angio-
c sarcomata. The "pearly tumors" (cholesteatomata) are formed
at endothelial cells. (*b*) *Benign connective tissue growths* (lipoma,
oma, and osteoma) are very infrequent.

Metastatic Tumors.—Cancer is not infrequent (7 per cent. of
n growths), especially from mammary or lung carcinoma, just as
g suppuration often secondarily involves the brain. It often invades
hemispheres and basal ganglia, and may attain huge dimensions.
e sarcoma, it infiltrates. Cancer is very rarely primary.

Cystic Tumors.—Cysts may result from hemorrhage or softening,
eneration in neoplasms, infantile inflammations or defects (poren-
halia), trauma, or inflammation in the choroid plexus. *Dermoids*
teratomata are most rare. *Parasitic cysts* occur largely in persons
ween the tenth and thirtieth years; *echinococcus* cysts may be
le or multiple; they occur chiefly on the surface of the brain, or
he meninges, and vary much in size. *Cysticercus* cysts develop
stly on the pia and arachnoid over the cortex along the sulci, or in
ventricles. They occur in $\frac{1}{10}$ of 1 per cent. in certain autopsies,
ugh Diamond (1899) could collect but 8 cases from the American
ature; they vary from the size of a pea to that of a hazelnut, are
ally multiple (200 in Snell's case), and contain clear fluid with
k dots, the heads of the parasites. A fibrous capsule frequently
elops, and calcification may occur later. The cysticercus race-
us may reach a great size.

Aneurysms.—Aneurysms have been considered under diseases
e brain vessels.

ocation.—The location in Gower's figures was:—cerebrum, 42
cent.; cerebellum, 25 per cent.; base, 11 per cent.; pons, 8 per
.; central ganglia, 7 per cent.; medulla, 4 per cent.; corp. quad-
mina, 2 per cent.; crus, 1 per cent.

ymptoms.—In rare instances, tumor is found at autopsy without
ical symptoms (*latency*). This is due to location of the tumor in a
lent" or tolerant brain area; it may occur in tuberculous menin-
s, mental disease, aneurysm, cysticercus, psammoma, cholesteatoma,
meningeal tumors, which slowly compress the brain. As a rule,
ptoms result from the following *mechanisms:* (*a*) *Direct pressure*
ted on the brain, which may flatten the convolutions, obliterate
sulci, force open the skull fissures in children, absorb or perforate
cranium. Pressure is conspicuous in growths of the posterior fossa
ed by the rigid tentorium. (*b*) *Internal hydrocephalus,* distention
he ventricles, may follow closure of the aqueduct of Sylvius, or com-
ssion of the veins, particularly of the choroid plexus and the vena
leni magna, and especially by tumors of the cerebellum and corp.
drigemina. (*c*) The *brain* may be softened by pressure on its vessels
uveret), inflamed, or hemorrhage may develop (as in glioma). (*d*)
iltration or destruction of the brain matter may result. (*e*) *Menin-*

gitis, local as in gumma, or general as in tubercle, may develop. (*f*) *Auto-intoxication* from the growth is possible, as suggested by Klippel. (*g*) Distortion of the cerebral or spinal nerves (Dinkler, Mayer), may cause their degeneration, or changes in the posterior columns of the cord and its posterior roots.

The symptoms produced are *general* (diffuse), and *focal* (local).

The general symptoms usually come first in the evolution, and include the following:—(*a*) *Headache* is present in almost all cases, is usually constant, with accessions (especially in the morning), is general, rarely unilateral, is severe and obstinate enough to keep the patient awake at night, is increased by alcohol, exertion and coughing, and rarely corresponds with the tumor's site except when the pain is persistently local and the tumor near the surface. (Frontal headache often results from tumor in the white substance of the frontal and parietal areas, but sometimes may result from tumor anywhere, even in the cerebellum, which more often is attended by occipital headache or neck-ache.) Headache is usually referred to irritation of the dural branches of the fifth nerve, an explanation which is not always satisfactory. It may be pulsating in aneurysm. (*b*) *Choked disk* is the most important general symptom, and occurs in 80 to 90 per cent. It consists of a grayish-red swelling of the disk, to even two or three times its normal dimensions, which lessens its sharpness of outline and physiological cupping; the arteries become less lustrous and smaller, the veins turgid and tortuous, extravasations of blood and white patches of fatty degeneration or exudation appear. (See Plate v, fig. 3.). It develops in weeks to months, and is due to increased intracranial tension, which causes mechanical hydrops of the optic sheath (Schmidt-Rimpler, Matz, Schwalbe), or to inflammation from choking of the veins by toxic products (Leber and Gowers). It is bilateral (90 per cent.), although more marked on the side of the lesion, is not an early symptom and depends neither on the size of the tumor nor on its location. It is almost always present in growths of the cerebellum and corp. quadrigemina, is less frequent in cortical and meningeal localizations, and is often absent or appears later in those in the pons, medulla, and corp. callosum. There is often, but not always, limitation of the field of vision (scotoma for color and lessened acuity of vision). In many cases it develops into atrophy, oftener still into stasis and blindness, while in some it may regress or very rarely recur. Transitory *amaurosis* results from compression of the optic tract or chiasm by the distended third ventricle. (*c*) *Convulsions* occur in 50 per cent., are called the "signal symptoms," by Seguin, and may be general; general tonic precede clonic convulsions in no special order; the pupils react poorly, the skin reflexes are increased unless deep coma develops, the tendon reflexes are not suspended, respiration is labored and coma occurs. Convulsions are more frequently late than early. Slight seizures, like the *petit mal* of epilepsy, may occur. Jacksonian epilepsy belongs to the focal symptoms. (*d*) *Vomiting* is usually an early general symptom, indicating irritation of the vomiting centre (it is sometimes [illegible]

al symptom). It is of the cerebral type, occurring without effort,
usea, or pain, often on an empty stomach, as in the morning, or at
ce after eating, regardless of the kind of food ingested; it is initiated
sitting up, turning of the head, or excitement. Later the tongue is
en coated. Vomiting is closely associated with headache and ver-
o, and is due to intracranial pressure or meningeal irritation. It is
rely absent in growths of the cerebellum or medulla, is very frequent
disease of the corp. quadrigemina, and occurs in 25 per cent. of
mors in the hemispheres. (e) The *sensorium* is often involved.
he most common, and sometimes the earliest, change is dulness,
hich often deepens into stupor and coma. It is not surprising that
mors affecting the association fibers and avoiding the motor centres,
ay cause disturbance of intelligence, judgment, or memory, only;
y are frequently discovered at autopsy in the frontal or temporo-
enoidal lobes in subjects committed to asylums, for psychoses or
eral paralysis. Hysteria may occur in subjects of organic brain
ease. (*f*) *Vertigo* is often an early symptom; it may be mere "giddi-
s," or it may affect the labyrinth, pons, or cerebellum, and be a
se of actual rotation. It is present in 31 per cent. (Mills and Lloyd),
urs especially in the locations mentioned and as a focal symptom,
en the middle cerebellar peduncle is implicated. (*g*) *Slowed pulse*
y result from direct, but more often from indirect, pressure on the
gus fibers behind and lateral to the hypoglossal nucleus. The vagus
ers become accustomed to pressure of long standing and the heart
e increases. It is often associated with yawning, hiccough, Cheyne-
kes breathing, glycosuria, or polyuria. Cardiac disease, cholæmia,
d anæmia must be excluded.

Focal Symptoms.—Focal symptoms may be absent when the disease
limited to "silent," or "tolerant" areas, when the tumor does not
stroy nerve structures, and when vicarious compensation by other
rts is possible. Focal symptoms of irritation or paralysis may result
rectly from actual lesion of a centre or its fibers, or indirectly from
listance pressure" upon them. (They are, therefore, most signif-
nt when no particular increase of intracranial tension exists.)

1. Frontal Lobes.—The reported changes in mentality are much
cussed; early observers, as Bernhardt, claimed that mental changes
re no more frequent in frontal localization than in localization else-
ere. Many cases show the symptoms indicated above. Jastrowitz
scribes a condition designated as moria, a dementia with character-
ic irritability and excitement. A tendency to make jokes (Witzel-
cht) is described by Oppenheim. Psychical changes, especially
ose developing early, justify a "probability" diagnosis only. Allan
arr records the first case in which diagnosis was confirmed by opera-
on. "The two prominent characteristics of the mental state are
pathy and suffering, which may be associated or alternate" (Church).
arly or late mental alteration sometimes follows other localizations.
ometimes tumors in the frontal lobes are thought to be in the motor
reas from indirect pressure upon the latter. The head and eyes may

deviate, and the neck and back are sometimes rigid. Bruns has recorded frontal tumors with cerebellar ataxia, which is caused by unequal innervation with disturbance of the sensorium (Oppenheim), or involvement of the trunk centres (Strümpell). Some would associate with frontal lesions a lack of initiative. The motor centres in the left lower frontal convolution may be directly or indirectly involved. Bradyphasia (slow speech) is probably psychical, and differs from the separation of syllables sometimes seen in tumor of the pons. Oppenheim speaks of "laryngeal aphasia," in which the patient moves his lips and attempts to speak, but finds the voice lacking.

2. Motor Cortex.—(*a*) *Jacksonian convulsions* are often (50 per cent.) dissociated, and follow a given sequence (see Cortex Localization); consciousness is often preserved unless the other side of the body is involved. The convulsions may be few, with long intervals, or may run into the hundreds (17,000 in eleven months, as reported by Gowers). They may be preceded by the general convulsions described above. When the conducting fibers are destroyed, they cannot occur, whence they may be observed in the part nearest the seat of greatest damage. As long as convulsions are present, the motor centres cannot be wholly destroyed. Tapping the tendons or the skull may initiate spasms, and it has long been recognized that convulsions can sometimes be aborted by electricity, by grasping the arm, etc. (*b*) *Cortical paralysis* or paresis is usually monoplegic, and is associated with cortical spasm. It is usually slight at first, for a weakness from exhaustion follows the spasms; later it becomes more marked. In some cases paresis (or total hemiplegia) may be the first sign of tumor, and sometimes may be most narrowly localized, as in the thumb alone, the four fingers, or great toe; paralysis is accompanied by increased reflexes, rigidity, and rarely by muscular atrophy. Surgical extirpation has relieved both spasms and paralysis. In infrequent cases the paralysis may be bilateral, as when a cortical tumor near the median line presses on the opposite hemisphere, or when there are bilateral symmetrical growths; these, however, are still rarer. (*c*) Sensory changes are less frequent, because the sensory area is larger than the motor area, and extends posterior to it, and it is possible that sensory representation is bilateral. (The gyrus fornicatus is thought by some writers to be the seat of tactile and muscular sensibility.) Hypæsthesia, especially for tactile and muscular sensibility and paræsthesia, are not rare. Cortical pains are rare. Vasomotor symptoms, tachycardia, and salivation (also in tumors of the frontal lobe), are observed, but are not localizing signs.

3. Parietal Lobes.—Some sensory disturbance is fairly common. Incoördinate movements, ataxia, and athetosis have been observed. Wernicke observed a tumor in the lower parietal lobe with conjugate eye movements, and Weil noted incoördination of the extremities on the opposite, and of the eyes on the same side of the body. Sensory aphasia, hemianopsia, deafness, and word deafness are also observed.

4. Temporal Lobes.—Symptoms are likely to be confused with those of the contiguous occipital lobes; hemianopsia, disturbance of eye

vements, chiefly toward the opposite side, ptosis, and agraphia are orded.

OCCIPITAL LOBES.—Hemianopsia is the most constant finding; esults from any occipital lesion large enough to compress the optic liation in the white substance (Henschen). Optical hallucinations thought to indicate a cortical occipital localization rather than one the optic radiation. Alexia, optic aphasia, and psychical blindness seen.

PONS.—Focal may precede the general symptoms; they have n enumerated under cerebral localization. *Hemiplegia with altering paralysis* of some of the first eight cranial nerves is the most mon type (in 50 per cent.). The fifth nerve may be involved in sensory or motor branches. Foci in the substance of the pons involv- the sixth nerve usually also involve the third nerve of the opposite e; foci near the surface involving the sixth nerve do not involve the d nerve. Sometimes the nerves may be involved without hemi- gia (25 per cent.), but rarely without crossed motor irritation or nianæsthesia; hemiplegia alone occurs in 25 per cent. Unilateral nor of the pons may also compress the opposite motor tracts (para- gia or diplegia) and cranial nerves. Motor irritation is not uncom- n, as trismus, facial spasm (Kolish), and sometimes trigeminal ralgia, convulsions in the contralateral limbs, etc. Tetanic rigidity are. *Sensory disturbance* is usual, as a result of involvement of the niscus and formatio reticularis, and occurs as hemianæsthesia ciata (in arm and leg of one side, and in the face of the opposite e when the focus is low in the pons), hemianæsthesia of face, arm, l leg of the same side (tumor in the upper pons), and anæsthesia of or perhaps of both sides. Dysarthria, dysphagia, difficult urination defecation and disturbed respiration on change of posture (Pichler) e been observed.

MEDULLA.—It is remarkable that not only cystic but also solid ors may produce no symptoms or only such that localization in pons or cord is first suggested. In a small number of cases only the symptoms not ambiguous. Bilateral paralysis of the ninth twelfth nerves with degeneration and atrophy of the lips, tongue, ate, pharynx, or larynx, is characteristic, and presents a picture like gressive bulbar palsy. Alternating hemiplegia is rare, but has been served in cases of gliosis. Sensory, motor, atactic, and nerve involve- nts are usually bilateral. Growths *outside the medulla* may cause ralysis of the neck muscles (spinal accessory nerve). Slowed or id pulse, singultus, embarrassed breathing, glycosuria, and polyuria not uncommon. Laslett recently described degeneration in the terior columns of the cord from pulling of the medulla on the pos- ior cervical roots, or from toxæmia, which was previously found Batten and Collier (1889.)

CEREBELLUM.—Cerebellar ataxia, referred by Nothnagel and wers to lesions of the vermis in 80 to 90 per cent. of the cases, occurs t half as frequently in disease of the cerebellar hemispheres.

Compression of the pons, medullar corpora quadrigemina, and fifth to twelfth cranial nerves is somewhat characteristic. Sachs regards involvement of the sixth nerve as almost pathognomonic. Of the general symptoms, choked disk is severe and almost invariable; occipital headache and rigid neck are very frequent. Convulsions are common, and opisthotonos with tetanic rigidity of the body and extremities is most suggestive.

9. OPTIC THALAMUS.—In most cases spastic hemiplegia results from invasion of the internal capsule; hemichorea, hemiathetosis, hemianæsthesia and Nothnagel's mimetic paralysis are not infrequent. Less common are unilateral convulsions, forced movements or postures, contralateral hemianopsia, paralysis of the eye muscles, widening of the opposite pupil and monoplegia.

10. CORPUS CALLOSUM.—Bristowe and others believe that the following symptoms are characteristic; dementia (in 90 per cent.), absence of initiative in speech and action, paraplegia, dysarthria and but slight general symptoms, as choked disk. Oppenheim holds that there are no distinctive symptoms.

11. CORP. QUADRIGEMINA.—Ocular paralyses (of the third nerve) are common, and are sometimes associated as double ptosis, and bilateral paralysis of the internal recti. The pupils are usually free but may be unequal. Oculomotor paralysis may exist with crossed hemiataxia from involvement of the tegmentum, or pressure on the cerebellum. Disturbed hearing from destruction of the upper layer of the tegmentum, amblyopia, and intention tremor, are also observed. No paralysis, except of the third nerve, is present, unless the tumor extends beyond the corp. quadrig. (Oppenheim).

12. PINEAL GLAND.—The symptoms are the same as when the corp. quadrigemina are affected, save that the fourth and sixth nerves are involved more than the third, and compression of the pons or medulla is more common.

13. TUMORS AT THE BASE.—These include bone tumors, aneurysms, hypophysis growths, and those from the nerves or meninges. (*a*) At the *anterior fossa*, hypophysis tumors (see ACROMEGALY) disturb vision, and produce bitemporal hemianopsia with amblyopia or sometimes irregular or transverse hemianopsia. In some instances, there is amblyopia of one eye and hemianopsia of the other. Usually there is no choked disk, but simple optic atrophy is present. The third nerve is paralyzed in most cases, and sometimes other ocular trunks are also affected. Exophthalmos is occasional and the growth may perforate externally into the eye, pharynx, or nose. Loss of smell (anosmia), and mental symptoms are also observed in tumors of the anterior fossa. (*b*) When they occur in the *middle fossa*, mental symptoms and convulsions are less common. The Gasserian ganglion may be compressed and result in neuroparalytic ophthalmia. Other cerebral nerves, as the third, and sixth, sometimes suffer. It must be recalled that a tumor elsewhere may indirectly compress the basal structures. Syphilis and sarcomatosis may be diffusely basal. (*c*) Lesions in the *posterior fossa*

simulate bulbar palsy. The lower eight cranial nerves may be ted and usually suffer more readily and earlier than do the pyraal tracts; the grouping of the cranial nerve implication is different; paralysis of the sixth nerve is rarely associated with the conjugate th nerve, as occurs when the lesion is central. The seventh and th nerves are often conjointly paralyzed in disease of the posterior ı, and but rarely so in central tumors. The symptoms often reble those of lesions of the pons, medulla, or cerebellum.

iagnosis.—Diagnosis depends upon the combination of focal and ral symptoms. Four general groups of cases are encountered:— Absolute clinical latency; (2) general and focal symptoms, the cal case; (3) general but no focal symptoms; (4) definite symptoms ome brain disease but of equivocal significance. According to ns, 80 per cent. of cases can be detected.

nalysis of the General Symptoms.—(*a*) *Headache* is suggestive n it is severe, keeps the patient awake, and when the skull is tender ercussion or pressure (the parietal regions are normally somewhat itive). Its associates, as choked disk or vomiting, are equally ortant. Organic irritative brain disease, as abscess or inflamma-, may cause headache, but mere increase of intracranial tension, ı hydrocephalus, need not do so. Functional nervous disease may e headache, as migraine, neurasthenia, hypochondriasis, hysteria; ay result from eye disease, morbid conditions of the blood or intoxions (uræmia, gastro-intestinal disorder, and poisoning by nicotine, m, alcohol, or lead), and from arteriosclerosis. (*b*) *Choked disk*) or 90 per cent. of cases indicates brain tumor (Oppenheim), and ry marked, it is seldom due to other causes. It is less frequent in ess and meningitis, in which neuritis optica is more frequent. It ıost rare in embolism, softening, hemorrhage, hematoma duræ ris, arteriosclerosis, poliencephalitis, encephalitis, the acute fevers, l poisoning, neuritis multiplex or alcoholism (in which central oma is present). *Uræmia* must be excluded; although the two ases have much in common, as headache, epileptiform or apoplectin attacks, hemiplegia, aphasia, and slow pulse, neuroretinitis is e common in nephritis, though local causes back of the orbit may ıetimes produce it. The choked disk of chlorosis is rare, but onds readily to rest and treatment by iron. Optic atrophy has the e significance as choked disk. (*c*) General epileptiform *convuls* occur in meningitis, pachymeningitis, abscess, vascular disease, ıilis, alcoholism, plumbism, multiple sclerosis, paretic dementia, mia, and gastro-intestinal disease in children.

EPILEPTIC CONVULSIONS.	CONVULSIONS RESULTING FROM ORGANIC BRAIN DISEASE.
coarse; more biting of tongue; re frequent cry; more sudden, and mplete loss of consciousness, freently a visceral (epigastric) aura; ually longer interval between tacks.	Convulsions more orderly, slow, clonic, broken by tonic contractions; often with gradual local commencement, without loss of consciousness, and with paresis in the Jacksonian type.

(*d*) *Vomiting* is probably cerebral and organic, and due to tumor, if hysteria, migraine, anæmia, toxæmia, and gastric disease can be excluded. It occurs on getting up in the morning, and is often associated with vertigo, syncope, and irritability of the nervous centres.

Combination of General Symptoms.—Headache and choked disk occur in chlorosis (*v. s.*), in plumbism (also with delirium, convulsions, and coma, but distinguished by the lead line on the gums), or in uræmia in which special examination for albumin, casts and cardiovascular changes are definitive. The same combination may also prevail in hypermetropia in anæmic girls, in amenorrhœa (Broadbent), and in injuries of the head, but care usually distinguishes them from brain tumor, which is characterized by its slow onset and usual focal signs.

Focal Symptoms and Topical Diagnosis.—*Focal symptoms usually follow the general*, whereas, in apoplexy, for example, the focal symptoms, as hemiplegia, are early. In tumor with hemiplegia, contracture or spasticity accompanies the paralysis rather than follows it as usually occurs in hemorrhage or embolism. Hemiplegia from tumor is less pure, less circumscribed, less systematic (Fournier). A tumor at the base may cause hemiplegia, not from direct involvement of the internal capsule or pons, but from pressure on the middle cerebral artery with encephalomalacia. Jacksonian epilepsy with paresis is, in 50 per cent. of cases, due to cortical tumors. It is also produced by hemorrhage, softening and inflammation, and is then earlier, is not confined to as small an area, and is not as progressive as in neoplasm. The cortical paralysis and epilepsy observed in alcoholism, paretic dementia, plumbism, uræmia, and rarely in tuberculosis, cancer, and diabetes (Senator), are not dissociated and are but transitory.

Nature of the Growth.—(*a*) This may be determined in part from its *location*. *Cortical* growths are most probably gumma or tuberculosis (possibly glioma, cysts, cysticercus, or sarcoma); *subcortical*, glioma, or sarcoma; *pons*, tubercle, gumma, glioma; *cerebellum*, tubercle or glioma; *extracerebral*, sarcoma; *multiple occurrence*, tubercles or gummata. (*b*) Its nature may also be determined *from its course*. A rapidly growing tumor is most probably glioma or sarcoma. An apoplectic attack indicates glioma or ruptured aneurysm. Growth with remissions is characteristic of tubercle or glioma. A very slow course suggests fibroma or osteoma. Coincident meningitis indicates tubercle or syphilis.

Differentiation.—Tumors must be distinguished from *brain absce* abscess and tumor have much in common, as their general sympto both may result from trauma, but brain abscess is almost always s ondary to trauma, or to a pus focus (otitic or metastatic); unilat optic neuritis is a much more common symptom than choked d and abscess (*q. v.*) when once "manifest" is more apt to produce f chills, a rapid course and apoplectic seizures than is tumor, involvement of the cranial nerves is less frequent. It must be bo mind that bulbar tumors may cause fever, and that in abscess time at least, there is a normal or subnormal temperature. Diff tion from paretic dementia, pachymeningitis hemorrhagica interna, neningitis, syphilis, and hydrocephalus, will be considered under each isease.

Chronic meningitis (syphilitic, tuberculous or actinomycotic) or neningo-encephalitis may be confused with diffuse tumors at the base, ut lack pressure symptoms and progression.

Multiple sclerosis must be differentiated. In both affections intenon tremor, disturbance of speech, mental change, vertigo, nystagnus, apoplectiform or epileptiform attacks and tonic spasms of the xtremities may occur, but marked brain symptoms rather speak gainst multiple sclerosis at least in its earlier stages. The pressure mptoms of tumor and increase of cerebrospinal fluid are absent nd in the disk there is an incomplete optic atrophy. If undue stress e placed on the tremor, it may be confused with the incoördination emor of tumor in the optic thalamus, pons or cerebellum.

Course and Prognosis.—The first symptoms are usually general, llowed by the focal symptoms. If left to itself, the disease runs a tal course in *one-half to two years,* although in rare cases it endures ur to fourteen years. A long latent tumor may appear to begin utely. *Exacerbations* result from aneurysmal rupture, hemorrhage to a glioma, cyst formation, ventricular effusions, meningitis, or local uritis. *Remissions* are not uncommon; healing of parasitic and nple cysts, aneurysms, tubercles (in the young) and gummata is ssible. It was thought that aneurysms and gliomata may be cured th iodide treatment (Wernicke). *Unfavorable* symptoms are prounced mental change, incoërcible vomiting, irregular and rapid lse, repeated fits, apoplectiform seizures or festinating neuritis optica. he *mechanism of death* is (*a*) exhaustion from vomiting, pain, or chexia; (*b*) inhalation pneumonia; (*c*) coma with disturbed respiraon, circulation, or other bulbar signs; (*d*) sudden death in tumors of ne base (rarely of the cortex); (*e*) meningitis or arterial disease; (*f*) ntercurrent infections.

Treatment.—Medical treatment does not produce encouraging esults, save in cases of syphilis, but every patient should be given a ourse of mercury and iodide for at least three months. Tubercle may ometimes be arrested by increasing the physiological resistance by rced feeding, fresh air, and administration of cod-liver oil and arsenic. he headache is often relieved by iodide, in any kind of tumor, by the e-bag, morphine, atropine, hyoscine, the coal tar products, and by ephining. Convulsions should be treated by chloral. Brain constion resulting from alcoholism, excitement, exertion, overeating, or nstipation should be avoided.

Surgical Interference.—Macewen, Horsley, Bennett, Ferrier, and odlee are the pioneers in brain surgery. (*a*) In *radical operation* he outlook is not very encouraging. Allan Starr estimates that but 18 per cent. of tumors are accessible, and but 7 per cent. removable; Siedel finds only 3 per cent. are operable. Thirty-four per cent. is basal. At operation the tumor is found in 64 per cent. of cases. The best results are obtained in meningeal and the next best in cortical growths, especially of the motor and occipital zones. Duret (1903) analyzed 344 operations; 18 per cent. died, 64 per cent. improved and vision was completely restored in 60 per cent., and partially in 18 per cent. In cerebellar tumors, sudden death is frequent, because of manipulation compression of the medulla. In 116 operations for cerebellar growth, the mortality, once 70 per cent., is now 38 per cent. (Frazier, 1905).

All varieties, even the syphilitic scar or tubercle, may be removed, excepting metastatic growths. Recovery from the operation is frequent, although the mortality ranges from 20 to 50, or even 80 per cent. But few ultimately recover. Recurrence is common. Death occurs from hemorrhage, sepsis, dislocation of the brain, basal compression, brain œdema and shock. Horsley and Macewen have suggested that the operation be performed in two steps; first exposing the tumor, and later removing it, after the brain has accommodated itself to the change of relations. (*b*) *Palliative operation* may be beneficial. Removal of a button of bone with incision of the dura often greatly relieves the headache and growing blindness, allows fluid to escape and permits the tumor to grow outward when it cannot be removed. Henschen claims that the palliative procedure hastens death more than twice as frequently as it relieves the symptoms. (*c*) *Ventricular drainage* has not proven helpful and lumbar puncture is dangerous; 17 cases of sudden death are reported to have resulted from it.

(D). INFLAMMATION OF THE BRAIN.

I. Encephalitis.

Acute encephalitis is not "inflammation of the brain" (as understood by the laity), which is meningitis. Encephalitis does not include the zone of inflammation which occurs about tumors, foci of hemorrhage, softening, syphilis, and meningitis. It was once held by Durand-Fardel that all softening was encephalitis (cerebritis). Gluge (1837) suggested that some cases were encephalomalacia; Coswell and Hasse (1850) definitely distinguished between them; and Virchow anatomically and Hayem clinically, followed by v. Leyden, Erb, Westphal, Foville, and Otto, described definite cases. The work of Wernicke, Strümpell, Leichtenstern, and Oppenheim will be mentioned later.

Etiology.—There are three main etiological factors; they are (*a*) *intoxications*, chiefly from alcohol, and far less often poisoning by sulphuric acid, ptomaines in decayed meat and fish, nicotine, and sunstroke. (*b*) *Infections*, especially influenza, then scarlatina, measles, hydrophobia, ulcerative endocarditis, and numerous other infections. In such cases, microbes may invade the brain alone. Some relation to chlorosis, sinus thrombosis, chorea insaniens or maniacal forms of

thalmic goitre is possible. (c) *Trauma*, by laceration or concus-
directly or indirectly may produce a *locus resistentiæ minoris*
condary microbic invasion, although an aseptic form is said to

hology.—Wherever located (*v. i.*), the process begins as mul-
lisseminated and often symmetrical foci, principally in the gray
r; these grow and diffuse, sometimes most widely. They vary
from microscopic dots to a diameter of several inches, but usually
e small "flea bite" areas of capillary hemorrhage, which
and stain the adjacent tissues, show above the cut section and
become foci of softening. Microscopically, hyperæmia, perivas-
exudation of red and white cells, serous effusion and changes
nervous elements are observed. Aggregations of mononuclear
re seen, somewhat resembling a tubercle, which Popoff claims
ctually enter the ganglion cells, which become degenerated, fatty,
wollen; the medullary sheaths and later the axis cylinders de-
te. Its issues are:—(a) regression (recovery); (b) necrobiosis
oftening; (c) cyst formation; (d) cicatrix formation with enclos-
fatty granules or hæmatoidin, or (e) it may possibly spread as
e brain sclerosis or (f) may become the foci of multiple sclerosis.
iated nervous changes are optic neuritis, multiple neuritis, and
nyelitis, and *general changes* are enlarged spleen, retinal or general
rrhages and parenchymatous degeneration in the kidney, etc.

**ecial Pathology and Symptomatology.—1. Poliencephalitis Acuta
ior Hemorrhagica (or Ophthalmoplegica).**—This was said by
icke (1881), and also by Bädeker, to occur in *alcoholic* sub-
and therefore in adults; it attacks the gray matter about the
and fourth ventricles, and aqueduct of Sylvius, whence it may
d upward or downward; it results in eye paralysis, and is anal-
to acute poliomyelitis. The *general symptoms* are delirium
ns, or somnolence in non-alcoholics, headache, vomiting, and
neck. The *local symptoms* consist of an early extensive ophthal-
gia, which involves associated eye muscles, as the two internal
or the internal rectus of one eye, and the external of the other,
sually leaves the pupil and the levator palpebræ superior free.
gait is almost invariably atactic and unsteady from involvement
corpora quadrigemina. Speech is frequently difficult. Neu-
s observed in the optic disk, and sometimes in the peripheral
s, as evidenced by the absent patellars, ataxia, tender muscles, etc.
times there is hemiparesis and facial paresis. The temperature
ally normal, or in alcoholics may be subnormal, though respira-
nd pulse are increased. Death occurs within two weeks after
violent symptoms. This type is most easily recognized, although
lis, tumors, or encephalomalacia may affect the same nuclei.
cases thought to be nuclear have been shown by Bädecker,
pson, and Jacobæus to be polyneuritic, and others have shown
thological alteration at autopsy. The diagnosis is easiest in this

2. Encephalitis (or Poliencephalitis) Hemorrhagica.—Strümpell (1884) described primary cases of infantile paralysis (*q. v.*) in which he referred the lesion to inflammation of the motor cortex; though his theory in explaining all such cases is too general, some anatomical confirmation of it is not lacking (Ganghöfner). In 1885–86, Leichtenstern saw cases which resulted from the infections named under etiology. Foci are found in the cortex, subcortex, or central ganglia, containing pus cocci, and influenza bacilli. In some cases the autopsy showed nothing, and intoxication was then assumed. The disease occurs mostly between the fifteenth and thirtieth years, but also in children. After an influenza, there may be (*a*) *general symptoms*, as headache, vomiting, delirium, mental obscuration, convulsions (in the young), high fever without chill, slowed pulse becoming rapid, and irregular breathing, or sometimes stiff neck or optic neuritis; (*b*) *focal symptoms* appear after a few days, as hemi- or monoplegia, aphasia, conjugate deviation, focal convulsions, or hemichorea (Friedmann). The reflexes are affected as in apoplexy. The course lasts one-half to three weeks, and is often fatal, but recovery is not rare. This form may resemble hemorrhage, syphilis or softening, but occurs in the young with normal arteries, and the paralysis is preceded by signs of infection, fever, enlarged spleen, somnolence or convulsions. Sinus thrombosis cannot be differentiated but meningitis is usually differentiated with ease.

3. Subtypes.—These consist of the following: (*a*) *poliencephalitis inferior acuta*, involving the pons or medulla, and giving acute bulbar symptoms; there is paralysis of the sixth, seventh, to twelfth nerve when it occurs in various combinations, or with types (1) and (2). Some of the cases (chorea insaniens, Basedow's disease with mania), may show nothing at autopsy, whence the diagnosis is very uncertain. Confusion with softening, hemorrhage, the late hemorrhage of trauma, or tumors is possible. (*b*) Another form is *poliencephalomyelitis*, acute (Kaiser), subacute (Kalisher), or chronic (Rosenthal), in which the type of Wernicke or of Strümpell-Leichtenstern may descend and co-exist with inflammation and paralysis, frequently symmetrical, of the anterior spinal roots. Hutchinson's facies is observed, *viz.*, fixed eyes, fallen lids, prominent eyebrows, obliterated nasolabial folds, and atrophic paralysis of the arms. Differentiation from multiple neuritis is usually possible, and Oppenheim's criteria are well-established in its separation from myasthenia gravis, in which there is (i) no atrophy, but (ii) remissions in the paralysis, (iii) great muscular adynamia, (iv) absence of reaction of degeneration, and (v) negative anatomical findings.

Prognosis.—The *prognosis* is unfavorable, as a rule. The peracute cases may die in two days; acute cases last one-half to three weeks, and death is most frequent; Oppenheim has shown that they may recover. Much involvement of the mind, decubitus, alcoholic delirium, chlorosis, sinus thrombosis, and weak pulse are unfavorable elements. In children recovery is common, but epilepsy, paralysis, mental alteraı and possibly multiple sclerosis are sequences. Even the bulbar e may recover.

Treatment.—This is wholly symptomatic. (*a*) The causal feature st be eliminated, (*b*) cerebral congestion avoided, and (*c*) the skin, wels, and bladder, the delirium and convulsions, the diet, and the ient's rest must be cared for as in brain hemorrhage, softening, or nor. (*d*) Mercurial inunctions, iodoform inunctions and potassium ide give the best results. (*e*) Electricity is indicated in the chronic ge.

II. Abscess of the Brain.

Brain abscess (encephalitis suppurativa or apostematosa) is not a nmon disease. Eichhorst found but three among 13,563 medical ies.

General Etiology.—All cases are caused by microörganisms, of ich the streptococcus is the most frequent, the staphylococcus com- next and then the pneumococcus, and other bacteria. Mixed infec- ns are common, and old abscesses are often sterile. Sixty-six to 75 cent. occurs in men. It is most frequent between the twelfth and rtieth year. Twenty per cent. occurs in the second, and 33 per cent. the third decade (Gowers). The cerebrum is involved in about 75 cent., and the cerebellum in 25 per cent., but the pons and medulla rarely involved.

General Pathology.—The focus is single in 80 per cent. of cases. e average size is from one to two inches, but may be almost micro- pic or may occupy nearly the entire hemisphere. The surrounding in may be inflamed. Durand-Fardel described the various plaques ntioned under embolism as various stages of inflammation. It is sible that the red softening may sometimes be the first stage of ammation, although far more often due to ischæmia. A zone of ema is common. The right side of the brain is more often affected n the left. Pressure is less common and less intense than in tumor, ugh large abscesses flatten the brain and may produce internal drocephalus. The abscess consists of pus of variable consistency, low or green, acid, sometimes rancid, fœtid or putrid; flocculi of organized brain tissue are common. The pus cells are often indis- ct and fatty needles, cholestearin crystals and sometimes corpora ylacea or foreign bodies are found. Putridity is due to communica- n with the nose or ear, or embolism from putrid lung foci. Encap- ation occurs in 50 per cent., chiefly in old, slow cases, and requires m two weeks to two months. The capsule rarely completely or manently limits the abscess which usually grows and often rup- es, although ultimate quiescence from inspissation and successful markation is barely possible.

Special Etiology and Pathology.—1. *Foci near the brain* are its ıal causes, and for this reason brain abscess is seen by internalists, neurologists, surgeons and otiatrists. (*a*) *Ear disease* explains at least 25 per cent. of brain abscess (even 33 or 42 per cent.). Otitis media is the most frequent cause and is chronic in 91 per cent. (Grünert). Insurance companies generally recognize the risk in these cases. Jansen found one abscess to 500 cases of chronic, and one to 2,650 of acute, otitis. The ear disease may date back thirty or forty-five years, and is especially dangerous when it recurs and when granulations and polypi exist. The acute infections are thus indirectly causative of brain abscess. Infection traverses the bone (92 per cent., Körner), to the dura by way of the thin tegmentum tympani, by the roof of the antrum, sometimes through the petrosquamous suture (in children), by the mastoid cells, lateral sinus, labyrinth or aqueduct along the sheaths of the seventh and eighth nerves (though this is more common in meningitis), and rarely by the external canal. Körner's figures show that in 42 per cent. there is a fistule between the abscess and the causal focus; in 15 per cent. the dura interposes between the two foci; in 15 per cent. there are dural adhesions, and in 17 per cent. the intervening substance is softened brain tissue. Toynbee (1885) remarked that (i) disease of the roof caused temporosphenoidal abscess, but (ii) disease of the mastoid cells chiefly produced cerebellar abscess. Of otitic abscess, temporosphenoidal constitutes 67 per cent. (75 per cent. in children under ten years), and cerebellar abscess 33 per cent. (Körner); the latter is most rarely due to any other cause than otitis, and almost invariably occurs in the anterior part of its hemisphere. Otitic abscess infrequently involves the occipital, frontal (two personal observations) or parietal lobes. The abscess is single (87 per cent.), and lies in the white substance of the brain just beneath the cortex. The smaller the abscess, the nearer it lies to the cortex. Cholesteatomata (pearly tumors) may cause some cases. (*b*) *Traumatism* produces 25 per cent. of the cases. Punctured wounds are more dangerous than open compound fractures. Fractures of the base may result in abscess by infecting the middle ear. Splintering of the inner table and operations on the brain are possible causes; abscess may occur without skull fracture, for contused areas may be infected through the blood. The injury may be direct, or in rare cases, on the opposite side by *contre coup*. Gowers states that but 20 per cent. occurs under ten years of age. Ninety-three per cent. are solitary abscesses (Oppenheim). Infection travels along the perivascular (and perineural) lymph sheaths (Biswanger), but may pass by other routes (arterial thrombosis, or by way of the diploë veins, with or without thrombosis, for "retrograde infection" is possible by the valveless veins). The early traumatic abscess is usually cortical; the late variety is usually subcortical. (*c*) *Other contiguous disease* is much rarer, causing but 3.5 per cent. of brain abscess. Disease of the nose and communicating sinuses (frontal, antral, sphenoidal, ethmoidal), may cause abscess of the frontal lobes near the diseased bone, and in antrum disease, sometimes of the parietal lobes. Disease of the orbit, as phlegmon, or penetrating wounds, and still more rarely cervical cellulitis, cranial osteomyelitis, erative tuberculosis, syphilis or tumor of the cranial bones, may ıse abscess by contiguity.

2. *Distant foci, or metastatic abscesses* cause 25 per cent. of brain scess. The abscesses may number from 2 to over 100 (v. Bergınn); they are multiple in 50 to 75 per cent. In location they are tical or subcortical, less frequently they occur in the central ganglia, ely in the cerebellum, and almost never in this location alone. The ıbolus is almost never found. (*a*) *Lung lesions*: "pulmonal cerebral scesses" are due to empyema, putrid bronchitis, and bronchiectasis 5 per cent. Williamson), gangrene, abscess, and sometimes tubercuıs cavities. This form rarely encapsulates. (*b*) *Ulcerative endocaris*, pyæmia, osteomyelitis, etc., are less common causes; paradoxical ıbolism may occur through an open foramen ovale, whereby the t escapes the lungs. Actinomycosis, suppurating tubercle (D'Esıe, Fränkel), and oïdium albicans (Zenker and Ribbert) have been scribed; in thrush Wagner directly traced the growth from the oat to the brain.

3. The existence of *idiopathic brain abscess* is denied by some, but wers found it in 16 per cent. of his cases; it may possibly result from orgotten trauma or ear abscess. Martius and Strümpell believe the ningococcus causes some cases of this class.

Symptomatology.—Symptoms are absent in 5 to 10 per cent. of es. In 17 cases of Martius' series, brain abscess was not diagnosıted. Symptoms of the *causal affection*, trauma, otitis, or metasis are described under diagnosis.

. *Symptoms of Sepsis.*—(*a*) *Fever* is frequent. Oppenheim considers he most important general symptom; Eichhorst describes cases sely resembling typhoid; it is present in acute cases, and in many ing the last stages when the abscess spreads or meningitis or sinus ombosis develops; the original disease may cause it. Macewen's es showed normal or subnormal temperature. Though it is probably rlooked in some cases, it is again totally absent in others. (*b*) Chills not frequent. (*c*) The pulse is often accelerated, but may later ome slowed to 30 or 40, the compression pulse. (*d*) There may be ier septic signs, as gastric disturbance, anæmia, emaciation, peptonıa, or leukocytosis.

2. *General (Diffuse) Brain Symptoms.*—These are due to compresn, œdema, inflammation, meningitis, or internal hydrocephalus, and clude (*a*) *headache*, which is most common, and usually severe, ough less so than in brain tumor; it is due to increased intracranial nsion; it is usually localized, but even then, the stiff, painful neck d headache are general rather than focal symptoms. It is frequently sociated with (*b*) *vomiting*, and (*c*) *vertigo*, which also occurs in simple r disease. (*d*) *Mental symptoms* are like those of tumor, "slow cereation, heavy comprehension, and a want of sustained attention" lacewen). (*e*) *General convulsions* are frequent only in the last ages, in cases of extensive abscess or rupture into the ventricle. (*f*) he *pulse* is often slowed, even with co-existent fever, to 40 (even 16 or

10), and sometimes with slow or irregular respiration. (*g*) *Optic neuritis* is more common than choked disk; it occurs in 33 to 40 per cent., develops rapidly, and may produce amaurosis and atrophy.

3. *Focalizing symptoms* are often absent, and when present, are less conspicuous than in tumor, because they are obscured by the primary affection or the general symptoms and occur in areas of less physiological dignity. When present, they usually signalize advance of the abscess, increase of surrounding œdema, softening or increased brain tension.

Frontal or mental symptoms may occur (see TUMOR), but foci as large as the fist may remain latent. They may impinge on the *motor cortex.* The symptoms are those of a cortical lesion (dissociated paralysis), which extends more rapidly than tumor, and is attended by Jacksonian fits, rigidity and increased tendon and decreased skin reflexes on the contralateral side; in subcortical lesions from traumatic and metastatic (pulmonal) abscess, hemiparesis occurs in 50 per cent., is sometimes progressive, is usually incomplete and at times is apoplectiform in onset (three personal observations), even when not embolic. If hemiparesis is early, it rather indicates involvement of the internal capsule, which also causes hemihypæsthesia. Conjugate deviation is not common. *Temporosphenoidal* lesions are largly otitic, are frequently latent and are less frequently cortical than subcortical, whence the centres are less involved than the deeper association fibers. The left side is more important; lesions here lead (in 42 per cent.) to *sensory aphasia,* partial word deafness, alexia, agraphia, amnestic and optic aphasia; Körner holds that aphasia is rare. (The right side concerns the memory for letters and figures [Oppenheim].) *Occipital* lesions or those in the contiguous parietal, or temporal lobes, or deeper in Gratiolet's optic radiation, produce hemianopsia (they occur more in traumatic or metastatic than in otitic abscess). *Cerebellar abscess* results almost exclusively from ear disease, and is often latent. Ataxia may also be caused by the ear affection or abscess elsewhere, even in the frontal lobes. Occipital headache and stiff neck occur; there may be a tendency to fall forward (Gravis) or backward (Dupuy), and hemiparesis, or yawning may result from compression; cranial nerve paralyses are not frequent and result from coincident meningitis. Paralyses of the seventh and eighth nerves are often due to bone disease. Abscess of the pons, medulla (of which Cassier, in 1903, found only 16 cases), and crus is exceptional. (See CEREBRAL LOCALIZATION.)

Symptoms by Stages.—(*a*) The *initial stage* is observed mostly in acute traumatic or metastatic abscess. Pus may form in thirty-six hours (Martius), but this usually requires ten to thirty days. The causal symptoms, those of sepsis and general brain symptoms, appear rapidly but focal symptoms usually appear only after one-half to three months; death may occur from coma, fever, and meningitis, or the next stage may appear. (*b*) The *latent stage* has no fever, is seen in chronic abscess, probably corresponds with demarkation and encapsulation.

s for months, years, or even decades, and is either complete or interted by symptoms of the third stage. (*c*) In the "*manifest*" *stage*, iptoms of sepsis and general or focal brain symptoms appear coniously or remittently, with evidence of extension. Most cases are i in this stage. Gowers estimates that after nervous symptoms e develop, (*d*) *the next, the fatal or terminal stage*, appears in five days per cent.), in ten days (33 per cent.), fourteen days (50 per cent.), in thirty days (75 per cent.). This stage may immediately follow latent stage when rupture into the lateral ventricle occurs.

iagnosis.—Recognition is impossible in latent cases affecting ent" areas, whence "no brain disease of equal severity so often ipes recognition." When focal symptoms are absent, various choses may be thought of. The diagnosis is often made only in the two stages. Few cases can be detected without consideration of etiology, which is definitive in 75 per cent. (Gowers), the sepsis, eral and focal brain symptoms.

)titic abscess may be confused with the otitis itself especially in ldren; otitis may produce general symptoms, as headache, vomiting, vulsions and even optic neuritis (probably due to serous meningitis), nial nerve symptoms (nystagmus, diplopia) but all symptoms disear when the middle ear, attic, mastoid or subperiosteal foci are ed for; otitic brain abscess runs an acute rather than a chronic rse, but the various stages may be observed; the period of latency ly exceeds one to one and a quarter years (Körner, Pitt), and ally averages two months or less (Pitt). Temperature is often er than in other forms, the headache may be local or unilateral, local œdema may be seen over the temporal region, with tender- on percussion and a higher percussion note (Macewen).

titis may also cause *suppurative meningitis*, extradural abscess or is thrombosis, either with or without brain abscess, from which must be differentiated. The table on page 1018 is borrowed n Oppenheim's classical description.

erous meningitis (Billroth and Quincke) is often mistaken for the purative form and is very important in ear disease. It is attended little rise of temperature, internal hydrocephalus, paralysis of the al nerves, often gives a serous fluid on lumbar puncture and is n cured. *Extradural abscess* (external suppurative pachymenins) was found in 78 per cent. of 184 cases of brain suppuration cluding meningitis) by Jansen, sinus thrombosis was found in 18 cent., and brain abscess in barely 3 per cent. It is more common per cent.) in acute otitis than is abscess (9 per cent.). Local iptoms, as œdema and tenderness, are common, but diffuse and il symptoms are not frequent. *Sinus thrombosis* is more often nded by pyæmic symptoms, high fever, chills, sweats, icterus and in the lungs, rapid pulse, choked disk, general convulsions and rnal symptoms of altered brain circulation (see SINUS THROMBOSIS).

	Brain Abscess (Otitic).	Diffuse Suppurative Meningitis.
Etiology:	Ear disease.	Same: favored by imperfor[illegible] tympanum and childhood.
Onset:	Acute or subacute, but often some latency.	Acute, stormy, even apoplectic.
Course:	Acute, subacute, chronic (latency stage), from weeks to seldom over one year.	Acute, stormy, more rapid th[illegible] abscess (66 per cent. last und[illegible] one week) though sometim[illegible] remissions occur.
Fever:	Normal, subnormal, moderate.	High, continuous and rarely l[illegible] or absent.
Pulse:	Slow.	Usually fast and irregular.
Sensorium:	Simple dulness, merging into coma.	Unrest, irritability, delirium[illegible] dulness later; sensorium seld[illegible] normal.
Headache, vomiting, vertigo:	Headache almost constant; others less constant.	Headache and vomiting alm[illegible] invariable.
General convulsions:	Not frequent.	Usual, clonic and tonic.
Retina:	Optic neuritis common. Choked disk not common.	Usually negative because of r[illegible]pid course.
Meningitic evidences:	Less by far. Stiff neck rare save in cerebellar.	More common psychical and m[illegible]tor unrest, jactitation, hyperæst[illegible] of special and general sens[illegible]on, trismus; retraction of abdo[illegible] general rigidity.
Focal symptoms:	Frequent, especially temporosphenoidal, internal capsule and sometimes cerebellar; paralysis of third nerve.	Diplopia, strabismus, unequ[illegible] pupils, conjugate deviation, [illegible]aralysis seventh and eighth ner[illegible]s; these are diffuse, transitory [illegible]nd variable; rarely hemi- or mo[illegible]oplegia; very rarely apha[illegible]a, hemianopsia (more frequent in meningitis tuberculosa).
Spinal symptoms:	Seldom. Few cases of absent patellars.	Almost always stiffness and t[illegible]nderness of neck, back and gene[illegible]ral muscles, Kernig's sign; par[illegible]thesia and hyperæsthesia, [illegible]lysuria, involuntary evacuati[illegible]ns, absent patellars.
Lumbar puncture:	Rather dangerous.	Positive findings (see meningit[illegible]).

ntiation is sometimes impossible.

velops *early*, is associated with hæmatoma, con-

:ortical (whence its focal symptoms), and often provokes her general symptoms. It must also be differentiated tic hemorrhage, which occurs days or weeks after the ften in the corp. quadrigemina, pons or medulla; from alitis, whose course is more rapid; from concussion, in 'mptoms are more diffuse and may involve the brain)use fever, though cortical symptoms are absent; from uroses, which lack focal and septic symptoms (Oppen- *ıte traumatic abscess* latency may last in extreme cases, ears. The abscess is not cortical (affecting the centres), al (affecting the conduction or association fibers). *Cere- v.*) must be considered.

metastasis. It is well known that lung suppuration rain with especial frequency, whence the chest must be)calization is largely in the hemispheres, thus obeying topography of simple embolism. The Sylvian district l lobes) are most often affected, and the multiplicity of hould be remembered. The course is usually precipitate. s of *idiopathic abscess* without proper etiology is always sually impossible.

—*Spontaneous recovery* by demarkation, inspissation and (Fenman) is recorded in a few cases, though it is then : such abscesses are healed tubercles. External rupture)nally occur. Without operation, *death* is practically) from rupture into the lateral ventricles (16 per cent., tastatic and otitic abscess), and into the membranes (28 cerebellar abscess), (*b*) from hydrocephalus internus, (*c*) ırombosis, (*d*) from pressure on the medulla, (*e*) from or rarely hemorrhage. Sudden death may occur, how- mor, without adequate postmortem explanation.

.—(*a*) *Prophylactic* therapy concerns the etiological fac- te drainage is always necessary, although granulations : ear are protective and should not be curretted in any ier (precisely as uterine curettage in puerperal sepsis is :nanced). (*b*) *Palliative* treatment is that of brain tumor :is. Special care regarding vomiting is indicated, lest : abscess. (*c*) *Surgical* intervention is indicated. Suc- were those of Morand 1868, Roux 1848, and Schede, as those of Hitzig and Wernicke. Macewen, Barker, 'errier, Horsley, and Schwartze are the pioneers in its tment. Von Bergmann's rule was that operation should 'ormed unless the diagnosis was certain, but adherence 'ould have cost many lives. The contra-indications are : abscess, (*b*) a fatal etiological disease, (*c*) inability to :sthesia or shock, (*d*) ventricular rupture and (*e*) diffuse lthough in the last two cases, isolated recoveries are nus thrombosis, circumscribed meningitis and incipient ot contra-indicate operation. In Körner's cases (otitic abscess), 51.6 per cent. recovered, Oppenheim (traumatic cases) reported recovery in 45 per cent., Wheeler's series gave 87.5 per cent., and Macewen's series showed 96 per cent.

III. Dementia Paralytica.

It is also known as paralytic or paretic dementia, general or progressive paralysis of the insane, and among the laity as softening of the brain. Bayle, 1822, and Calmeil, 1826, first accurately described the affection. It is often classed among the mental rather than the brain diseases, but it is so important in everyday practice and constitutes such a large proportion (10 to 25 per cent.) of all insanities, that its description is in place.

Definition.—Dementia paralytica is a chronic inflammation or degeneration of the brain, characterized by progressive vasomotor, motor and psychical disturbances, and ending in death after two or three years.

Etiology.—(*a*) Syphilis is the cause in at least 75 per cent. of cases (Mendel and Strümpell). That it is not the sole cause is shown by its rarity in Egypt and Japan where syphilis is endemic. (*b*) Other causes, as *modern life*, with its strenuousness, competition, hurry and worry must aid the syphilitic toxins which cause degeneration of the brain after the syphilis is apparently spent (a parasyphilitic affection, according to Fournier). Short sleep, dissipation, overwork, city life and the so-called civilized life account for its increasing frequency. Krafft-Ebbing summarized the etiology as "syphilization and civilization." These causes may coöperate with (*c*) *hereditary nervous taint* (15 to 20 per cent.), and (*d*) *alcoholism* (20 per cent., though this is denied by many). (*e*) *Certain occupations*, as those of officers, artists, and actors, are predisposing factors, and generally speaking, the higher classes are more subject to it. Krafft-Ebbing did not find any clergymen among his 2,000 patients. (*f*) It occurs between the *thirtieth and fiftieth years*, and (*g*) is 7 to 10 times as frequent *in men* as in women. Other factors are less prominent as exposure to heat, trauma of the head, plumbism or chronic toxæmia.

Pathogenesis.—The causes enumerated probably produce (*a*) vasomotor paresis in the fore-brain, followed by (*b*) lymph stasis in the cortex and pia mater, (*c*) degeneration and atrophy of the brain and cord, and (*d*) interstitial inflammation. Though all cases end in brain atrophy, opinions still differ regarding the role of the inflammation; some regard encephalitis interstitialis (chronic meningo-encephalitis) as primary (Mendel), and others hold that it is secondary to encephalitis parenchymatosa (Tüczek and Wernicke), just as in liver cirrhosis it is now thought that the connective tissue formation is secondary in time and importance to cellular alteration.

Pathology.—(*a*) The calvarium is thickened in 33 per cent. The Pacchyonian granulations are often enlarged. (*b*) The dura in 50 per is thick, opaque, vascular and adherent to the skull (*pachy- gitis,* which is often hemorrhagic). In attempting to strip off ia, it is found granular and adherent to the brain cortex (in 80 ent.), especially over the frontal and motor areas, which are ied by the internal carotid artery. Later the adhesions may dis- r. (*c*) The *gross brain changes* are constant. There is less tunity to inspect the brain in the early stages, when it is swollen yperæmic than in the later stages when it is *wasted;* it may but a third or quarter of the normal (40 ounces or 1,260 gm.); hy affects largely, if not wholly, the areas supplied by the internal id artery, especially the frontomotor cortex and the island of The gray matter is yellow-gray, soft and wasted, but the atro- white substance is firm. The *convolutions* are greatly *narrowed* he sulci are widened, for the degree of atrophy corresponds to or 8 ounces of serum which fills the deficit (hydrops ex vacuo). per cent. of cases granulations are seen on the ependyma of the l ventricles, resembling the back of a "cat's tongue," and the icles contain excess of fluid (hydrops ex vacuo). Areas of rrhage, softening and cyst formation are due to complicating lar disease. (*d*) The *minute alterations* in the brain are as follows: anglion cells are early swollen and cloudy; the later findings are: lated, degenerated and atrophied ganglion cells; *wasting of the llary sheaths* and of the axis cylinders, especially in the association ngential nerve fibers running parallel to the cortex (Tüczek), h are said to waste before any inflammation can be seen; corpora acea represent what is left of them; the same changes occur in ranial nerves; the bloodvessels show mural proliferation, *exu- n,* colloid or hyaline metamorphosis, thickening and later atrophy literation; the lymph vessels show proliferation, transudation compression of the cortex, where lymph cysts, from snaring off mph vessels, sometimes cause cystic degeneration of the cortex; euroglia increases even sevenfold (sclerosis). (*e*) Alteration of *pinal cord,* described first by Westphal, is present in 90 per cent.; ateral columns often show descending *degeneration,* but most nonly changes *like those of tabes* are found in the posterior columns mn of Goll) and in the posterior roots. Pachy- and leptomenin- re seen in 40 per cent. As in tabes, the cerebrospinal fluid contains y mononuclear cells. (*f*) Changes in the cranial nerves, especially rst to the seventh, may result from syphilis or alcoholism.

neral Symptomatology.—As a rule, the symptoms are first vaso- r, second psychical, and third motor, in the order of their usually development. It is possible for the psychical symptoms to appear with the motor, or more rarely to follow them. The leading toms are referable to alteration in the frontomotor cortex. *Three* are usually distinguished: 1. In the *prodromal stage,* recogni- is often difficult, because the first symptoms are equivocal or to (i) *neurasthenia* because of the "irritable weakness," ready e in mental processes, headache or sense of cephalic pressure, introspection and rheumatic pains. Again, the initial symptoms are (ii) *congestive,* as headache, migraine, especially significant when it has not occurred before, or attacks similar to minor epilepsy (*petit mal*). (iii) Most important are mental changes. The patient is described as "not himself" but "he does not remark his own alteration" (Schule). Krafft-Ebbing gives a lucid picture of these alterations. The intellect is cloudy; the patient is confused as to time, and comes too early to work or remains after time; he confuses places and may smoke in church; his observation is faulty as well as his judgment; he confuses dreams with actualities, and his memory is confused, for he forgets entire epochs, especially recent impressions. Æsthetic and social lapses are common; the subject becomes vulgar, careless, neglects duties, disregards amenities, and from lack of will power may steal or dissipate most openly. Emotional outbreaks occur, as violent or brutal conduct, or maudlin sentiment over soon forgotten incidents. The patient is often perniciously active. (iv) Focal symptoms, especially amnestic aphasia may appear. (v) General findings are noted after a time, as small and unequal pupils or other tabes-like findings, double vision, unequal innervation of the face, fibrillary contractions of the tongue, tremor, pains, headache, vertigo, congestive attacks, slow pulse, rough, unmodulated voice, skipping of words in reading (paralexia), and irregular, incorrect writing (paragraphia). This stage covers months or years. In rare instances the symptoms may follow tabes; this is "the ascending form of general paralysis." 2. At the *height* of the disease, (i) the psychical symptoms take various types, as maniacal exaltation, melancholia, hypochondriasis, or simple progressive dementia. Psychical blindness or deafness occurs. (ii) Congestive, apoplectiform attacks are frequent, and the general findings become more distinct, as immobile and unequal pupils, hesitant speech with elision of syllables, altered expression, trembling or paresis of the face, incoördination of the hands and gait, disturbances in writing and reading, and retention of urine. During the not uncommon remissions, some of these symptoms can almost always be found. 3. *The terminal stage* is characterized by complete dementia (the final type of all initial varieties), complete disorder of speech, incoördination so extreme as to necessitate feeding the patient and keeping him in bed, sensory disturbances, vasomotor paralysis, grinding of the teeth, trophic complications, and death from pyelonephritis, bed-sores, complications or bulbar symptoms.

Symptoms in Detail.—Psychical Symptoms.—The weakness, *alteration in character,* loss of memory and reason, and the ethical, æsthetic, and moral loss have been described. During the height of the disease there is one of three main mental manifestations: (*a*) *megalomania,* with simple exaltation or mania. This classical expansive type occurs in 66 per cent., is associated anatomically with the characteristic meningo-encephalitis, and is characterized by ideas of grandeur, the patient believing he is a king, God, a millionaire, an athlete, etc. He makes ridiculously large plans, buys extravagantly, lacks will power.

lges himself excessively, changes with great "facility" from one
g to another, is optimistic, friendly, even philanthropic, and rap-
runs through his capital. He loses himself, forgets names, and
uses the real with the unreal. Periods of acute mania, with fever,
grinding of the teeth are expressions of brain congestion; com-
e dementia is the final outcome, even though remissions occur,
ng which some mental or somatic change can be detected. (*b*)
melancholic or hypochondriacal form is thought to occur with
ic degeneration of the cortex. It may be of the dull or agitated
. The patient dwells on the condition of his bowels; one subject
ght that they would "burst and flood the world." There is often
omania, characterized by "self-belittlement," in which the indi-
al fears everything and thinks himself a "dot, nothing, or denies
existence." These cases may commit suicide. Dementia is the
ome. (*c*) *Simple primary dementia* may be present from the
inning; this type is increasing in frequency, while the first classical
n is decreasing. It is thought to be based anatomically on simple
ical atrophy in which inflammation is absent or late and secondary.
se subjects are often deceptive in the early stage, being quiet,
ane and optimistic. In the ultimate dementia of all types, mentality
holly gone, "there is no sense of time, place or identity," and the
ent babbles or is speechless.

otor Disorder.—This usually occurs with mental changes, is con-
t, though variable in its expression, incomplete in degree, extensive
parts involved, and progressive. (*a*) *Speech* is pathognomonically
rbed as a result of mental change, wasting of association fibers
later of bulbar atrophy. It is incoördinate, involving letters
teral" ataxia) or syllables. In pronouncing such words as "elec-
ty" the patient characteristically stutters over syllables. It is
se in the morning and after resting. Loss of word memory is the
nary trouble; the patient forgets expressions, speaks ungram-
ically and "does not notice it." Speech becomes atactic, there
araphasia, the voice is unmodulated, gives out suddenly, is some-
es hoarse, nasal or slow, but never scanning. Later, speech is
urbed from bulbar alteration, causing dysarthria from disease of
facial and hypoglossal nuclei; it is ultimately unintelligible (abso-
aphasia). Closely associated with speech disturbance is (*b*)
rdered reading (paralexia), which is rather characteristic and occurs
y. (*c*) *Writing* is disturbed first because of the mental alteration;
patient has difficulty in collecting his thoughts, although he does
realize the cause (the ataxia by which he drops syllables, repeats
ds or sentences—*paragraphia*); and it is disturbed secondly be-
se of tremor. The writing is uneven, up and down or zig-zag.
se changes are diagnostic. (*d*) *Paralysis of the eye muscles* (in
er cent., Marie) is usually partial and transitory. When total,
hilis or tabes is the probable cause. (*e*) The *pupils*: Myosis may
ur, often just before maniacal outbursts. Mydriasis is frequently
lateral and results from irritation of the sympathetic nerve. In

60 per cent. of cases, the pupils are now large, now small, and this is considered as especially ominous. They are often irregular or triangular. The Argyll-Robertson pupil, responding to accommodation but not to light, usually appears early, is marked in 47 per cent., partial in 20 per cent. and absent in 33 per cent. Reichert holds that it is always due to degeneration in Bechterew's column (*i.e.*, between that of Goll and Burdach). (*f*) The *facial nerve*: The paresis of the lips is cortical, as are fibrillary twitchings in speaking or mimetic movements, automatic chewing movements, and almost constant grinding of the teeth. The "fatuous" or "masked" expression is psychical. Salivation results from cortical irritation. There may be difficulty in swallowing. (*g*) *The limbs*: (i) The cortical changes cause the small-waved, rapid, constant tremor, the ataxia, loss of muscle sense, and the uncertain, ungraceful, tripping, paralytic gait. (ii) The gait may be tabetic, with absent knee jerks, immobile pupils and vesical symptoms; or it is more often spastic with increased patellar reflexes, ankle clonus, contractures and changes in the joints. The patellars are usually increased in the early stages. (iii) Neuritic atrophy of the cranial nerves may occur as in tabes. (*h*) *Apoplectiform and epileptiform attacks* may occur. (i) Apoplectiform seizures result from vasomotor paralysis and brain œdema, and differ from apoplexy in their incompleteness, evanescence, increased temperature and increased reflexes. They last part of an hour, and may produce temporary hemiplegia, but especially aphasia with paresis of the right arm. (ii) Epileptiform attacks occur later, result directly from cortical disease, are usually partial (involving the face or arm), and are either Jacksonian or are attended by partial loss of consciousness; conjugate deviation of the eyes and increased temperature are also observed. They are not frequent but, once initiated, recur and aggravate the mental alienation.

Sensation and Special Senses.—Coincident neurasthenia, tabes and pachymeningitis may emphasize the sensory manifestations, as the lancinating pains of the tabetic form. Sensation is often dulled and, like the analgesia, is cortical in origin. It is thought that the hypochondriacal complaint that certain organs, as the bowels, are absent, is due to visceral anæsthesia. Attacks of migraine, when they first appear late in life, may indicate an organic origin, and Charcot described a form with hemianopsia and symptoms like glaucoma. Migraine is cortical when associated with paresis of the face, tongue or arm. Alteration of the special senses includes optic neuritis and atrophy (4 per cent.), optic hallucinations and psychical blindness.

Other Symptoms.—(*a*) *Vasomotor* changes occur in the skin (lividity, œdema and sweating) and in the brain, lungs, bladder, intestines and stomach (resembling the so-called bilious attacks). (*b*) *Trophic* symptoms are sweating of blood, rough skin, phosphaturia, perforating ulcer of the foot (3 per cent.), bed-sores and friability of the bones, which may be broken without the patient knowing it. The "insane ear" is frequent. Many of these symptoms are spinal and neuritic.

l **Symptoms.**—The pulse is often slow and monocrotic, the
ure low, although the heat centres may be involved in conges-
res with great febrile elevation, and the body weight is often
n the early and late stages, though at the height of the dis-
ten increases, especially before the congestive seizures. The
stinct, at first increased and sometimes perverted, later de-
nsomnia may be severe, may last for months, and sometimes
te.

sis.—The diagnosis depends upon (*a*) the *etiology*, as syphilis
tal strain, head trauma, or excesses; (*b*) the grouping of the
symptoms, as character, æsthetic or ethical changes with
symptoms, as the speech, writing or tremor; (*d*) *vasomotor*
s, as migraine or apoplectiform insults, and (*e*) the *progressive*

ntiation.—1. Diffuse Brain Diseases.—(*a*) Cerebral *syphilis*
onfused with diffuse meningo-encephalitis of the convexity.

PROGRESSIVE PARALYSIS——*vs.*	——SYPHILIS.
absent or vague and transi-	Headache present and severe.
alteration characteristic; ymptoms are coördinated.	Somnolence, variability in mental symptoms. Merely coincident.
m mono- or hemiplegic seiz- .	More frequent.
toms very rare.	Focal symptoms common, together with diffuse symptoms.
ech disturbance, literal para- nd fibrillary tremor of face ue.	Less or no tremor; speech far less frequently disturbed; no literal paraphasia.
ertson pupil.	Absent: oculomotor affections and optic neuritis much commoner.
. slower. No therapeutic re-	In installments, more rapid jumps, remissions. Therapy helps or possibly cures.
ilitic disease.	Active syphilis.

se cerebral sclerosis presents no clear picture and cannot be
ated; though it leads to dementia and attacks of delirium,
t the speech and mental alteration which is present in general
(*c*) *Senile dementia* seldom occurs until after sixty years of
ourse is longer, the symptoms more gradual and less intense,
maintained longer and there is less megalomania than fear
ation and poisoning. (*d*) *Multiple sclerosis* occurs earlier,
philitic etiology, is complicated by late and mild mental
and an intention tremor, which is wider in amplitude and
ring repose. The speech is scanning, staccato, and there is
ataxia. Nystagmus is common, while it is rare in general

paralysis. Motor symptoms are largely referable to disease of the lateral columns.

2. Focal Brain Diseases.—(*a*) Although migraine, headaches, apoplectiform attacks and partial sensory or motor irritation of the Jacksonian type may occur in *brain tumor*, it does not cause difficulty in pronouncing syllables, peculiar mental alteration, or immobile pupils; it is characterized by general symptoms, as choked disk, or vomiting and focal symptoms, which are foreign to general paralysis. In tumor there is simple dulling of the intellect and dementia only with marked internal hydrocephalus. (*b*) Hemorrhagic pachymeningitis complicates 20 per cent. of cases of dementia paralytica; when it exists as an independent affection, it often leads to intense local headache, vomiting, recurrent hemiplegia or monoplegia, cortical irritation, and more rapid course. (*c*) Softening usually produces focal symptoms.

3. Psychoses and Neuroses.—(*a*) *Mania* and *hypochondriasis* may require differentiation. Every case of mania between the twenty-fifth and forty-fifth year not due to alcoholism or to acute somatic disease should arouse suspicion of general paralysis (Mendel). The same may be said of hypochondriasis. (*b*) *Neurasthenia* may be confused with dementia, and the etiology may be identical. In neurasthenia the onset is more acute; headache accompanies the lassitude and "irritable weakness," the memory is fatigued but never lost, the patient recognizes his incapacity; and oxalates and urates abound in the urine. In dementia paralytica, however, there is phosphaturia, hypochondriasis with absurd conceptions, changes in the pupils, disk and field of vision, alterations in character, speech and intellect which are never seen in neurasthenia.

4. Intoxications.—These may produce many of the symptoms of dementia paralytica and may cause many of the forms known as general pseudoparalysis which are due to alcohol, lead, etc. (*a*) Chronic *alcoholism* is not progressive, recovery is frequent, delirium tremens and convulsions are common, the course is acute covering but weeks or months, and the alcoholic tremor, headache, hallucinations, and neuritis are found. (*b*) Chronic *plumbism* (saturnine encephalopathy) is often progressive, even fatal, and in its last stages indistinguishable from dementia paralytica. It is more acute, and 80 per cent. of the cases recover. There is headache, cardiac distress, anæmia, and gingival lead line. (*c*) Chronic *bromism* is distinguished by its stupor, dementia, bromide acne, fœtid breath, coated tongue, pharyngeal anæsthesia, weak heart and acute course, which usually ends in recovery.

Prognosis.—The disease is fatal within three years and in 66 per cent. of cases within two years, though remissions may occur. Krafft-Ebbing saw no recoveries in 2,500 cases; if recovery occurs the diagnosis is probably incorrect and the disease is considered pseudoparalysis generalis. The following courses are distinguished: (*a*) *acute* or galloping form, which lasts months or a year; (*b*) *classical*, expansive type, which lasts three to four years and in which the progis the most favorable; (*c*) the depressive type, which lasts two to : years; (*d*) dementia forms, which last four to five years; (*e*) lar form, in which depression alternates with mania; (*f*) female ty which has a longer course than in males; (*g*) spinal form, with ıtively long course; (*h*) juvenile or adolescent; and (*i*) senile form. .h results in 50 per cent. from the disease itself, by apoplectiform .res, suicide, inanition or bulbar symptoms. In 50 per cent. it .ts from complications, tuberculosis (18 per cent.), bed-sores, with .s (10 per cent.), lung gangrene (6 per cent.), pneumonia (5 per), choking (10 per cent.), and cystitis.

'eatment.—(*a*) *Etiological* treatment. Recovery is barely pos- in the early stages, and many physicians advise that mercurial :tions be given. It must be recalled that the disease is a - or parasyphilitic disease, not active syphilis, and that some der mercury distinctly injurious. Iodides promote resorption and a milder course. (*b*) *Hygienic* treatment, as rest, protection heat, and quiet surroundings, is indicated. (*c*) The patient d be put in an asylum, for suicide or other violence is always ›le. In the simple dementia form, alone, may the patient be d at home. (*d*) *Hydrotherapy* is valuable within limits. Cold lessen excitation, although full cold baths are contra-indicated. *'edicinal* treatment is unsatisfactory. Drugs produce dispropor- e effects and must be exhibited with caution. For delirium and ınia bromides are most efficacious, but chloral and opiates should 'en less frequently or only in combination with hyoscine, digitalis, ›romides, or with the ice-cap. Ergot is said to produce remis- but is valuable chiefly as a vascular tonic for brain congestion. he *skin*, *bladder* and rectum should receive treatment as in exy and spinal diseases.

(E). DEGENERATIONS OF THE BRAIN.

I. Cystic Degeneration.

›tic degeneration occurs mostly in the shrunken brain of aged ns, and largely in the perivascular spaces of the cortical and white substance. No essential symptoms result from the fluid; ıre those only of the causal atrophy. Possibly the vessels are to dilate and rupture from shrinkage of the brain and conse- lack of support.

II. Chronic Progressive Softening.

.ronic progressive softening, when not due to vascular disease, is e affection; it was described by Wernicke. It affects the white er chiefly, occurs mostly after the sixtieth year, its pathogenesis is obscure, and its clinical symptoms are weakness on one side, slight irritative phenomena, as tingling or pain, and involvement of the intellect. The length of the course varies from a month to a few years, and remissions are uncommon.

III. Multiple or Insular Sclerosis.—(See SPINAL CORD.)

IV. Diffuse Sclerosis.

This may involve one or both hemispheres of the brain or cerebellum. (*a*) It is often secondary to meningeal hemorrhages at birth, infantile hemiplegia, arrest of development, and trauma. In these cases the sclerosis is secondary to brain atrophy. (*b*) In other cases there is sclerosis without atrophy. The causes are congenital syphilis, chronic alcoholism, contracted kidney or other exhausting diseases. The symptoms are indefinite. Hemiplegia, impaired mentality, convulsions, contractures, spontaneous movements (Strümpell), vertigo, syncopal attacks and slow speech have been noted. Miliary sclerosis is very rare and consists of minute foci in the cortex, where the gray and white substance join. Many reported cases are due to artefacts in hardening.

V. Atrophy of Brain.

Genuine atrophy is always acquired. It is often associated with microcephaly, and the brain is usually normal. Unilateral atrophy is observed in some cases of chronic insanity. Local atrophy may occur anywhere, is usually associated with sclerosis (*q. v.*) or neuroglia increase, is usually acquired and is very often secondary to previous trauma, tumor, or hydrocephalus. Some reported cases are clearly agenesis and arrested development. The symptoms are not distinctive. Hemiplegia, hemiathetosis and imbecility may accompany the infantile forms; they occur bilaterally when both hemispheres are diseased. *Senile atrophy* occurs in old age, when the brain becomes smaller and firmer and *hydrops ex vacuo* occurs in direct proportion to the shrinkage. Mental changes are not as common as is usually stated.

VI. Hypertrophy of the Brain.

This is most infrequent. Increase of the neuroglia is sometimes present and so-called local hypertrophy is frequently only gliomatous infiltration. Hypertrophy occurs at birth, in the first year, when it is often associated with rickets, and sometimes in older children or in adults. A family tendency is said to exist. The skull is enlarged, the ventricles decreased, and the convolutions are flattened. Coincident trophy of the lymphatic glands, thyroid and thymus has been ved. The symptoms are vague and diagnosis is impossible.

VII. Chronic Bulbar Paralysis.

hough mentioned by Robinson (1815), and Dumesnil (1859), ffection was first thoroughly described by Duchenne (1860), as glossopharyngeal (laryngeal) paralysis, and his observations confirmed by Trousseau, who made the first autopsies. Wachs- (1864) advanced the name, progressive bulbar paralysis, and cot and v. Leyden demonstrated alteration in the bulbar nuclei.

iology.—Most cases occur in persons between *fifty and seventy* of age, although some are observed in children, and sometimes e same family. Heredity is a factor only when the disease is iated with progressive muscular atrophy. Sixty-four per cent. es occurs *in males*. Trauma, cold, infections, mental or physical stion and toxæmias, are uncertain factors. Rheinhardt holds t may follow acute inflammation of the medulla.

hology.—To the naked eye the medulla usually appears normal, h sometimes shrunken. Microscopically, (*a*) the *motor nuclei* ymmetrically and bilaterally wasted, their cells and processes ken. The hypoglossal nucleus suffers most, and few normal remain; its accessory nucleus is usually normal. The spinal ory nucleus is next most frequently affected, but the vagus us suffers less. Degenerative atrophy may invade the glosso- ngeal or exceptionally the facial and fifth nuclei. In some cases lohr), perivascular exudation occurs within the nuclei, and the elements are degenerated (the parenchymatous form), but in s increase of connective tissue or thickening or multiplication of loodvessels may be seen. (*b*) The respective *nerve trunks* in course or in their terminal intermuscular endings, are soft, gray how parenchymatous and interstitial change. This may occur within the medulla, and the posterior longitudinal fibers waste rect ratio to the hypoglossal nuclear atrophy, especially those form the anterior fundamental fibers of the spinal cord. Some- the loop of the seventh nerve wastes, although the cause is not (*c*) The *muscles* supplied by the above nuclei and nerves , as those of the lips, tongue, palate and larynx (the process times extends to the neck and arms). The greatest change is tip of the tongue. The changes are those of progressive muscular hy, *i. e.*, atrophy which is unequal in its distribution, as granular, or waxy alteration, increased nuclei in the muscular sheaths and connective tissue, reddish pigmentation, and sometimes over- h of the fatty tissue. The nuclei of the medulla are rarely the eat of the disease, and when they are alone affected, the disease a rapid course of a few months to a year (Remak), suggesting other structures have not time to be affected. (*d*) In many

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tongue is sometimes large and flabby, but more often
wasted. The *lips* become weak; and kissing, whistling a
of the labials (O, U, P, B, M) becomes impossible. At
seems strange that paralysis of the tongue and lips should
associated, yet physiologically they are more closely ass
any other muscles of the body; anatomically it is prob
facial fibers for the orbicularis oris originate in the hypogl
or arise very close to it. Forced attention may for a wh
imperfect enunciation, but later the paralysis is complet
cannot be closed, the lower lip sags and the zygomatic
come the weak fibers of the upper lip, and accentuate t
furrow. The lips are frequently thin, but may appear
expression is peculiar, the forehead is corrugated, the eye
what lifted, but the mouth is passive, its expression is
laughing and emotional displays produce a strange effect,
seau compared to the mask of the Greek actors. The orig
tions graphically picture the patient constantly holding a
before the lips to collect the free flow of saliva from the
the salivary secretion is probably also increased from
bulbar centre. The chin muscles are also weak. *Swallo*
difficult from the inability of the tongue to propel the
pharynx, so that the subject must push the food back wi
and again the palate (which is controlled by the spinal a
vagus nerves) is weakened, producing a nasal voice and
to regurgitate into the nose. The *pharynx is also paretic* (
by the glossopharyngeal and vagus nerves), and the l
(spinal accessory and vagus nerves). Semi-solid food
swallowed, because fluid more easily regurgitates into t
solids more easily reach the larynx. The *larynx* is oft
The adductors are much more often affected than the ab

hing and weeping are not uncommon. The general nutrition
ers from the dysphagia. In some cases the process extends beyond
nuclei mentioned; the *eyes* may be involved; as in progressive
ear ophthalmoplegia (which is an identical process in the pons),
fly in young subjects and in "family forms"; occasionally the
ical, facial, masseteric, temporal and pterygoid muscles are in-
ed; affection of the latter was considered ominous by Duchenne.
optic nerve is very rarely implicated. Bulbar paralysis frequently
xists with progressive muscular atrophy of spinal origin, affecting
limbs or trunk, and Kussmaul first pointed out that the two pro-
es are identical in nature and differ only in location. The spinal
ase may progress upward to the medulla, causing secondary bulbar
y, or the bulbar palsy may be primary, followed by the secondary
analogous spinal atrophy. In the typical case of bulbar paralysis
e is wasting, paralysis and absence of the reflexes. In some
ical forms there is paralysis but no wasting, and at times the reflexes
increased (clonus in the muscles of mastication); in these cases
probable that the lesion is in the supranuclear fibers, and this is
e bulbar analogue of amyotrophic lateral sclerosis of the cord,"
which it may also be combined later.

the *progressive course* of the malady, the pulse becomes rapid
to 150), and irregular; tachycardia is often preceded by running
ther of the heart tones, which Duchenne compared to the "heart
ing in water." Respiration is difficult, dyspnœa develops and the
ration is weak.

iagnosis.—This is based on (*a*) the distribution of the process in
motor nuclei; (*b*) its bilateral symmetry; (*c*) gradual onset, and (*d*)
nic progression. *Neuritis* of the bulbar nerves is very rare; the
escape, because the nerve fibers to the orbicularis oris, arising in
ear the hypoglossus nuclei run to the pons and leave it by the
al nerve fibers; there is more atrophy, more rapid course, more
ory disturbance and the reaction of degeneration is present. It
rs in acute infections, in leukæmia, etc. *Brain tumor within the*
ulla occurs most often in young subjects and gives the general
local signs of tumor; growths, exostoses, dislocations, caries or
rysms *without the medulla* but rarely produce bilateral or exactly
metrical paralysis; the lips escape, dysphagia is the leading symp-
and other symptoms foreign to bulbar palsy develop, as motor
ation, convulsions, vomiting or hemiplegia (see BRAIN TUMORS,
DULLA page 1005). The bulbar symptoms of multiple sclerosis,
syringomyelia are almost never symmetrical. *Pseudobulbar*
lysis may be confused. Bilateral disease of the cerebral hemi-
eres, sometimes in the cortex, but more often in or near the internal
sule, especially when due to arterial lesions, may cause dysarthria
roy, 1872). One lesion may be in the cortex and the other in the
iculostriate area (Halipré); Jolly found this condition in sym-
rical sclerosis of both hemispheres. Pseudobulbar palsy causes
bral symptoms, as mental change, aphasia, hemiplegia, double

facial paralysis, and hemianopsia; one side is involved after the other and it is attended by no wasting, no loss of reflex action and no reaction of degeneration.

Prognosis.—The disease is almost invariably fatal in one to three years (or seven), especially in advanced life or when co-existent with spinal muscular atrophy. Remissions are possible, and in the rarest instances the disease may be arrested in youth or middle life. Death results from inanition, inhalation pneumonia, intercurrent disease or paralysis of the cardiac or respiratory centres.

Treatment.—Treatment is discouraging. Nutrition must be maintained by feeding with the nasal catheter, to which a funnel is attached. Iron, arsenic and strychnine may be given. Electrotherapy is of little value. Faradization of the neck and tongue may help deglutition; the positive pole should be applied to the spine and the negative pole to the throat. The disease steadily progresses in spite of treatment.

Asthenic Bulbar Palsy; Myasthenia Gravis.

Chronic bulbar palsy *without anatomical lesion* may be discussed in this connection. It was first described by Wilkes (1877), then by Erb (1879), who recognized its light forms, by Goldflam (1891), who with Oppenheim published reports of severe and fatal cases. It is sometimes known as the Erb-Goldflam syndrome. E. Bramwell has collected 90 cases. Its etiology is unknown, but it occurs most often in persons under the thirtieth year, and in 25 per cent. a neuropathic heredity is demonstrable. The cardinal feature is *extreme exhaustion (myasthenia) after the slightest exertion.* Weakness slowly develops in the eyelids, face, palate, masseters, pharynx, vocal cords and the tongue although but slightly expressed by ptosis (the first symptom in 33 per cent. of cases), dysphagia, dysarthria, and difficult mastication, and disappears after rest, to return anew after a few muscular efforts. In six cases myasthenia was confined to the eyes. From these bulbar symptoms the affection is also called *asthenic bulbar paralysis.* In some cases the arms, legs and trunk are similarly involved, so that walking, or arm movements become impossible after short exertion; this, together with the bulbar symptoms, is known by the term *myasthenia gravis pseudoparalytica.* The symptoms are symmetrical. The *myasthenic reaction of Jolly* consists of normal tetanic response to faradic stimulation, but becoming weaker with each repetition, until at last no reaction is elicited. The reflexes are normal or perhaps exaggerated but soon also become exhausted. Some atonicity of the digestive tract is fairly common. The mind, special senses, sensation, muscles and sphincters are normal, although Collins noted a rapid exhaustion of the special senses. Remissions are usual, four years being the maximum clinical duration. *About* 40 *per cent. of cases die* from exhaustion of the cardiac and respiratory centres but the *autopsy* findings are *negative* (in 66 per cent) or doubtful. Kalisher found ıges in the medulla, and C. Mayer found vascular and nuclear ıs. Lymphoid cells have been found between the muscle and Hun and Blumer describe lymphoid and glandular sia in the thymus gland. The *diagnosis* is based on (*a*) of atrophy; (*b*) absence of reaction of degeneration; (*c*) of twitchings and sensory phenomena; (*d*) the presence of adynamia occurring with (*e*) remissions in the course of se; (*f*) Jolly's myasthenic reaction; (*g*) the slight involve- the hypoglossus.

ıent.—Rest and massage are helpful, but iodide and strych- less useful. Galvanization of the head and neck is recom- by Goldflam, who has reported recovery from its use.

Apoplectiform Bulbar Paralysis.

ı or apoplectiform bulbar paralysis does not properly come generative brain disease, but deserves discussion in this place ke of topical differentiation. Its usual *causes* are hemorrhage, ı, thrombosis, and less frequently bulbar encephalitis, trauma ression, bulbar neuritis, the ascending paralysis of Landry, ılysis after acute infections. Its *symptoms* are as follows: *onset* is apoplectiform with symptoms (which are rarely focal) o, vomiting, dyspnœa, yawning, slowed heart, which often thout coma. They may progress or regress. (*b*) *Motor* are frequent, as *double hemiplegia;* monoplegia is possible, lesion occurs at the pyramidal decussation, there may be ia cruciata, *i.e.*, paralysis of one arm and the opposite leg. ral focus may produce bilateral symptoms, which, however, wholly symmetrical. The pyramidal tracts in the medulla lied by the inferior cerebellar artery. Descending pyramidal tion follows severe involvement of these tracts. (*c*) *Sensory* may develop, as hemiataxia, or hemianæsthesia, (possibly listurbance of muscle sense). (*d*) The *bulbar nerves* may be paralysis of the tongue, pharynx, masseters, larynx, and, of the vascular supply, frequently of the eyes, face and other tres may result. The reflexes of these parts are often sus- though in the extremities they are increased. The lesion ıt the level of the nuclei, or supranuclear. The hypoglossal al accessory nerves are supplied by the anterior spinal and arteries; the vagus, glossopharyngeal and auditory nerves by bral; the fifth, seventh and eye-nerves (third, fourth and the basilar artery. There may be hemiplegia with paralysis posite side of the tongue if the lesion is in the anterior part of ılla.

iagnosis is usually easy from the acuity of the lesion in the *Chronic bulbar palsy* is excluded by the onset and the asym- d irregularity of the bulbar symptoms, as double hemiplegia, which is unequal on the two sides, or dysphagia without paralysis of the tongue or lip. Exact differentiation between hemorrhage, embolism and thrombosis is frequently impracticable. *Pseudobulbar paralysis of cerebral origin* presents the characters of disease of the hemispheres and is clearest when one attack is followed by a second. Differentiation is impossible should lesions in both hemispheres occur simultaneously.

The *prognosis* is most grave at the onset, but later there is less danger of progression. Improvement and even recovery are possible. *Treatment* concerns chiefly the underlying vascular disease, as in hemorrhage, or embolism (*q. v.*).

VIII. Progressive Nuclear Ophthalmoplegia.

This is a degenerative atrophy of the oculomotor nuclei, similar pathologically to chronic bulbar paralysis. It is more conveniently considered under affections of the third nerve (*q. v.*).

(F). HYDROCEPHALUS.

Acute hydrocephalus is an acute accumulation of fluid either between the dura and the brain (acute external hydrocephalus) or in the ventricles (acute internal hydrocephalus). In some cases the fluid is a *transudate*, being part of a general œdema resulting from cardiac or renal disease, or being due to local causes, as sinus thrombosis. In most cases it is an *exudate*. Acute meningitis is the only certain cause of acute hydrocephalus, and is most often tuberculous (80 per cent.); it is less often the serous meningitis of Billroth and Quincke. It is difficult to distinguish between transudation and exudation, and diagnosis is uncertain. Quincke thinks some cases are angioneurotic.

Chronic Hydrocephalus.

External form (fluid between the dura and brain).—The causes are (*a*) compensatory *hydrops ex vacuo*, occurring where brain development is arrested (the congenital form) or when the brain wastes (paretic dementia, softening, hemorrhage, sclerosis, senile atrophy, porencephaly). The hydrops is a transudate under the dura in the arachnoid meshes, or between the pia and brain, and has no clinical importance. (*b*) *Stasis*, as in sinus thrombosis; and (*c*) *chronic diseases*, as cancer, nephritis or alcoholism are also causes. The symptoms are indeterminate.

Chronic Congenital Internal Hydrocephalus.

The etiology is uncertain. It has been thought that the cause was fœtal meningitis, inflammation in or obstruction of the choroid plexus,

truction in the branches of the vena Galeni, in the lymphatics, lateral apertures of the fourth ventricle near the glossopharyn- oots, in the aqueduct of Sylvius, or in the foramen of Magendie schka. Hereditary influences and syphilis (10 per cent.) are imes suggested; parental alcoholism and trauma are questionable
.

ptoms and Pathology.—The head is large and may be an obsta- labor. It may measure over 5 feet (167 cm.) in circum- e. If it appears after the seventh year, the head is not enlarged, cretins the head may be undersized. Often the head cannot be pright. The skull is thin, even membranous and translucent candle test. If the disease halts, the bones may here and there n, and Wormian bones may develop. The head projects beyond se, the sphenoid is luxated forward and downward, the ear s points downward, the occiput is forced back, the sella turcica tened and widened, the roof of the orbit may be pushed down to become palpable, the fontanelles are large, the fissures gape, uctuation may be felt. The hair is thin, the veins are large and olic murmur may be heard, the cause of which is doubtful. The *is pale* and may be flattened almost beyond recognition. The ranes are often thickened, and the ependyma are often thick, lar and sclerotic. The average amount of fluid is 1 quart, aximum 13 quarts. The ventricles, especially the lateral, are ed; sometimes the third or fourth ventricle escapes. The ed third ventricle easily compresses the optic chiasm. The en of Monroe is often very large. The fluid is usually clear, imes slightly yellow, and rarely blood-stained. It may contain dial flocculi; it is neutral or alkaline, its specific gravity is 1,001 09, the albumin is 0.2 to 2.0 pro mille; and nuclein, fibrin, urea, tearin, a leucin-like body, and sugar, are found. More than 2.5 ille of albumin or a specific gravity higher than 1,009 indicates mation, according to Huguenin.

face is small, triangular, and the eyes are prominent. Much sclera is shown and the upper lid is small, though the lower lid over much of the iris. *Nervous symptoms* are not unexpected, brain may be reduced to a small fraction of an inch in thick- nd is a mere flask for the fluid. The demarkation between gray hite matter is lost and in marked cases the convolutions and re obliterated, the basal ganglia flattened, the cerebellum forced e spinal canal, and many structures, such as the fornix or corpus m have disappeared. *Mentality,* as a rule, is impaired or ed, speaking is learned late if at all, will power is lacking, and ility is common. Patients are prone to psychoses. Epilepti- ttacks, headache and vomiting are fairly common. Eye symp- re frequent, as diminished vision or blindness from initial optic emia and subsequent atrophy, strabismus, nystagmus, and wide, ly reactionless, pupils. Other special senses suffer. All the or the legs alone, often present spasticity, contractures, paresis and increased reflexes, if not secondary degeneration in the pyramidal tracts. The face may be similarly affected. The child walks late, if at all. Sensation is practically normal. In some instances there is constipation, which may be followed by vomiting, diarrhœa, emaciation and death. Puberty may be delayed or the infantile testes may persist. Lumbar puncture shows increased intraspinal pressure, amounting to a column of 36 mm. of mercury.

Complications are largely anomalies of development, as syringomyelia, hydromyelus, meningocele, microcephaly, porencephaly, anencephaly, and encephalocele.

Course.—The course varies considerably. Some subjects die during or after birth. Many die in the first three years from increase of brain pressure with coma, from intercurrent disease or diarrhœa. In some cases temporary remissions are seen, and in still rarer instances, permanent arrest of the process occurs, the mind improves with partial recovery, the speech shows some defects or the gait remains paretic and spastic. In very exceptional cases, rupture externally is observed after head trauma, or spontaneous breaking into the ear or nose occurs, as in Lebert's case, which "leaked" for five years. Gall reported a case which lived fifty years, and Goelis one of seventy-one years' duration.

Acquired Chronic Hydrocephalus.

It is difficult and often impossible to differentiate between the acquired form and an acute exacerbation of the congenital variety.

Etiology.—The causes are (*a*) *stasis*, either general (cardiac or pulmonary) or local (brain tumor or sinus thrombosis); (*b*) *cachectic* transudation, as in carcinoma or nephritis; and (*c*) *inflammation*, beginning as acute meningitis of the sporadic or epidemic type, or as slowly beginning chronic basal meningitis, especially in young children. The same points of obstruction may exist as in the chronic variety. (*d*) Rickets is possibly a cause or at least a frequent associate. Quincke's idea that some forms are angioneuritic has been mentioned. Some cases show nothing at autopsy.

Symptoms.—Meningitic symptoms usher in some cases in which after weeks or months new evidences of brain pressure appear, as alteration in the pulse and breathing, unequal pupils or coma. Headache, vomiting, vertigo, visual disturbance, spastic paresis, epilepsy, altered mentality, idiocy or involvement of the cranial nerves are seen, as in the chronic congenital form. The shape of the head, however, is not altered.

Diagnosis of the chronic congenital and acquired forms.—Differentiation between the two forms is usually very difficult, unless there is an accurate history. (*a*) From *rhachitis:* The two affections are often associated, for some unknown reason. The rhachitic head is square rather than globular, it protrudes more in front and laterally than behind, the sutures are less widely separated, the fontanelles are wide t do not bulge, and there are no signs of brain tension. The head ›uld be measured, because the rhachitic head often seems larger .n it actually is. (*b*) From *brain tumor:* The two conditions are netimes coincident. In each, symptoms of brain pressure, both al and general, are observed. The congenital form is more easily tinguished by means of the history of the disease from birth, and slower course, but focal symptoms are less common, as hemiplegia, noplegia, and hemianæsthesia, and spinal symptoms are more com- n than in tumor. (*c*) *Hyperostosis* of the skull (Paget's disease), in hypertrophy and acromegaly increase the size of the head, but y are rare and are separable by the same methods of argument as rhachitis.

Treatment.—Few therapeutic results can be obtained. *Bandaging* head is sometimes helpful, but must be carefully done, lest it ravate the brain compression. *Puncture* of the *lateral ventricles* es from the time of Hippocrates. Isolated recoveries are recorded, the evacuation must be slow. Permanent drainage is usually l, but occasionally has resulted in cure. Attempts to obliterate the tricles have recently been made, as in Mikulicz's, Troje's and Leur's rts to effect anastomosis between the ventricles and the subdural e; McArthur was successful in three cases. Lumbar puncture cquired forms may be useful; though less dangerous than radical sures, death may result.

DISEASES OF THE CEREBRAL MENINGES.

nflammation of the dura is termed pachymeningitis, and inflam- tion of the arachnoid and pia is known as leptomeningitis, or simply meningitis.

PACHYMENINGITIS.

xternal pachymeningitis is essentially a surgical disease and pos- ses no independent significance, being secondary to skull disease philis, trauma), various infections and atrophic brain affections. symptoms are indefinite, as pain, headache, or possibly hemi- gia, or they are those of the primary lesion; the diagnosis is difficult. gical treatment in Macewen's 22 cases gave 100 per cent. recovery. *Internal pachymeningitis* is divided into (*a*) the purulent form, which lows bone disease or leptomeningitis; and (*b*) the pseudomem- anous type, sometimes with fluid between its layers, which is known serous pachymeningitis and is seen chiefly in general paralysis or children with perisplenitis. These varieties are not as important as the last variety, known as *pachymeningitis hemorrhagica interna;* this form was described by Heschl (1855), and a year later Virchow gave it the present name; he regarded the inflammation as primary and the hemorrhage as secondary. Kremiansky (1868) developed the etiology and produced the disease in dogs by administration of alcohol.

Etiology.—(*a*) Most cases occur in *middle and advanced years*, although a few are seen in marantic children. Fifty per cent. occurs in persons over fifty years of age, 40 per cent. in those over sixty, and 25 per cent. in those over seventy (Gowers). (*b*) Seventy-seven per cent. of cases occur in *males*. (*c*) *Paretic dementia* causes 19 per cent. Pachymeningitis is common in *other dementias* and chronic psychoses, whence its far greater frequency in asylums and poorhouses than in private or hospital practice. It also occurs in senile atrophy, arteriosclerosis, and encephalomalacia. (*d*) *Chronic alcoholism*, frequently with cardiac and renal disease, is a more frequent cause than the statistics of König show, *viz.*, 6 per cent. (*e*) *Trauma*, as during birth, from forceps delivery, or from small pelvis. (*f*) *Hemorrhagic diathesis* (scurvy, hæmophilia, leukæmia, pernicious anæmia), chronic nephritis. (*g*) *Infective diseases*, as typhoid, scarlatina, puerperal fever (9 per cent.), heart disease (18 per cent.), hereditary syphilis (11 per cent.), tuberculosis (23 per cent.), bronchitis, and pertussis are also causes.

Pathology and Pathogenesis.—**1. Gross Pathology.**—In slight cases the inner surface of the dura is cloudy, yellow or brown from punctate hemorrhages or hæmatoidin deposits. In pronounced cases the dura shows membranous laminæ, which are adherent by proliferation of bloodvessels and by organization. The laminæ may number even 20 and may be a quarter of an inch thick. Those layers nearest the brain are the most newly formed. Repeated hemorrhage into the layers of the newly formed tissue or between them is very frequent, and may be an inch or more thick or may reach the size of an egg. The free hemorrhage may spread over the entire surface of the brain, even reaching the retina; older foci are apt to be encapsulated. Cyst formation in the clot, suppuration and calcification are rare issues. The leptomeninges are tinged and often somewhat inflamed. Corresponding to the most frequent seat of pachymeningitis, the convexity of the brain is compressed, especially over the motor areas. The process is bilateral in 56 per cent. of cases, and the basal cranial nerves are seldom implicated.

2. Minute Examination.—The normal dura is vascularized from its external aspect, and the branches spread into a capillary mesh on its inner surface. The fresh fibrin of the earliest hemorrhage and the exudation contains red cells with large nuclei, and, nearest the brain, a covering of endothelial cells. Later the exuded fibrin develops capillaries, organizes, and again hemorrhage occurs from the newly formed vessels. This organizes in turn, so that successive laminæ develop. The older layers become firm and fibrous.

3. Pathogenesis.—Virchow viewed the affection as primary pachyingitis with secondary hemorrhage, but Spierling and Hueguenin that the hemorrhage was primary, with secondary inflammation. how's views still hold for most cases, although more recently s has proven that two classes of cases exist. (*a*) In traumatic s, the hemorrhage is primary and a simple clot forms which later nizes into firm connective tissue; this variety is called *regressive.* same sequence is thought to occur in cases due to the blood dis- s, acute infections, and arteriosclerosis. (*b*) In chronic pachy- ngitis proper, the inflammation is primary, the process is *pro- ive,* the connective tissue formed in layers is looser, more vascular, its inner, successively formed layers, are the seat of repeated rrhages.

mptoms.—In most cases there are no symptoms, because the brane is small and the hemorrhage is slight, or because the pachy- ngitic symptoms are overshadowed by those of the original dis- In the minority of cases, symptoms exist depending on the sity of the process, its extent and localization.

General Symptoms.—The most frequent one is headache, which *r se* of little differential value. It is diffuse and is most marked coholics. Vertigo is also of no diagnostic value. These symp- increase in value when mental dulness or irritation and coma lop with focal signs (*v. i.*). There may be prodromal delirium. pulse is sometimes slow and irregular. Choked disk is not rare. lerate albuminuria sometimes occurs. The temperature is vari- , but may be slightly elevated.

Focal Signs.—The most important is hemiplegia, which is pre- d for some time by coma (Fürstner). The paralysis is contra- al, rarely complete, varies with increase or decrease of the clot, involves the cortical facial and hypoglossal centres, is sometimes ral and is associated with aphasia (33 per cent.), hemihypæsthesia cially involving the muscle sense and therefore causing hemi- a), hemiamblyopia, contractures in the paretic members and onian epilepsy. Conjugate deviation of the head and eyes, gmus and forced postures may be observed. The patient may at his clothes, or at his hair or beard (Fürstner). The pupil alateral to the lesion is usually larger (Griesinger); the pupils poorly, and later both are wide. The tendon reflexes are increased, e skin reflexes are decreased or abolished during the coma. The nerves are very rarely involved; trismus and rigidity of the neck ncommon. Statistics on lumbar puncture are as yet lacking.

rse and Prognosis.—Death may occur in the first seizure, or the toms may subside for months, years, or possibly permanently. ual signs are common and include headache, ataxia, psychical tion, paresis and aphasia. Recurrence is frequent, with stupor, , and hemiplegia until death occurs (*a*) from the fundamental se, (*b*) from the hemorrhage, or (*c*) from intercurrent affections. outlook depends on the etiology; for instance, it is better in olism than in dementia.

Diagnosis.—A correct diagnosis is rare. A patient with paretic dementia may die from a pseudo-apoplectic shock, or from pachymeningitis, or a nephritic subject may succumb to pachymeningitic hemorrhage, uræmia, brain œdema or heart failure. A presumptive diagnosis can be made from (*a*) the age, (*b*) basal disease (paretic dementia, alcoholism), (*c*) preceding dulness or coma, (*d*) hemiplegia or (*e*) irritative phenomena (small pupils and convulsions), (*f*) from variability and (*g*) recurrence of the symptoms.

Differentiation.—It should be distinguished from *localized brain disease* as (*a*) paretic dementia (*q. v.*), and (*b*) cerebral hemorrhage. Differentiation may be impossible, especially when pachymeningitic hemorrhage is very large. The etiology is different, and in pachymeningitis there is more cortical irritation, the coma and paralysis are more variable, and temperature, narrow pupils and optic neuritis are more common. (*c*) In encephalomalacia, the hemiplegia is more often attended by aphasia and hemianæsthesia, and there is less cerebral irritation. (*d*) Brain tumor is easily distinguished. Trauma, apoplectiform attacks and progression with remissions occur in both affections. Tumor usually appears earlier in life, does not depend on alcoholism, and focal signs, intense headache, and choked disk are much more common.

Diffuse brain disease chiefly concerns meningitis, whose cardinal symptoms (*q. v.*) and course are usually distinctive.

Treatment.—Treatment should be (*a*) prophylactic and etiological; and (*b*) that of the seizure. The use of the ice-cap, quiet, elevation of the head, application of leeches back of the ear, and possibly venesection are indicated. Nourishment should be given by rectum. Morphine and chloral relieve the headache, excitement or convulsions. Lumbar puncture has not proven beneficial (Ricken). Operation was first performed by Ceci in traumatic cases and by Annandale, Jaboulay, Monroe, and Ballard in other varieties. In the future, surgery will probably benefit a larger number of cases.

MENINGEAL HEMORRHAGE.

Meningeal apoplexy may be extradural, subdural, arachnoideal, subarachnoideal, and subpial.

Etiology and Pathology.—(*a*) Trauma may rupture the anterior, middle or posterior meningeal arteries, with or without fracture of the skull. Rupture occurs usually at the site of trauma, but sometimes by *contre-coup*. The corresponding veins may rupture, also the sinuses, or the pial vessels from the latter of which blood may percolate to the base of the brain, affecting the basal nerves, or may reach the spine. The internal carotid may be ruptured, and death then occurs precipitately. External hemorrhage is either diffuse or circumscribed and results most frequently from middle meningeal rupture. The blood clot may weigh 3 to 17 ounces or may reach the size of the

compressing the brain, and dislocating the falx. If the dura is
red, blood may be found in its substance. *During birth* (see IN-
TILE CEREBRAL PARALYSIS) trauma may be a factor, and hemor-
ge has even occurred during gestation from maternal trauma
ale), but asphyxia and precipitate labor are more important;
e symmetrical hemorrhage of the convexity is common. (*b*)
eriosclerosis and rupture of cerebral aneurysms may cause menin-
hemorrhage. (*c*) Acute infections, hemorrhagic diathesis, sinus
mbosis and rarely, heart and lung disease, may produce it. (*d*)
per cent. of cases occur under ten years and 50 per cent. under
y years of age (Gowers). Small clots may be absorbed or micro-
a, porencephalia, cysts or brain sclerosis may result.

ymptoms.—Clinical latency is not rare in the new-born, possibly
use of the poor development of the motor centres. Small hemor-
ge may in the adult produce no symptoms. Very large ones result
arly sudden death. In rupture of the *middle meningeal* artery there
(*a*) symptoms of brain pressure with those of brain concussion or
tusion, as loss of consciousness, irritative symptoms, increased
thing, slowed pulse, pallor, vomiting, and slight fever (100° to 101°).
er death or improvement may result. (*b*) In other cases improve-
t from the concussion and contusion occurs; there may be an
rval of hours to often of days when *hemorrhage occurs or recurs*, with
a, pressure symptoms, mono- or hemiplegia and convulsions as in
hymeningitis. Rigidity is rather rare; it is less frequent than in
ingitis, but Oppenheim has observed rigidity on the hemiplegic side
flaccidity on the sound side, which may cause error in localization.
pupils are variable. Hemorrhage of the sinuses and pia are said
ause monoplegia frequently; the brain nerves are sometimes
hed by infiltration to the base. In the new-born, there is asphyxia,
then rapid pulse, and coma; this is followed later by epilepsy,
cy and spastic paralyses.

ifferentiation.—*From concussion:* an exact diagnosis is possible
after trephining, or by the clinical evolution. From *cerebral
orrhage:* the course is very rapid. Differentiation from slow,
avescent cerebral hemorrhage is impossible.

rognosis and Treatment.—Ninety per cent. of cases die from
ctant treatment (Wiesmann); 33 per cent. die in twenty-four
rs, and, when the hemorrhage is due to aneurysmal rupture, 30 per
. die in ten hours (Lebert). Early surgical treatment is indicated;
was especially described by Krönlein.

OUTE LEPTOMENINGITIS; SUPPURATIVE, EPIDEMIC AND TUBERCULOUS.

n 1768, Whytt, of Edinburg, noted the acute hydrocephalic symp-
s of meningitis but failed to recognize the meningitis, which Quinn
'9) described. In 1805, Vieusseux, of Geneva, described the epi-

demic variety; in 1806, it was seen by Danielson and Mann in New England, and they performed the first autopsies (Osler). Coindet (1817) described the cry, *cri hydrencéphalique*, and Senn in 1825, coined the name meningitis. In 1819, Guibert described the granulations and Guersant spoke of *meningite granuleuse* as associated with phthisis. Parpavoine (1830) recognized the tuberculous form and Gerhardt of this country two years later drew the profession's attention to the role of tuberculosis. In 1855, Rilliet and Barthez distinguished the suppurative, secondary, tuberculous and other forms.

Acute leptomeningitis, or meningitis, is of various forms which may well be considered together, because the main symptoms are common to all, though epidemic meningitis might be classed under the acute infections, and tuberculous meningitis is usually part of a generalized tuberculosis. Their contrast is better effected, however, under the grouping followed below.

General Etiology.—**1. Trauma.**—The microörganisms of the ear or nose may find ready access to the brain, when its resistance is lessened by wounds, concussion, or contusion.

2. Regional Extension.—(*a*) From the *ear:* acute or chronic disease of the middle ear, antrum or petrous bone may invade the brain through the bone, along the lymph or bloodvessels, the nerve sheaths or sinuses (see BRAIN ABSCESS) and may result in suppurative, tuberculous or serous inflammation. (*b*) From the *nose:* extension by this route is generally less common, but may be frequent in the epidemic form (Weigert). The disease may be acute or chronic, spontaneous or operative (by sounding with a probe), syphilitic, tuberculous, meningococcic, neoplastic or suppurative, of the nasal mucosa or of the ethmoidal, sphenoidal or antral cells. Infection may travel through the bone, along the veins which communicate with the dura, or along the olfactory nerve endings. (*c*) From the *eye:* the avenues to the brain are numerous, and yet disease of the eye itself, even panophthalmitis is less important than orbital cellulitis or enucleation, which exposes the sheath of the optic nerve. (*d*) Extension may also result from other less frequent sources, as *parotitis*, carbuncles, especially of the face, whose veins communicate with the cavernous sinus; scalp affections; erysipelas; bone disease, especially abscess and tubercle; and sinus thrombosis.

3. Metastatic or Secondary Extension.—(*a*) *Tuberculosis* is the most frequent cause, and this is easily understood when we study the statistics of Nägeli, who found tuberculosis in 97 per cent. of autopsies. (*b*) *Septicopyæmia*, from lung suppuration, empyema, arthritis, endocarditis and kindred affections; (*c*) *pneumonia* (about 0.5 per cent. in 5,540 cases); and (*d*) rarely exanthemata may produce it. In group there is probably an intermediate link, as otitis media in latina and empyema or lung infarct in typhoid. (*e*) Syphilis, gene subacute or subchronic may also cause it.

4. The Epidemic Form.—This is in itself an etiological entity.

5. Diseases of Metabolism.—Meningitis may rarely occur in the course of diabetes, nephritis, gout, arteriosclerosis or malnutrition, but it is probably only a terminal infection.

General Bacteriology.—(*a*) The *tubercle bacillus* is the most common microörganism. (*b*) The *pneumococcus* is probably the most frequent among the non-tuberculous varieties. It was first found by Fränkel and simultaneously by Bardoni-Uffreduzzi. This form may be primary, or secondary to pneumonia. (*c*) *Pyogenic organisms*, especially the Streptococcus pyogenes and the Staphylococcus pyogenes aureus; (*d*) the *Meningococcus intracellularis* (Diplococcus intracellularis meningitidis) which was found by Marchiafava and Celli, then by Weichselbaum (1887), and Jäger, and was first withdrawn by Heubner by lumbar puncture; and (*e*) the *typhoid bacillus* (Roux, Adenot, Lenhartz), the *colon* bacillus (Braun, Chantemesse, Widal, Netter), the *influenza* bacillus (Pfuhl and Hägerstedt), Friedländer's pneumobacillus, Bacillus pyogenes fœtidus, Bacillus pyocyaneus, proteus, gonococcus, Bacillus anthracis, Bacterium lactis aërogenes, Bacillus aërogenes meningitidis (Cantani), and trypanosoma are also found.

I. Suppurative Meningitis.

This is usually a secondary affection.

Pathology.—1. *Macroscopically* a purulent exudate is found between the dura and leptomeninges and in the meshes of the arachnoid. The dura is tense and its inner surface dry. The leptomeninges are reddish, cloudy and are often dotted with punctate hemorrhages. The exudate is clearly purulent, yellow or whitish-green, and succulent. The infection spreads rapidly, follows the perivascular lymphatics, as streaks of pus, imbeds the vessels and nerves in its meshes, obliterates the sulci, and involves not only the brain membranes but also those of the cord. It is a misapprehension that the epidemic cerebrospinal form (*v. i.*) involves the spinal meninges, and that other suppurative forms do not. The brain is always altered; it is succulent, swollen, springs out as the membranes are cut, is vascular, and sometimes locally anæmic from compression by the exudate; its surface is most affected, by such changes as small abscesses, punctate hemorrhages or minute islets of softening or encephalitis. The convexity is most often involved in the metastatic form (convexity meningitis) and the base most frequently in ear disease. There may be primary or secondary sinus thrombosis. The ventricular fluid is often increased, and is cloudy, purulent or sometimes serous. The apertures of the ventricles may be occluded (see HYDROCEPHALUS); inflammation may be noted in the velum interpositum, choroid plexus or ependyma. In the spinal membranes the changes are similar and are most marked over the posterior part of the dorsal region. The process is usually diffuse. On incising the membranes, the swollen cord bulges out and the same changes are seen as in the brain. The process may extend along the nerve trunks and bloodvessels, even into the orbit.

2. *Microscopically* there are the usual findings of inflammation, perivascular exudation of red and white cells, and fibrin formation. Vascular dilatation and exudation are seen in those cases in which the membranes appear normal to the naked eye. Exudation is most marked over the cortex and near the ventricles, but may also occur deeper in the medullary substance. The ganglionic cells show degeneration, although they have not been studied systematically. Analogous findings are present in the cord, on its surface or about the central canal. In the optic nerve, exudation, hemorrhage and œdema are seen, possibly also in other cerebral and spinal nerves. Various bacteria are found (*v. s.*), as the pyogenic organisms, the pneumococcus, and the meningococcus. Mixed infections are not uncommon. It must be remembered that pathologically (though not bacteriologically), the suppurative and epidemic forms are identical, and some writers still maintain that the epidemic is but a subtype of the suppurative variety.

Symptoms.—Prodromal phenomena are ambiguous and vary with the causal disease; otitis, pneumonia or pyæmia may long overshadow or totally obscure meningeal manifestations. Complete *latency* is possible, as in a case seen by the author with Dr. Besley; a convalescent pneumonia patient, while dining, felt dizzy and died in a few minutes. The autopsy revealed a massive, purulent cerebrospinal meningitis, which had produced not a single sign or symptom. (*a*) The *onset* may be sudden with chill and fever. (*b*) *Headache* is usually the first and most prominent symptom, and is often associated with vomiting. (*c*) The *sensorium* is disturbed, there is vertigo, and delirium which is succeeded by stupor and finally by coma, in which, however, headache may still be evidenced by the patient holding his head. (*d*) *Convulsions* may occur; they are particularly equivocal in children. (*e*) *Optic neuritis* may develop if the course of the disease is not too precipitate. (*f*) *Rigidity* of the *neck and spine* is soon noted as well as (*g*) *hyperæsthesia*, hyperalgesia and muscular rigidity. (*h*) *Fever* is usually present, although in very rare instances (*v. s.*) it may be absent. It is usually higher than in other types, perhaps 104° to 105°, and rather more continuous. Toward the end it may be 107° or it may even drop to subnormal. (*i*) The pulse is usually faster and often irregular. (*j*) *Irritation* and *paralysis* of the *cerebral nerves* develop, (they are considered more fully under the epidemic form), and are manifested by ocular paralysis, nystagmus, strabismus, pupillary inequality and tardy reaction to light, facial paresis, grinding of the teeth or trismus especially if the inflammation is basal. (*k*) If the process involves the convexity, *cortical irritation* may be manifested by Jacksonian epilepsy and mono- or hemiplegia (which is sometimes due to reflex inhibition). (*l*) *Lumbar puncture* (see DIAGNOSIS) reveals purulent or clear fluid and frequently the organisms of suppuration. Increased tension is also observed. (*m*) *Kernig's sign* (see DIAG-

NOSIS) is present. (*n*) There may be other symptoms. Constipation is usually present. The abdomen is often retracted, there may be vomiting, the urine is febrile and frequently contains albumin and peptone. The course is generally rapid; it lasts a few hours to more usually a few days (two to ten) and almost invariably results fatally in coma. The diagnosis, differentiation and treatment are considered with the other types.

II. Epidemic Cerebrospinal Meningitis.

This form has been definitely recognized only during the last century. Though many authorities, as Schultze, Dieulafoy and Netter, deny that it is an absolutely independent affection, it is usually described as a *specific infection* involving the cerebrospinal meninges, caused by the *Diplococcus intracellularis meningitidis* and occurring *epidemically* or *sporadically*.

Bacteriology.—The meningococcus resembles the pneumococcus and gonococcus. It lies in pairs or even tetrads, its contiguous sides are flattened, and it resembles two coffee grains with their flat sides apposed. It is wider than the pneumococcus and less lanceolate. It is usually but not always intracellular, being enclosed in the polymorphonuclear leukocytes. Unlike the pneumococcus, it usually has no capsule, although one may develop in serum cultures. It decolorizes by Gram's method, although Jäger has sometimes observed the contrary. Cultures do not show very active growths; it develops best on agar or blood serum as white, viscid, shining colonies. It grows poorly on gelatin. Subcutaneous inoculations are usually negative, therein differing from the pneumococcus, but it produces inoculation-meningitis, and peritonitis. The meningococcus is found in the nose, from which it is thought to invade the brain. Lord found it in the nose, in cases with and without meningitis. Goodwin and Scholly (1906) isolated the cocci from the nasal mucus in 50 per cent. of meningitis patients and in 10 per cent. of those in close contact with them. Its presence has been demonstrated in the blood (in 25 per cent. of cases, Elser), kidneys, joints, spleen, ear, tonsils, lymphatic nodes, pleura, lung, heart valves, pericardium and more rarely in the sputum and urine; it probably travels by way of the lymph vessels.

Predisposing Etiology.—Children and young adults are especially predisposed; it occurs more often in males, is apparently more frequent in the country, although it is often endemic or epidemic in barracks, prisons, hospitals and tenements. The disease has closely followed the movements of armies or regiments. Though not very directly contagious, its contagiousness seems proven beyond question. As many as seven members of a family have been successively stricken. Most cases are seen in the late winter and early spring; the author has always seen some cases in these months. One thousand deaths occurred in New York in the first four months of 1905. Trauma, overwork, and depression are but predisposing causes. Like pneumonia, it often attacks the robust. It may recur; Councilman collected five instances of recurrence. The disease is also seen in the horse and goat.

Pathology.—The findings in the main are those of the suppurative form, *viz.*, those of an acute fibrinopurulent, or less often a seropurulent leptomeningitis. The cerebral dura is seldom, and the spinal dura often, involved. The inflammation follows the lymphatics and vessels penetrating the brain and cord, resulting in encephalitic or myelitic foci, small abscesses, parenchymatous and interstitial changes. The axis cylinders are sometimes lost. Flexner and Barker observed rows of cells "like miniature tubercles," of which some were two to eight times the size of the leukocytes. The exudate is rich along the fissures, fossa of Sylvius and at the base about the chiasm or the surface of the pons and cerebellum. Hydrops ventriculorum is fairly frequent, in cases of long standing. Some consider that caseation may occur as the beginning of resolution and healing. In other organs the changes are those of acute infection, the muscles are brownish-red, dry and degenerated, there is parenchymatous degeneration in the heart and kidneys, and sometimes acute splenic tumor, arthritis, pneumonic foci or swollen glands in which (*v. s.*) the coccus may be found.

Symptoms.—The period of *incubation* is not definitely known, but lasts from a few hours to several days (three to eleven). Prodromes are vague, as malaise, or headache. Strümpell and others observed initial coryza. The onset may be gradual or sudden, sometimes with a chill and usually with fever. In general, the course is febrile, attended by brain irritation; by an almost constant triad of symptoms, *headache, rigid neck* and *hyperæsthesia;* by vomiting, mental, motor and sensory symptoms, and in most cases death results from paralysis, convulsions or coma.

1. General Nervous Symptoms.—(*a*) *Headache* is almost invariable, though it is sometimes absent in very young children, in alcoholics and in sepsis. It develops early, is most severe and is at first localized (frontal or occipital), but soon becomes generalized. Though it varies somewhat in intensity, it is constant, with exacerbations, and provokes the short, plaintive, clear meningeal cry, *cri hydrencéphalique* (Coindet, 1817), which is largely heard in children. The headache is throbbing or lancinating, even under narcotics, and persists after delirium and even coma have set in. Its cause may be compression of the dura, changes in the brain, inflammation of the nerve trunks, or ventricular exudation. (*b*) *Hyperæsthesia* or hyperalgesia affects the special senses (causing intolerance of light and noise), or general sensation (affecting the arms most, the trunk less and the legs least). It is rarely absent, is always suggestive, and is probably due to root neuritis. (*c*) *Rigidity of the neck and spine* is caused by inflammation of the nerve-roots, possibly from lesions in the pons or medulla. The *head is retracted*, sometimes so severely that the occiput lies between the lders, producing decubitus. The entire body may be lifted by head. Swallowing is rendered difficult. Marked *opisthotonos* may resent. Flexion and extension of the head are impossible, although ral movement is sometimes possible. Retraction, although absent ceptional instances, is one of the cardinal triad. Closely associated caused in the same way, is severe *spinal pain with tenderness,* fly in the neck and loins, which is felt rather to the side of the e than directly over it. It may radiate to the trunk and in 33 per . of cases to the extremities, and may affect every muscle. (*d*) *tractures of the limbs* are caused by root inflammation, increased sure, or lesions in the pyramidal tracts. *Kernig's sign* (see DIAG-is) comes under this head. (*e*) *Mental symptoms* often occur early, cially in children, neurotics and alcoholics; they include unrest, mnia, delirium (sometimes with periods of normal intelligence), mania which is followed by stupor and incomplete coma. (*f*) *niting,* in the initial stage, is due to vagus irritation; later it results n ventricular hydrops or increased cerebrospinal pressure. It is he cerebral type, *i. e.,* it occurs without nausea, without relation to ng, is often projectile and is not amenable to gastric therapy. niting is more typical and common in children. The tongue may r become coated and foul. (*g*) The *pulse* at first is increased, and ometimes disproportionately rapid, considering the fever. Some-es it slows later because of brain pressure but ultimately it is fast in. It may be irregular in rhythm and oscillating in rate. Friis ed variation from 84 to 144 within a minute. (*h*) *Respiration* is gular in 20 per cent. of cases; Cheyne-Stokes breathing is not mon, save toward the end. Deep breathing with apnœic pauses ot's breathing) is quite common, though less so than in the tuber-ous type. (*i*) *Convulsions* are most common in the young; they y occur at the onset or later. They are important, but ambiguous n their frequent occurrence in other diseases of childhood.

. **Focal Nervous Symptoms.**—These are partly irritative and partly alytic. (*a*) *Symptoms relating to the cranial nerves* are as follows: ese nerves are most involved in basal localization, while the limbs most affected in that of the convexity. The optic nerve is fre-ntly inflamed, if the disease lasts over four days. The disk is llen and its margin is "washed" (see Plate V, Fig. 4). Neuritis ss frequent than in tumor; hemorrhage and choking are uncom-n. The ocular muscles may be involved. The pupils at first are lly small, often unequal, and sometimes variable like the pulse er they widen spontaneously or on movement of the head, and ation of the skin, but do not react to light. These as well as following changes, result from cortical or basal disease. Ptosis, opia, nystagmus, and strabismus (spasmodic or paralytic) are uent but often vary from time to time. Complete paralysis of third nerve is not common. Involvement of the fifth nerve is sual, although trismus, grinding of the teeth and trigeminal neural-have been noted. Involvement of the facial nerve is frequent, ranking next to that of the third nerve. If diseased at the base, its paralysis is usually total; if at the cortex it is of the cerebral type, and its upper third escapes. The paralysis varies from time to time (*v. s.*). The facial expression shows suffering, the brows are slightly elevated and the forehead is corrugated. The *risus sardonicus* is not frequent. The auditory nerve is often bathed in pus, and hemorrhagic inflammation in the labyrinth results from extension. The tongue on protrusion may deviate to one side; dysarthria or dysphagia are sometimes seen. (*b*) *Symptoms relating to the limbs* occur as follows: Paralysis of the limbs is not very frequent, although Belor noted it particularly in one epidemic. Hemiplegia is rarely complete, occurs with or without participation of the tongue and face, and is not always caused by involvement of the cortex or changes in the pyramidal tracts, but sometimes is apparently due to reflex inhibition. It is frequently accompanied by rigidity of the limbs and aphasia. It is most frequent in children, occurs early or late and may be temporary or permanent. Monoplegia is not common and most often concerns the face or arm. Choreiform or athetotic movements, twitchings and tremor are occasional. Convulsions are frequently Jacksonian in type. Sometimes the paralyses are spinal, as shoulder atrophy or paraplegia, which also involves the functions of the bladder and rectum. The tendon reflexes, as the patellars, may be increased at first and are often decreased or abolished late in the disease. The reflexes of the skin are variable.

3. General and Somatic Symptoms.—(*a*) There is almost always *fever*, though afebrile cases exceptionally occur. The temperature rises suddenly, is sometimes very high and although it follows no definite cycle, lysis is usual in cases which recover. An inverse form is sometimes seen. A temperature of 107° or higher may be registered before death. (*b*) The *blood* shows leukocytosis, which is early, constant and of no prognostic import; the white cells may number 25,000 to 40,000; the meningococcus has been found (*v. s.*). (*c*) The *abdomen* is often retracted, and is scaphoid, especially in the iliac regions, although less so than in the tuberculous type. It may be due to retraction of the abdominal walls, but Traube gave the best explanation, namely that the intestines are tonically retracted from irritation of the vagus, which also causes the constipation. (*d*) The *spleen* is often palpable. (*e*) The *urine* is febrile, albuminous (33 per cent.) and may show peptonuria, hæmaturia (in malignant cases), and rarely glycosuria or polyuria (Traube). (*f*) The *nutrition* suffers, which Leube explains as due to cerebral influences. (*g*) As to the *skin*: the *tâche cérébrale* described by Trousseau has no differentiating value, for it occurs in many febrile and other conditions. Eruptions are more common in the epidemic than the sporadic cases, and vary with the individual epidemic. Herpes (40 to 75 or even 90 per cent.) varies with the epidemic; it is most frequent on the face, but may develop anywhere; it spreads more rapidly than in any other disease (v. Ziemssen); in one case the author saw it extend rom the lip to the ear, neck, chest and shoulder. Purpura, which ave the name "*spotted fever*" to the disease, occurs in the more severe ypes. Other eruptions, as erythema, roseola, urticaria or gangrene re uncommon. (See General Diagnosis for lumbar puncture.)

Clinical Forms.—Hirsch distinguishes (*a*) the *acute* or subacute type, he ordinary form in which the acute symptoms of onset are followed y the general nervous symptoms enumerated, then by focal nervous ymptoms, somatic symptoms, death in coma, or slow recovery; (*b*) he *foudroyant form*, with violent onset, rapid course and death in a ew hours (three to thirty-six) with apoplectic symptoms, or purpura, igh fever, irregular heart and early coma; (*c*) the *abortive form*, with ow fever and symptoms so mild that the disease is recognized only y its association with an epidemic; the ambulatory type is included ere; and (*d*) *Heubner's chronic form*, which lasts several (two to six) onths, with marasmus, and recurrent fever.

Complications.—These vary with the epidemic. Pneumonia, bronhitis, tonsillitis, pleurisy, parotitis, endo- and pericarditis, arthritis J. Jackson, Sr.) and nephritis are the most common. Pneumonia ay be due to the pneumococcus, but the meningococcus has been ound in small isolated foci in the lung (Councilman). The arthritis s either poly- or mono-articular, simple or suppurative, and may heal ven when suppurative; it occurred in 25 per cent. of Flexner's and arker's series.

Sequelæ.—Mental alteration, paralysis, contractures, obstinate eadache, optic atrophy, deafness or Ménière's syndrome may remain fter recovery. Chronic hydrocephalus, paroxysmal headache, vomitng, pains, and convulsions (v. Ziemssen) may result. In an epidemic n Heidelberg, Moos found as sequels deaf-mutism in 60 per cent., bsolute deafness in 31 per cent. and an atactic gait in 50 per cent. f the cases.

Prognosis.—The average mortality is 30 to 40 per cent. (37 per cent., irsch in 15,632 cases), but varies with the epidemic, and may reach 5 per cent. Fifty per cent. of the deaths occur in the first five days. ecovery is exceptional, although possible, after deep coma, repeated onvulsions, high fever or paralysis of the vagus. Obstinate vomiting, isturbed respiration, sudden drop of temperature and inanition are minous signs.

III. Tuberculous Meningitis.

Etiology.—Most cases occur between the second and tenth year, me cases develop in adolescence, and few late in life; at least three mes as many cases occur in children as in adults. Tuberculosis in ildren is usually generalized, and miliary tuberculosis is in 80 per nt. associated with meningeal disease. Tuberculous meningitis is most always secondary, and follows pulmonary or glandular tuberlosis, less frequently joint disease, genito-urinary, brain tubercle and her localizations. Extension by contiguity from the nose, eye, or spinal cord is possible. Strümpell and Leube believe that infection may creep from the pleura, along the nerve sheaths to the spinal cord, whence it reaches the brain. Heredity is clearly a factor in some cases. Measles, whooping-cough, typhoid or pneumonia may be the proximal cause, for the virus of the disease seemingly arouses latent glandular or other tuberculous foci. Trauma is not a frequent cause but is of distinct clinical and medico-legal importance. In the case of a baggage man thrown upon his occiput by the sudden stopping of the train, an acute tuberculous meningitis developed, which resulted in death in three weeks. The autopsy disclosed an apical focus, from which the bacilli infected the injured brain. In a case seen with Dr. J. H. Hess, the affection immediately followed an anæsthetic (given to examine a tender spine), and was fatal within a week.

Pathology.—The diagnosis is made *macroscopically* in the majority of cases, although in some instances the changes are slight. The exudate is gray, yellow or green, gelatinous, sticky, purulent or serous, and covers the base (basilar meningitis) from the chiasm to the medulla. Tiny tubercles are found on the basal nerves and deep in the fissure along the Sylvian artery where "they look like lice eggs on a hair." Careful search is sometimes necessary for their detection. They are most clearly seen in the translucent arachnoid, and on superficial examination look like minute air bubbles. They are seen quite frequently over the convexity and may cause adhesions between the two hemispheres. The ventricular fluid is increased in 80 per cent. of cases, and usually measures a few ounces; tubercles and inflammation of the ependyma are not uncommon; the external and internal communications of the ventricles may be obliterated. The substance of the brain shows the same changes as in other forms, as ischæmic or encephalitic softening or sometimes large tubercles. The cerebrospinal fluid is usually increased and is clear or cloudy. In the cord the exudate may be a quarter of an inch in thickness, and is largely disposed over the posterior part of cord because of the patient's dorsal decubitus. The cord membranes are almost regularly affected, and sometimes appear granular, as "though sown with fine sand." Tubercles in the substance of the cord are not considered common, but Wartmann found them in 87 per cent. of his cases. The cranial and spinal nerve sheaths are pyriform and bulging. *Microscopically*, exudation is found as in the other forms above described. The nodes are proven to be tubercles; bacilli are found in them and in the cerebrospinal fluid. The microscope differentiates between tuberculous inflammation and tuberculosis of the meninges (without inflammation), and again in some cases proves the existence of tuberculous meningitis not visible to the naked eye. Neuritis of the cranial and spinal nerves is constant. The optic, oculomotor and facial nerves are the most often affected. In the cortex, base and ependyma of the brain the usual histopathology of tubercle is seen, with tuberculous endarteritis from mural inflammation (Hektoen), and periarteritis, small encephalitic and myelitic foci in practically every case, vessel thromswelling of the neuroglia and degeneration of the nerve cells f the lateral columns, especially of the axis cylinders.

nptoms.—*Prodromes* are more prominent in children than in s. Fever, emaciation and anæmia are due to preëxisting lung or ular tuberculosis. Irritability, change in disposition, tendency to ability in children to play or in adults to work, unrest, insomnia, che, nausea and constipation may develop in a week or two or e to three months. Trauma, measles, and pertussis may be the ent cause. The outbreak develops with much the same symp- as those described under the epidemic form, but they appear gradually, so that the classical description comprises *three stages:* he *stage of irritation*, with headache, vomiting, remittent fever ing 102° in the evening, the *cri hydrencéphalique* of Coindet, terrors, pain and rigidity in the neck and spine, small irregular s, delirium, grinding of the teeth, hyperæsthesia, rigidity of the and Kernig's sign; (*b*) the *stage of brain pressure*, with paralytic rritative conditions in the eye muscles, as ptosis or strabismus; paralysis of other cranial nerves or of the extremities, as mono- i, especially of the face, or hemiplegia from involvement of the c, and very frequently aphasia and dulness; with general or onian convulsions, slow, irregular and quite variable pulse; ted abdomen, constipation, continued vomiting; sighing respi- i; and very frequently with optic neuritis; and (*c*) the *stage ralysis*, in which the vomiting and often the headache cease, ensorium is dulled, absolute coma develops, the pulse becomes and vacillates in rate and rhythm. Biot's or Cheyne-Stokes hing appears, emaciation is extreme, the pupils are wide and bile, the eyeballs roll upward, and a typhoid state with dry e or abdominal distention exists. Temporary remissions of days, s, or even a year are occasional in which the pulse remains lar, but the patient lapses again into coma, the fever rises very (107°) or falls low (even to 93°), and death occurs in several s from cardiac or respiratory failure, dysphagia, inanition or stasis. This division into stages is not wholly satisfactory, se the symptoms of one stage may merge into those of another he progression of symptoms is somewhat as described.

bercles in the choroid were first found anatomically by Autenrieth), and Guéneau de Mussy (1837), and clinically by Jäger, z, and Busch; they are rare clinically, being found by Garlick per cent. and Krämer in 13.6 per cent. of cases. Heinzell did nd them in 41 cases. At postmortem examination Litten found in about 75 per cent. (See Plate VI.) *Leukocytosis* is usually t, unless it occurs during the agony or from mixed infection. pleen is enlarged in 33 per cent. of cases.

ny variations exist. The process may be wholly *latent*, being ed by miliary tuberculosis (or meningeal symptoms may dominate the greatest pathological changes are in the lungs). Coma may dden and the disease may run a short course, resembling apoplexy, especially in adults; in these cases the hemiplegia probably results from changes in the arteries. It may appear to be a psychosis. In adults the course is more rapid, the headache and delirium more marked and the convexity more frequently involved than in children.

Death is usual, but rare recoveries are recorded by Waterhouse, Ord, Leube, Fürbringer, Dujardin-Beaumetz, Freyhan, Rilliet and Barthez, Jansen, Carrington, Hensch, Politzer, and lately by Gross, even after bacilli were found by lumbar puncture or after choroidal tubercles were demonstrated.

IV. Serous Meningitis.

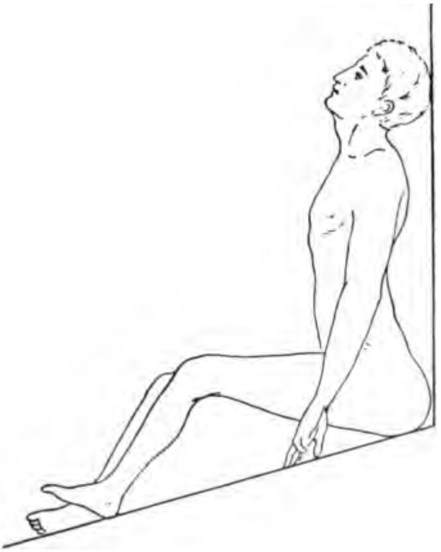
This is more frequently seen at the bedside than at the autopsy. It is often tuberculous. The inflammation is slight, the membranes are glistening, clear or flocculent fluid is seen in the arachnoid meshes and ventricles, which may be the sole seat of the disease, and the pia especially shows exudation. It is often mistaken for œdema of the brain. The microscope may be necessary to demonstrate inflammation, which frequently is circumscribed. The process sometimes occurs with encephalitis, poliomyelitis, Landry's ascending paralysis, typhoid or pneumonia.

Symptoms.—In the serous (non-suppurative and non-tuberculous) form, no definite symptoms can be outlined. The disease certainly exists, although it is relatively infrequent, but with equal certainty it is too frequently diagnosticated. Headache is common; vomiting, rigidity, fever, slow and labile pulse, optic neuritis, hyperæsthesia, spasms, and paralysis of the cerebral nerves are much less frequent than in the other forms of meningitis though they are occasionally observed. The symptoms resemble those of brain tumor. Lumbar puncture in some instances has given absolute relief; the prognosis is most favorable in this type.

General Diagnosis of Meningitis.—The *first step* is the diagnosis of meningitis, which in general has an acute or subacute onset, with signs at first of general, then local inflammation of the surface of the brain. The *second step* is determination of the variety (suppurative, epidemic, tuberculous, primary or secondary), or the cause, and the locality (convexity or base).

Cardinal Symptoms.—The *early triad* of (*a*) headache, (*b*) retraction and rigidity of neck and (*c*) hyperæsthesia, is important and develops early. (*d*) *Other general symptoms*, as causeless vomiting, abdominal retraction, pulse and respiratory changes, fever or convulsions, are important in their grouping. (*e*) *Lumbar puncture* (Quincke, 1890) is of diagnostic value. Its *technique* is as follows: The patient lies on the side with the knees drawn up and the body bent as far forward as spinal rigidity allows; with or without chloroform, an aspirating needle or small trocar is introduced in the median line in children, and a quarter to half an inch to one side in adults, between the third and fourth lumbar vertebræ (Quincke) or between the last lumbar tebra and sacrum (Chipault), and is pushed forward toward the dian line and a little upward, one and a half inches (2½ cm.) deep children and 1.5 to 2.5 inches (4 to 6 cm.) in adults, until it passes the canal. Fürbringer prefers to have the patient in the sitting ture. Not more than 4 to 5 drams (15 to 20 cm.) should be with- wn, lest fatal results ensue. Negative taps result from needle lusion by clots. *Results:* (i) normally, the pressure with the patient he lateral position, equals 100 to 150 mm. of water and the escap- fluid only trickles out. In meningitis, the *pressure is greatly in- ised* (200 to 700 mm.) and the fluid often spurts out. (ii) The mal fluid is clear, has a specific gravity of 1,007–09 and contains e albumin (½ to 1 pro mille). Though the fluid may be clear in all ms of meningitis, it is usually clear in the serous form, but *cloudy, culent* or sometimes bloody in other forms; in the tuberculous iety it is usually clear or slightly yellow with a whitish clot forming standing; in the epidemic form, it may be turbid or clear, with a lowish clot (Netter); in the purulent type it is opaque, and in rare es it is pure pus. The specific gravity is about 1,010 in the serous, 11 in tuberculous, and 1,015 in purulent or epidemic variety, al- ough these figures are not absolute. The percentage of albumin is small he serous, 2 pro mille in tuberculous, and 3 or even 8 or 9 pro mille in suppurative forms. (iii) *Bacteriologically* (see BACTERIOLOGY) in purulent type the strep- occus, staphylococcus, eumococcus, typhoid and on bacilli are found; in epidemic variety, the men- gococcus, which was posi- e in 100 per cent. in the t New York epidemic, is esent; in the tuberculous pe, tubercle bacilli exist in per cent. of cases as an aver- e (73 per cent. Bernheim d Mosler, 81 per cent. Für- inger, and 100 per cent. oltzmann); even in this rm, mixed infection with e pus organisms and men- gococcus may be seen; the bercle bacilli are often found cover slip preparations; ltures grow too slowly for inical diagnostic value. (iv) he *cellular morphology* (*cyto- agnosis*) is a valuable aid. the purulent and epidemic form the leukocytes are largely polymor- onuclear; in the tuberculous form the lymphocytes predominate,

FIG. 62.



Kernig's sign, showing retraction of head and back and prominence of knees in the erect posture, *i. e.*, the impossibility of extension of knees. (Dieulafoy.)

according to Widal, Sicard, and Rivault (see CYTODIAGNOSIS, PLEURISY); some dissension exists on this point, as well as on the statement that iodine given by mouth is found in the tuberculous and not in the purulent (epidemic) exudate.

(*f*) Kernig (St. Petersburg, 1882), described a sign known as *Kernig's sign.* When the patient sits, it is impossible (in 75 to 95 per cent. of all meningitis cases) to extend the knees, as shown in FIG. 62. The legs and thighs are flexed (flexor contracture) and the knees cannot be pressed down, *i. e.*, cannot be extended when the patient is sitting but this is possible when he is lying. It indicates that the spinal meninges are involved. It lasts well into convalescence, whence as Netter states, "a retrospective diagnosis may be made." It occurs at times in subdural hemorrhage, sinus thrombosis, encephalitis, and cerebellar tumor. It is sometimes unilateral. It may possibly indicate degeneration of the pyramidal tracts.

(*g*) *Local signs* usually follow the general signs, and are more important. They include the pupillary changes, strabismus, facial paralysis, and hemiplegia. Often their late development lessens their value for early diagnosis.

	EPIDEMIC FORM.	TUBERCULOUS.	SUPPURATIVE
Onset:	Sudden, with few prodromes.	Longer prodromes; family history; other evidences of tuberculosis.	Sometimes primary but oftenest secondary to ear disease, acute infections, trauma, pneumonia.
Fever:	Fairly high.	Less; inversed type frequently; sometimes absent.	Especially high, with chills, perhaps pyæmic type.
Eruptions:	Herpes and purpura very common.	Rare.	Rare—polymorphous septic rashes.
Leukocytosis:	Regular and marked.	Exceptional.	Frequent.
Rigidity of neck:	Most frequent.	Spinal symptoms somewhat less frequent.	Sometimes absent (pneumococcic form).
Other symptoms:	Peptonuria.	Choroidal tubercles.	Peptonuria.
Course:	Rapid—more recoveries.	Subacute, slower, fatal.	More acute, very rarely recover.
Lumbar puncture:	Meningococcus; leukocytes polynuclear.	Tubercle bacilli; mononuclear.	Pyogenic cocci, pneumococcus; polynuclear.

Diagnosis of the Variety.—This is more difficult. Cases of tuberculous meningitis sometimes increase when the epidemic form prevails, and the meningococcus may be found in these cases (Heubner, Lenharz, Netter). Sporadic cases, due to the meningococcus, may be indistinguishable from primary pneumococcic meningitis, save by lumbar puncture.

Diagnosis of Location.—(*a*) Localization on the *convexity* results from trauma, or metastatic infection; it is characterized by more frequent delirium, Jacksonian convulsions, mono- or hemiplegia, less cranial nerve paralysis and less optic neuritis. (*b*) Localization at the **base** results from the tuberculous form, chronic nephritis, cachectic states, and the non-tuberculous forms which occur in childhood; it is characterized by more frequent cranial neuritis, and optic neuritis. (*c*) The *ventricular* form rarely involves the cranial nerves.

Differentiation.—1. From nervous toxæmic symptoms, as those of typhoid (*q. v.*). Jenner pointed out that in acute infections the headache ceases when delirium begins. This is also true of the pseudomeningeal symptoms of pneumonia, typhoid, sepsis, etc. Some cases with focal symptoms are most confusing (see URÆMIA), but the albuminuric retinitis, the cardiovascular changes, and albuminuria, casts, and deficient elimination are usually sufficient for distinction. Acute *inflammation of the labyrinth* may resemble meningitis, for it sometimes produces headache, vomiting, fever, convulsions, stiff neck, and even optic neuritis. If labyrinthitis involves the meninges, the facial nerve is likely to be affected. Such conditions resembling meningitis, Dupré (1894) called meningismus, and Bouchut (1895) pseudomeningitis.

2. From Brain Disease.—*Syphilitic meningitis* (*q. v.*), *brain abscess* (*q. v.*), cerebral hemorrhage (*q. v.*), and encephalitis (*q. v.*), have been considered. Great difficulty is sometimes experienced in differentiating between slow tuberculous meningitis and *tumors* of the brain, as tubercle of the pons, or rapidly growing glioma. A moderately severe optic neuritis may suggest tumor or meningitis; intense neuritis, with choking, swelling and hemorrhage is indicative of tumor. Paralysis is more sudden in meningitis than in tumor.

3. From Hydrencephaloid (see BRAIN ANÆMIA).—4. *Hysteria* is marked by its usual stigmata, *viz.*, psychical alteration, limitation of the field of vision, anæsthesia and other stigmata. Hysterical strabismus is always spastic and convergent, and is often associated with small pupils; meningitic strabismus is often divergent (always a sign of organic disease), and the pupils are unequal.

Treatment.—Prophylaxis and treatment of the causal disease are indicated. The patient should be kept quiet in a dark room, the head elevated without flexion, an ice-bag applied to the head and spine, or an ice-pillow should be used. *Nutrition* should be maintained by feeding with the nasal tube or by nutrient enemata, if retraction of the neck causes dysphagia. The *bowels* should be mildly relaxed, and the bladder should be watched. *Vomiting* is treated as in acute gastritis (*q. v.*), but therapy is usually unavailing, for the cause is cerebral irritation; of drugs the bromides are customarily most servicable. Feeding by the rectum, sucking of ice, and the remedies indicated for the headache give the greatest relief. Counterirritation by blisters and the use of the cautery are dangerous because the skin sloughs readily. *Headache*, restlessness and convulsions necessitate the use of opiates, though theoretically opium congests the brain and its membranes. The hypodermic use of morphine, or the use of the deodorized tincture of opium by mouth, is necessary, and free from harm; no other analgesic is equally potent. Ergot should relieve the congestion, but it never replaces the opiates. Atropine reinforces the analgesic action of opium. Chloral is used chiefly for the convulsions and for unrest. *Warm baths* were first recommended by Aufrecht and Waroschilsky, and in the epidemic type are considered almost specific by Netter. *Stimulation* is indicated when the heart weakens, and coma is imminent, until which time alcohol should be withheld. *Resorbents*, as iodides, are of little value, (although Niemeyer recommended them). Mercurial inunctions are regarded as inefficacious, although milder types of meningitis, as the serous form, are apparently benefited.

Lumbar puncture can permanently relieve only the simple or serous forms. Its repeated use may induce punctate hemorrhages, although sudden death is much less likely to occur from its use than in uræmia or brain tumor; isolated cases of recovery in the pneumococcic and tuberculous forms have followed repeated puncture. *Surgery* is indicated in circumscribed rather than in diffuse meningitis. Macewen, Jaboulay, Greave, Gluck, Joel, and Barker, have operated with, as a rule, no remarkable results. Macewen saved all of six cases of suppurative cerebral meningitis, and one of six cerebrospinal cases. Ventricular puncture, first recommended by Wernicke, in 1883, lapsed into disuse and was needlessly revived by Horsley, Paget, Rickett, von Beck, and Benninghous. *Diphtheria antitoxin* has been used (Wolff), but its exact benefit is still uncertain.

CHRONIC LEPTOMENINGITIS.

Etiology.—(*a*) It may result from acute leptomeningitis. (*b*) The leptomeninges thicken after trauma, in atrophic and sclerosing brain disease, as paretic dementia, hydrocephalus, and softening, and in alcoholism, gout, nephritis, or plumbism. (*c*) Chronic productive inflammation occurs in syphilis, tuberculosis and suppuration.

Pathology and Symptoms.—The leptomeninges are diffusely or locally dense, thick, opaque and microscopically show fibrous tissue. In the chronic tuberculous form, the membranes are thick, fibrous, and caseous; they compress the cortex, especially the psychomotor areas, and produce generalized and Jacksonian convulsions, headache, monoplegia, aphasia, and mental changes. It may sometimes heal. In the chronic suppurative form, the pus loculi are situated in a firm, fibrous tissue. The symptoms, if cortical, are like those of the chronic tuberculous form; if basal, nerve paralysis and optic neuritis are common. In the *chronic simple form* (Gee and Barlow) the process may begin acutely and may persist, especially at the base and in the posterior fossa. Possibly it is a sequel of a localized epidemic type (this theory was rather favored by Koplik in a recent paper), or of hereditary syphilis, trauma, or otitis. It is seen especially in children ("simple meningitis of children"); 76 per cent. of cases occur in the first year of life. Rigid neck is the leading symptom, though convulsions and rigidity of the extremities do occur. Paralyses are rare. Fifty per cent. of patients die. The *alcoholic form* is slight, diffuse and cortical; meningitic symptoms are obscured by alcoholic disease of the liver, kidneys, heart or peripheral nerves. Headache is little developed; there is moderate delirium and slight optic neuritis. *Diagnosis* is difficult, and *therapy* is wholly etiological.

DISEASES OF THE SPINAL CORD.

(A). GENERAL ANATOMICAL, PHYSIOLOGICAL, AND SYMPTOMATIC CONSIDERATIONS.

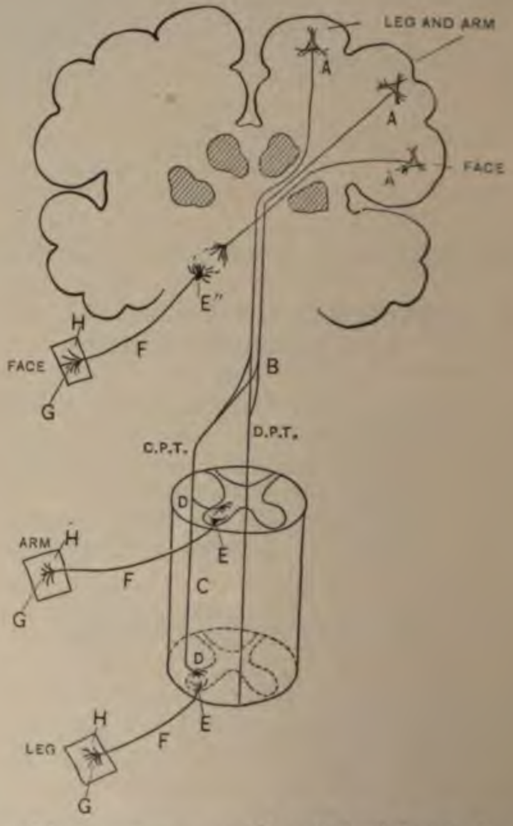
The nervous system is made up of numerous similarly constituted units, called *neurones* (Waldeyer). Each neurone comprises (*a*) a *central nerve cell*; (*b*) protoplasmic processes or *dendrites* from the cell which conduct (cellulipital) impulses to the cell; (*c*) an axis cylinder or *axone* arising from the cell and conducting impulses (cellulifugal) from the cell; and (*d*) the terminal ramifications of the axis cylinder, also known as end brushes or *arborizations*. The axis cylinder often gives off lateral branches, known as collaterals or *paraxones*. The *nutrition of the nerve cell* probably depends upon the integrity of its nucleus, and the nutrition of the cell governs that of its neurone. Disease of the cell causes degeneration of the entire neurone; division of a process or axis cylinder results in its degeneration below the point of separation from the nutrient cell. It is still uncertain whether the neurones are wholly independent of each other, although most authorities favor this view. At times, and especially in the retina, some degree of anastomosis between the dendrites of different systems exists (Dogiel). The nerve cells are closely grouped in the gray cortex, gray substance of the cord and in the ganglia of the peripheral nerves. The axis cylinders largely course through their white substance. To comprehend the most important point in spinal diseases, the localization, we must recognize that pathological processes are in general of two varieties: (*a*) those involving certain neurones, or *system diseases*, as tabes, and (*b*) those not involving given tracts or neurones, or the *non-system diseases*.

The functions of the cord are (1) conduction of motor impulses to the muscles; (2) conduction of sensory impressions to the brain; (3)

certain reflexes; (4) centres governing the bladder, rectum, etc., a
(5) trophic influences.

1. The *motor tract* is composed of *two sets of neurones or segmen*
The *upper segment* has its origin in the cells of the motor cortex (A F
63), and runs through the white substance, *i. e.*, the corona radia
internal capsule, crus, pons, and in the anterior pyramids of the medu

Fig. 63.

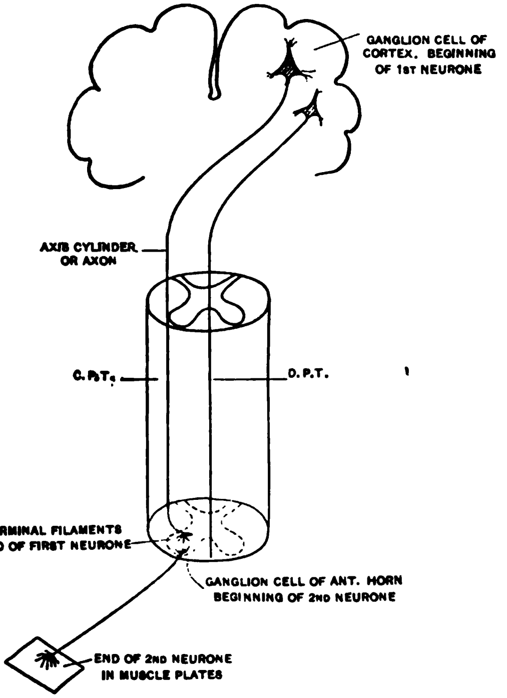


Illustrating the course of the two motor neurones: A, cortical cell of origin of motor tract; B, de
cussation in medulla; C, course in lateral columns of cord: DD, branches to anterior horns
cord; in second (lower) neurone, E, cells of anterior horns; E", cells in pons; F, nerve trun
G, motor end plates; H, muscles.

(whence the name pyramidal tracts), it crosses to the opposite side,
Fig. 63), running down in the lateral columns of the cord (C)
giving off branches (DD) to the anterior horns, where the tract end
fine terminal filaments, which surround the motor ganglion cells in
anterior horn. (For the general course of the upper motor neuron
see Fig. 56). The *lower segment* begins with the motor cell in t
anterior horn (E), the dendrites of which are in contact with the te

minal ramifications of the axis cylinder of the upper segment by contact (Ramón y Cajal) or by concrescence (Held); it extends along its own axis cylinder into the anterior nerve roots and nerve (F), and ends in terminal ramifications (G) (motor end plates) in a muscle of the arm or leg, which is on the side opposite to the origin of the cortical motor centre. From the cells of both segments the motor impulse travels downward from the cell (cellulifugal conduction). About 75 per cent. of the upper motor neurones cross as above described, are called the *crossed pyramidal tracts* (C. P. T. in Figs. 63 and 64), and lie in the *lateral columns* of the cord. Twenty-five per cent. of the motor fibers (rarely 40, even 90 or 100 per cent., Zenner), do *not cross* (decussate) in the medulla, but pass down in the *anterior columns* of the same side and are called the anterior or *direct pyramidal tracts* (D. P. T. in Figs. 63 and 64). The anterior direct pyramidal tract usually ends in the mid-dorsal region, but if small may reach only to the cervical enlargement of the cord, or if large may even reach the fourth or fifth sacral nerves. This tract probably crosses to the anterior horn of the opposite side by way of the white portion of the commissure of the cord. There are probably some other motor tracts in the cord. The upper segment is

FIG. 64.

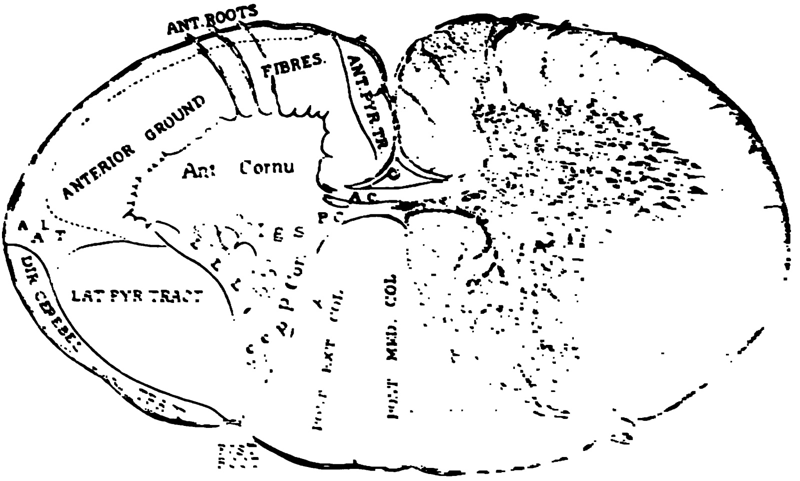

GANGLION CELL OF CORTEX. BEGINNING OF 1ST NEURONE
AXIS CYLINDER OR AXON
C.P.T.
D.P.T.
TERMINAL FILAMENTS END OF FIRST NEURONE
GANGLION CELL OF ANT. HORN BEGINNING OF 2ND NEURONE
END OF 2ND NEURONE IN MUSCLE PLATES

Illustrating the general outlines of the upper and lower neurones and of the direct (DPT), and crossed (CPT), pyramidal tracts.

largely (75 per cent.) crossed, but the lower segment (or neurone) is a direct tract, the cells in the anterior horns supplying muscles of the same side of the body.

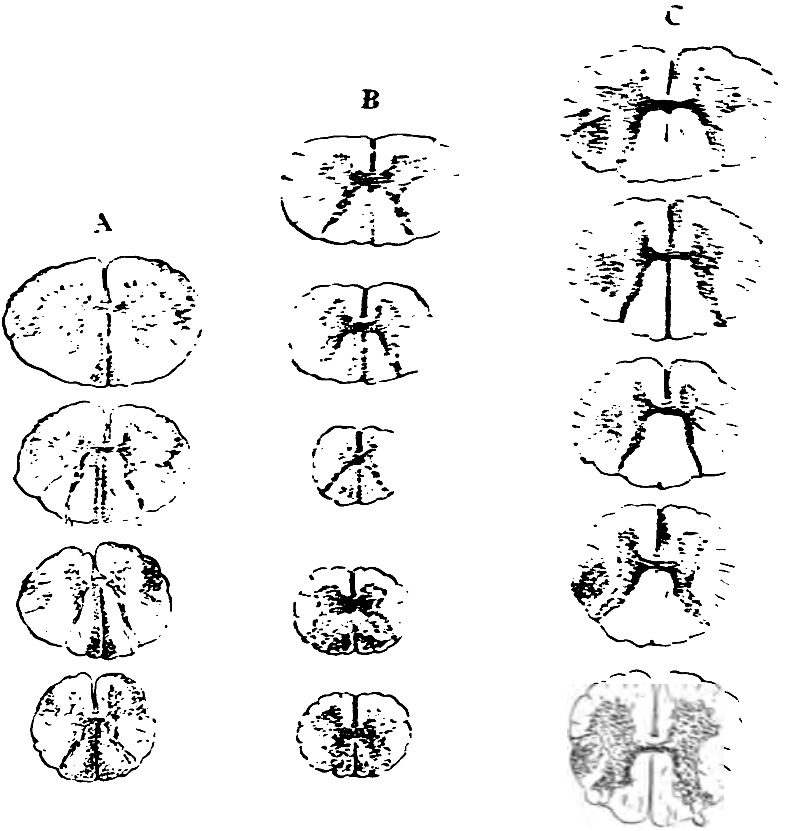
The cord, therefore, contains parts of both neurones, the course of which has been ascertained by the *secondary descending degeneration* which follows neurone lesions. Morgagni, Cruveilhier, and Rokitansky noted some atrophy in the cord after brain lesions which destroyed the motor paths, but Türck first fully described its occurrence and Bouchard, and Barth confirmed his findings. Flechsig, and later Bechterew added information from embryological studies. After a lesion, as hemorrhage in the internal capsule, the fibers of the upper neurone below the lesion show signs of degeneration. In a few days granule-bearing cells appear and somewhat later the axis cylinders swell, degenerate and stain poorly, especially when studied by the Marchi method. These changes occur because the axones are separated from the nutrient c[illegible] cells. The neuroglia tissue [illegible] amylacea develop. Degeneration is seen in the opposite lateral pyramidal tracts, and in the anterior column on the same side as the lesion. Sometimes slight degeneration occurs in the lateral pyramidal tracts of the same side. Pitres, Hadden, Sherrington [illegible] have seen [illegible] of the same side [illegible] Brain [illegible] as their [illegible] is [illegible] Brain [illegible] Pathology [illegible]

FIG. 65.



[illegible]

PLATE XVI.



A. Ascending degeneration in the posteromedian column and anterolateral ascending tracts from lumbar lesion. (After Gowers.)

B. Ascending degeneration after injury to cauda. (After Schultze.)

C. Descending degeneration of pyramidal tracts from right-sided cerebral hemorrhage. (After Mott.)

downward; possibly they are part of the lateral pyramidal rom Deiter's nucleus in the medulla, Russel]. Fibers lying just r to the lateral pyramidal tract also rate downward [from the posterior a quadrigemina, Boyce, and Sakow- Schultze describes a comma-shaped ding degeneration in the anterior of Burdach's column). See Plate

eneration in the Lower Segment.— e of the anterior horns, division or of the nerve trunk, causes degener- of the nerve toward the periphery ophic muscle changes. The degen- nerve responds neither to the faradic, the galvanic current. The muscle ot answer to the faradic current, but galvanic; it answers slowly instead rply (as is the case with the nor- uscle). These differences constitute *ction of degeneration* (see NEURITIS). amputation, the motor nerves and r roots concerned, waste (Bérard, very slowly because of lack of func- r because of peripheral irritation.

two neurones do not correspond in r, because one neurone of the upper nt is connected with several lower es, *i.e.*, various cells in the anterior at different levels are probably ex- by a single pyramidal fiber. For ex movements, as of the hands, are more pyramidal fibers than for movements, as of the intercostal es. The anterior spinal root nerves ith fibers from the posterior roots, assing downward, leave the spinal between the vertebræ, thus form- e spinal nerves, whose area of origin cord is called a *segment.* The peri- l nerves often arise from several seg- , which are sometimes considerably ted; muscles with similar function similar spinal localization, because *ents rather than muscles are localized cord,* as well as in the brain. The ded table gives the localization of the es, with special regard to their cells

FIG. 66.

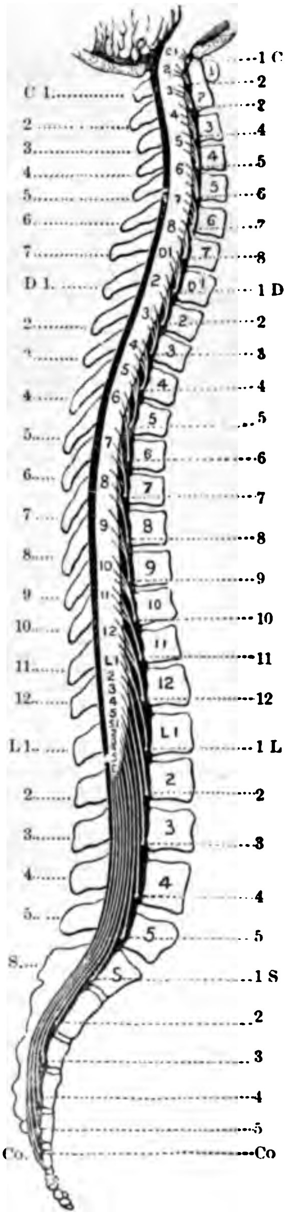
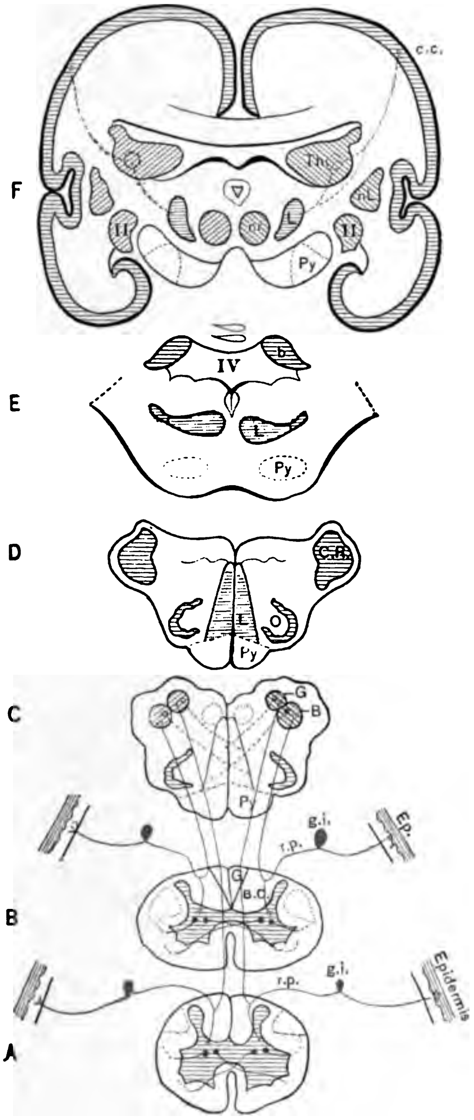


Diagram from Gowers, showing relation of vertebral spines to their bodies and to the nerve-roots.

Segment.	Muscles.	Skin: Sensation.	Reflexes.
2–3 Cervical	Sterno-cl-mastoid, trapezius, scaleni, neck muscles. Diaphragm (3, 4, 5, 6 cerv.).	Neck and occiput.	Sudden inspiration by sharp pressure below the ribs.
4 Cervical	Lev. ang. scapulæ, rhomboideus, supra- and infra-spinatus, deltoid, supinator longus, biceps.	Shoulder (anterior part, Dana).	Widening of pupil from irritation of neck, 4-7 cervical.
5 Cervical.	Supinator brevis, serrat. mag, clavicular portion of pectoralis maj., teres minor.	Radial side of arm (volar and dorsal surface) to the insertion of deltoid. Post. surface of shoulder?	Scapular reflex (fifth cerv. to first dorsal). Tendon reflexes of this group of muscles.
6 Cervical. 7 Cervical.	Pronators, coraco-brachialis, brachialis ant., triceps, long extensors of hand and fingers. Lower neck muscles and middle part trapezius, 6, 7, 8, C and 1 D.	Dorsal and volar surface of radial side of hand to midline of middle finger, and up to base of hand, narrow strip on volar and dorsal surface up to axilla, connecting with above area.	Tendon reflexes of muscles named.
	Costal portion of pectoralis maj., latissimus dorsi, teres maj. Long flexors of the hand and fingers.	Ulnar part of hand (dorsal and volar) from middle of the fourth to middle of third finger and connecting with it, a moderate strip on volar and dorsal surface of arm.	Volar reflex of hand, periosteal reflexes of radius and ulna. Tendon reflexes of muscles named.
8 Cervical.	Extens. poll. long. et brev. Small muscles of hand.	Dorsal and volar surface of hand to middle of fourth finger, narrow strip on dorsal and volar surface of arm up to axilla.	Corresponding tendon reflexes.
1 Dorsal.		Narrow strip on ulnar surface arm and forearm down to base of hand. (The upper part, perhaps, belongs to the second dorsal segment.)	Tendon reflexes.
2–12 Dorsal.	Muscles of back (also lowest part of trapezius) and abdomen. Intercostals D, 1–10.	Chest, back, abdomen and upper gluteal region. (Umbilicus, tenth dorsal: ensiform, sixth to seventh dorsal).	Abdominal reflex. Epigastric reflex (according to Dinkler, the epigastric is at the ninth dorsal, the middle and lower abdominal at the tenth and twelfth, respectively.)
1 Lumbar.	Abdominal muscles, quadratus lumborum. Ileopsoas.	Pubic area, anterior aspect of scrotum.	Cremasteric reflex (1–3 lumbar).
2 Lumbar.	Sartorius (or third lumbar), flexors of hip. Quadriceps femoris (extensors of knee), cremaster.	Anterior and inner side of hip, inner side leg to malleolus, inner side of foot, external surface of hip, lumbar regions.	Patellar reflex (2–4 lumbar). Erection (lumbar cord). Uterus (lumbar cord).
3 Lumbar.	Internal rotators of thigh.		
4 Lumbar.	Adductors of hip, Sartorius (?), Abductors of hip, Tibialis anticus, Calf muscles, Flexors of knee (?), gluteals (extensors of hip).	Posterior surface of hip, thigh, external surface of leg and foot. Back of foot.	Gluteal reflex (4–5 lumbar).
5 Lumbar.	External rotators of hip, Flexors of foot (?), Extensors of toes. Peronei.		
1–2 Sacral.	Flexors of foot and toes. Small muscles of foot.	Skin of sacrum, anus, perineum, genitalia.	Plantar reflex; ejaculation (3-4 sacral). Achilles tendon reflex.
3–5 Sacral.	Muscles of perineum.		Bladder and rectum reflex.

The brackets to the left concern only the muscles, and indicate the origin of the muscles from several nuclei.

PLATE XVII



Scheme of Sensory Conduction. (Strümpell.)

A. Entrance of posterior sensory roots into lumbar cord: *g.i.*, intervertebral ganglion; *r.p.*, posterior root; part of the fibers end in the posterior horns, from whose cells other fibers arise and enter the lateral columns, partly on the same and partly on the opposite side; other fibers from the posterior roots, course upward in the posterior columns and form in—

B. (the cervical cord), Goll's columns (G); B.C., Burdach's columns.

C. Medulla oblongata, G and B are the nuclei wherein Goll's and Burdach's columns end. From these, new fibers arise which decussate and form the fillet (lemniscus, L) in—

D. L, lemniscus; Py, pyramidal tract; O, the olive; *c.r.*, corpus restiforme.

E. Pons; IV, fourth ventricle.

F. L, lemniscus of fillet; *n.r.*, nucleus ruber; Py, pyramidal tract in crus cerebri; N.L., nucleus lenticularis; T.H., optic thalamus (beginning of new neurone to cortex?).

e beginning of the second neurone) as well as of the skin and exes. The cord is shorter than the spinal canal, reaching only to second lumbar vertebra. The nerve roots descend, and therefore ir level at the point of exit does not correspond with their level origin. Fig. 66 shows this relation.

Table giving localization of function in the various spinal segments om Edinger, Starr, von Leyden, Remak, Thoburn, Ferrier, Yeo, wers, and Kocher) will be found on page 1062.

. *The sensory tract* conducts upward and is more complicated and clearly understood than the motor tract, because the interpreta- n of experiments made on animals is frequently confusing. It is mposed of three or more neurones. The cells of the *first neurone* in the intervertebral ganglia, whose cells end in an axis cylinder iding into a branch to the skin, and another to the cord by the pos- or root. These ganglia are the homologues of those of the vagus, trigeminus (*q. v.*). Embryologically studied, it would seem less ly that the axis cylinder of the ganglion cell divided than that the nches from the skin (the peripheral sensory nerves) represented protoplasmic process or dendrites of the ganglion cells, and the nch to the cord represented the axis cylinder. However, all periph- l sensory fibers originate in the ganglia, and almost all fibers of posterior nerve roots come from these ganglia. From the posterior ts the fibers reach the cord, where (*a*) some pass into the posterior umn, (*b*) some into the gray matter, and (*c*) others run to the cells he anterior horns, and are clearly part of reflex arcs.

a) The *fibers entering the white substance* (the *posterior columns*) ide into a short descending branch (which probably is concerned eflex processes, giving off some collaterals and ending in the gray ter), and into a long ascending branch, which is the more important ; they enter the posterior column obliquely and by the entrance of fibers at higher levels become more centrally located in the column oll (posterior median column) in which the lowest fibers (*e. g.*, the tic), are most posterior and the highest (*e. g.*, the cervical) are t anterior. The *posterior columns* convey fibers for *muscle sense* se of posture and movement association) and possibly also for ple tactile sensation. "Sensations we do not feel" travel in these mns (Gowers). The posterior median (Goll's) column ends in nucleus gracilis of Goll (or postero-median nucleus) in the medulla. is uncrossed, *i. e.*, direct. The posterior external (Burdach's) umn ends in the nucleus cuneatus of Burdach (or posterior external leus) in the medulla. It is likewise uncrossed or direct. The first rone ends in these nuclei. The *second neurone crosses* above the tor decussation in the medulla (interior arcuate fibers) to the opposite e and unites with fibers that have already crossed in the cord, thus ming the fillet (see below, *b*). This neurone is crossed. The pos- or nuclei communicate with the opposite cerebral cortex, with the ebellum of the same and opposite side and with the external arcuate rs.

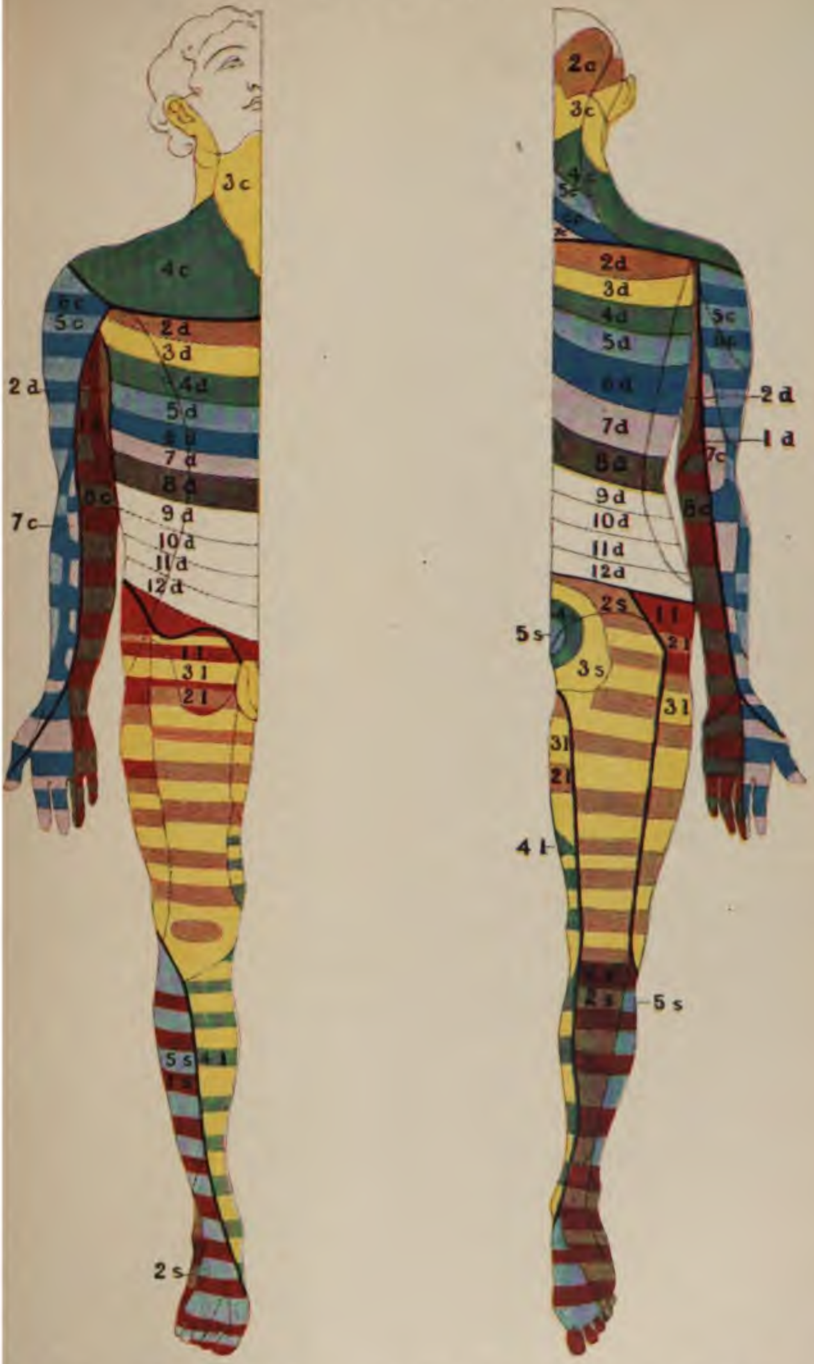
(*b*) The *fibers entering the gray matter of the posterior horns* also divide into ascending and descending branches. Some lie at the zona terminalis, the most posterior part (Lissauer's zone) of the posterior horn. All these fibers end with terminal ramifications about ganglion cells in the gray matter; they form the beginning of a second neurone which crosses in the commissure of the cord and courses upward in the antero-lateral column and anterior ground fibers of the opposite side (just anterior to the lateral pyramidal tracts), and they unite in the medulla with the fibers of the second neurone of (*a*) (*v. s.*). (See Plate XVII. The fibers entering the posterior horn convey *temperature and pain conduction*, and travel between the posterior median column and the central canal. *Tactile sensation* is probably conveyed in the antero-lateral column. Gowers holds that the antero-lateral columns convey most of the upward sensory impulses, since their division causes anæsthesia. Both (*a*) and (*b*) then run in the median lemniscus of the fillet, and in part of the longitudinal bundles of the formatio reticularis of the pons, tegmentum of the crus and posterior limb of the internal capsule, beyond which their course is somewhat uncertain. Some fibers run directly to the motor cortex or back of it to the parietal lobes, while others end in the optic thalamus, whence perhaps they are connected by a *third neurone* coursing to the cortex.

The *sensory tracts degenerate upward* (ascending degeneration). Plate XVI Fig. A shows degeneration in the posterior and antero-lateral columns. Most ascending degenerations stop in the medulla. The *direct cerebellar tract*, which begins at the first lumbar nerve, also degenerates upward. It increases somewhat in size as it rises because of reception of additional fibers, but most of its fibers come from the lower dorsal and first lumbar nerves; some of them come from the gray substance of Clark's columns to which collaterals are sent from the posterior fibers; its function is uncertain, though Flechsig believes that it conveys muscular impressions from the lower part of the trunk and the legs; its destination is the middle lobe of the cerebellum by way of the restiform body; some fibers then cross to the opposite lobe.

The *spinal sensory nerves* descend lower than do the corresponding motor nerves, and skin sensation for a given area is often supplied by two, three, or more nerves, whence anæsthesia results only when all of them are diseased (Sherrington). See Wichmann's plate, Plate XVIII.

Lesions of the *conus medullaris* down to the fourth sacral nerve produce paralysis of the bladder and rectum, and saddle-shaped anæsthesia on the anus, gluteal region, perineum, genitalia, under surface of the thigh (pudendal and coccygeal plexus) but the scrotum escapes because the spermatic plexus and external spermatic nerve connect with the lumbar plexus. The cremaster reflex is normal. In Bernhardt's case, sexual desire and erection were preserved, while ejaculation was incomplete.

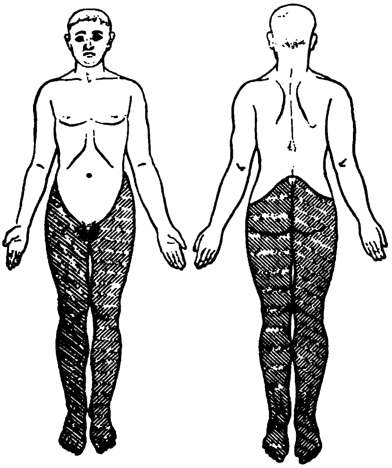
PLATE XVIII.



se figures have been devised by Wichmann in order to show the distribution of the sensory areas
ponding to the segments of the spinal cord. The colors used correspond to those of the normal spec-
red, orange, yellow, green, blue, indigo, and violet, with brown; respectively—the first, second, third,
, etc., segments in each portion of the spinal cord—cervical, dorsal, lumbar, and sacral. The last four
nts in the dorsal region are left white. The advantage of the plate is that it shows very clearly the
pping of the segments that has been demonstrated by Sherrington. The dark heavy black lines
arms indicate the division between the two sides of innervation—that from the upper portion of
rvical enlargement and that from the lower portion. The heavy black lines of the legs indicate the
ns between the sacral and lumbar areas of innervation. The figures and letters indicate the seg-
in which the supply has been drawn and have been introduced for the sake of making the diagram
available for ready reference. C—cervical, D—dorsal, L—lumbar, and S—sacral. The horizontal
of color without dividing lines between them indicate that both segments innervate the areas
ed. (Musser.)

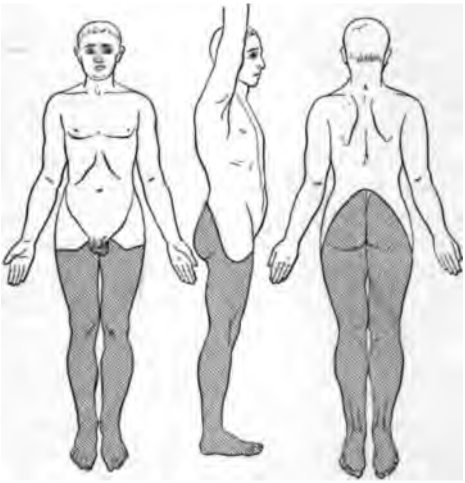
Lesions of the *cauda equina* (where the roots lie closely together) are usually symmetrical. In its upper part, the above symptoms with paralysis of both legs are noted; paraplegia dolorosa is frequent

FIG. 67.



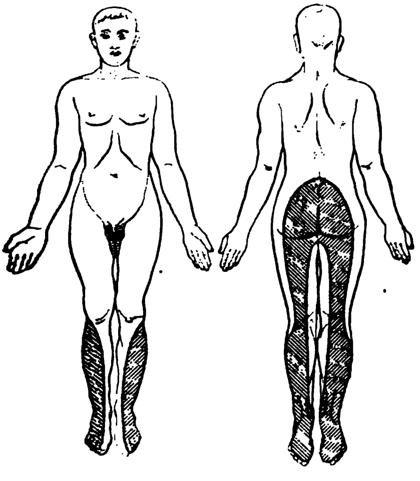
Lesion at the level of the second lumbar segment (Strümpell and Müller, from whom also following figures are borrowed).

FIG. 68.



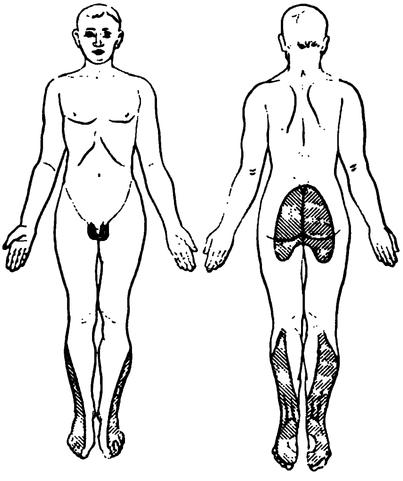
Lesion at third lumbar segment.

FIG. 69.



Lesion at fifth lumbar segment.

FIG. 70

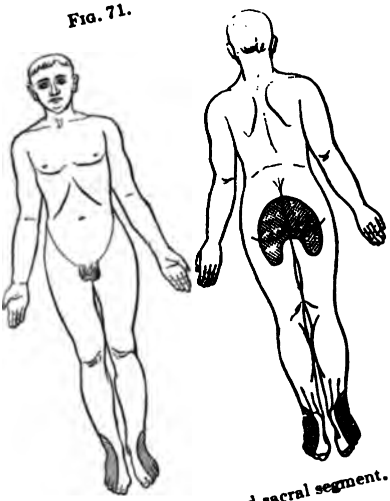


Lesion at first sacral segment.

(See Plate XVI, Fig. B). A lesion below the third lumbar vertebra is usually limited to the pudendal and sciatic distribution. Von Leyden holds that there is no certain distinction between cauda and conus

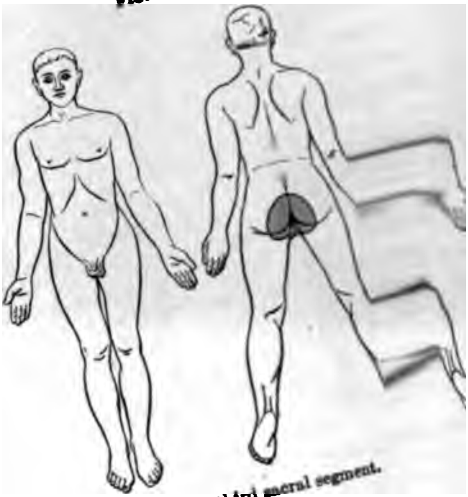
nless external signs exist, as trauma, tumor or caries, or p[illegible]er-
sence of the reaction of degeneration or the occurrence of
y contractions (Strümpell and Schultze). Paraplegia dolor[illegible]osa
in pure lesions of the conus, while it is common in those of [illegible] the

Fig. 71.



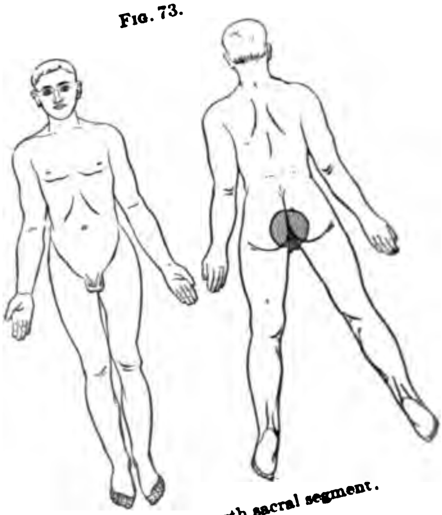
Lesion at second sacral segment.

Fig. 72.



Lesion at third sacral segment.

Fig. 73.



Lesion at fourth sacral segment.

cauda. In conus lesions there may be partial or dissoci
thesia (Schlesinger and Higier) which never develops in
he cauda.

n the gray matter there are *reflex centres* which are enumerated ;alized in the table given above. The reflex arcs comprise the t sensory fiber with its termination in the gray matter and the nucleus with its efferent motor fiber. Collaterals probably t the centres at different levels. Hutchinson first suggested ie fibers of the *oculopupillary reflex* ran by way of the sym- ic nerve (rami communicantes) to the cord. Irritation of its produces dilatation of the pupil (mydriasis), especially in spina

Paralysis from disease of the centre produces myosis, narrow- the eye fissure, sometimes retraction of the eyeball, and less lattening of the cheek; Madame Klumpke-Déjérine found that .ralysis was produced by section of the eighth cervical and first segments; *i. e.*, the localization is the same as for paralysis forearm and anæsthesia of the ulnar surface of the hand and It occurs more often in disease of the nerve roots than in dis- ' the cord itself. The *tendon reflexes* will be considered under lividual cord diseases. They are inhibited from the brain and ›ly also from the cord. Evacuation of the *bladder and rectum* ly a reflex and partly a voluntary process. There are muscular to evacuate the organs and sphincters to retain their contents. ›rmer are stimulated by distention and the latter relax; they ntrolled normally by the will. If inhibition fails, the bladder um is evacuated, sometimes with the knowledge of the patient, lisease of the pyramidal tracts, or at other times unconsciously, ›ma or when the sensory nerves of the parts are diseased. When mbar centre itself is affected, there is continuous evacuation. the rectum, Gowers discovered that disease of the centre dis- itself by relaxation of the sphincter on digital examination; : above the centre, by normal tonicity of the sphincter.

n the above considerations, the leading points are given regard- : *level of the lesion* and the *course of the chief systems*. *Total ›rse disease* of the cord produces paralysis of the muscles at and that level, and sensory interruption. From these symptoms per level of the lesion is determined, but it might be difficult de whether the cord were affected through a narrow horizontal ›r whether all the cord, below it, were diseased, unless the reflexes were noted. Hyperæsthesia usually discloses the level of the

Lesions of the pyramidal tract (first neurone) produce motor sis at the level of the lesion, increased reflexes and spasticity tonic muscular rigidity) but with no muscular atrophy, reaction eneration or sensory disturbance. In lesions of the *posterior ›s*, the chief alteration is sensory disturbance, especially in the : sense, and therefore incoördination or ataxia. The reflex arc e interrupted. Lesions of the *anterior horns or anterior roots* :e a flaccid paralysis followed by atrophy and the reaction of :ration if the process is acute. (If it is chronic, the atrophy is narked than the paralysis, and the reaction of degeneration is or atypical. The trophic cells are not specialized in the horn, but all cells are probably trophic.) The motor nerve (Nasse, 1839) and the muscle degenerate; the reflexes are abolished; the muscle tension (myotatic irritability) is lost, there is vasomotor paralysis (since the fine fibers of the anterior roots are vasomotor), and contractures result (from sensory irritation, Goldscheider). If the lesion is in the anterior horn, there may be fibrillary muscular contractions. Irritation of the anterior horns may increase the reflexes and induce contractures. In lesions *of the posterior horns*, there is disturbance of the temperature and pain sense and some alteration of the tactile sense; the reflexes may be disturbed. (Some maintain that vasomotor symptoms occur). In lesions of the *posterior roots*, the reflexes are disturbed, there is anæsthesia and therefore ataxia. Irritation of these roots causes pain, hyperæsthesia in the form of a girdle sensation in the trunk or lancinating pains in the extremities and reflex contractures. The importance of the spinal ganglion as the trophic centre must be remembered (Waller, 1852), although Vejas and Joseph declare that some fibers pass directly through the ganglion without connecting with it. *Vasomotor fibers* leave the cord through the anterior roots, by the rami communicantes, and course down either in the lateral columns, or in the intermediolateral tract (Gaskell) from the vasomotor centre in the medulla. Most of the vasoconstrictor fibers leave the cord between the third dorsal and second lumbar segments, while the vasodilator fibers are much more diffusely scattered through the cord.

(B). DISEASES OF THE VERTEBRÆ; FRACTURE, DISLOCATION, TUMOR, AND COMPRESSION OF THE CORD.

I. Fracture of the Vertebræ.

Its incidence is rare, for it constitutes but $\frac{1}{3}$ of 1 per cent. of all fractures (Gurlt).

Etiology.—Fracture in the dorsal and lumbar regions occurs mostly (66 per cent.) in the vertebral bodies, while in the cervical region the processes are often (50 per cent.) fractured. Falls, sudden flexion, or blows are the most common causes, though in rare cases it is caused by simple muscular exertion. Previous disease, caries and tabes may favor fracture. Corresponding to the obvious angle in the spine, the upper portion usually slips downward and forward, crushing, compressing or even severing the cord, while the dura is rarely ruptured, although the abundant epidural plexus of veins ruptures and forms a hæmatoma back of the cord. Gurlt found fracture most often in the fifth and sixth cervical, the last dorsal and first lumbar vertebræ. The cord is the seat of hemorrhagic softening or later of inflammation (myelitis), which sometimes develops without cord compression. Marked or permanent injury results in ascending and descending degeneration.

ıptoms.—They may be (*a*) *local i. e., direct.* Dislocation with
ıity (in 68 per cent.) is the most frequent sign; *pain* is frequent;
ıtion (in 38 per cent.) is uncommon save when the processes
ıctured; its elicitation should not be attempted. (*b*) *General*
ms include those of shock and concussion. (*c*) *Spinal symp-*
re more or less complete *paraplegia* (in 71 per cent.), *i. e.*, paral-
both arms, legs, or of parts below the lesion. The reflexes are
sed as a result of the shock; in complete division of the cord,
tellars are totally abolished, even when their lumbar centre is
ıplicated, as in cervical fracture (Bastian, Bruns, Bowlby); the
is not clear; Jackson held that the cause was withdrawal of
rebellar influence; others have suggested that shock or irritation
ausative; the abruptness of the lesion is certainly a factor,
he patellars are present in slow compression (Gerhardt). The
sis may be unilateral (see Brown-Séquard's Paralysis under
MA OF THE CORD). *Sensation* is *less involved*, but radiating pains
ıe extremities may occur when the lesion is at the cervical or
r enlargement; they are sometimes associated with muscular
. Continuous *erection* (priapism) is almost constant, especially
rical fracture, and ejaculation occurs at the time of the lesion.
sis of the bladder and rectum is usual. The bladder can often
ɔtied by suprapubic pressure. Meteorism may result. Decubitus
ores) is common.

cture of the first and second cervical vertebræ usually results in
ı death; most cases result from caries (Bergmann). Some
ts die in a few days, while in a few instances recovery is possible.
ıthor has seen two cases with deformity in the spine and pharynx
ıt any sign of cord compression. There is *pain in the neck*,
n movement, fixation of the head, often palpable deformity
pharynx, dysphagia, dyspnœa, vomiting, and paralysis of the
, palate and of all the limbs, usually with preservation of the
s. The temperature may reach 111°. The pulse is often slowed
or 20) by paralysis of the accelerator fibers of the heart which
ɔm the medulla by way of the lateral columns to the fourth to
dorsal anterior roots and cardiac plexus. Their irritation may
e paroxysmal tachycardia. Fracture of the *third and fourth*
l vertebræ. Involvement of the phrenic nerve results in respira-
ıralysis and early death. In fracture of the *fourth cervical to*
dorsal vertebræ, paraplegia, which increases after the injury
ıemorrhage, is the chief symptom. In the arms it is often some-
ısymmetrical. Atrophy may follow injury to the cervical en-
ıent. Pains, hyperalgesia and tonic or clonic spasms in the
re frequent. Anæsthesia is usually less marked than the paral-
The intercostal muscles being paralyzed, the patient breathes
ıe diaphragm and the accessory respiratory muscles of the neck.
ıotor manifestations may occur in the pupils, face, trunk or
ıities. In fracture of the *third to twelfth dorsal* vertebræ, para-
, anæsthesia and often paralysis of the bowel and bladder

result. The temperature is often low in lesions of the lower cord. *Lumbar fracture,* except in the first lumbar vertebra, is infrequent. Paralysis is not a frequent result, unless there is marked dislocation when paraplegia, especially in the peronei, and atrophy, pains in the legs along the sciatic and femoral nerves, and anæsthesia occur (*v. s.*, LESIONS OF THE CONUS AND CAUDA).

Diagnosis.—The diagnosis is based on the trauma, symptoms shown by the cord, and deformity which, though sometimes absent, serves for differentiation from spinal concussion. In doubtful cases, the examination for tender points, fixation of the spine and radiating pains must be made most carefully, lest the cord injury be thereby increased.

Course.—Healing is difficult, uncertain and incomplete, because callus formation is rarer than cartilaginous and fibrous union, and dislocation may occur after the trauma. Early death frequently results from fractures in the higher vertebræ while in those lower down a later death is likely to occur from cystitis, decubitus or sepsis. The *prognosis* is most favorable in fractures between the third dorsal and second lumbar vertebræ (Gurlt). According to Burrell—from whose collation of 244 cases (1905) most of the above statistics are taken,—the average mortality is 65 per cent.; the mortality is 86 per cent. in cervical, 76 per cent. in upper dorsal, 56 per cent. in lower dorsal and 50 per cent. in lumbar fractures.

Treatment.—Surgical interference is not indicated in fresh cases, unless a comminuted process may press on the cord. If paralysis persists from pressure of a process on the cord, especially in the lumbar region, operation is indicated.

II. Dislocation of the Vertebræ.

Dislocation is traumatic, or spontaneous from previous verteb disease. Traumatic luxation is sudden, injures the cord to a grea extent and results from falls or blows on the head or neck. Fractu very frequently accompanies dislocation. Luxation from previo vertebral disease is gradual, though sometimes sudden, and injur the cord less, since it is often incomplete (subluxation). It occu most often in the cervical region, especially in the first and second, fifth and sixth vertebræ. It is infrequent elsewhere in the spine. T vertebræ sometimes are torn apart without actual displacement.

Symptoms.—The head is often bent forward toward the sternum shoulder, very rarely backward. Movement is suspended or limite Tenderness and pain are elicited and the local deformity may be fe or even seen. The cord is injured by direct compression, contusion, hemorrhage and later by myelitis, with much the same symptoms as in fracture. Sometimes no spinal symptoms are observed. Sudden death is very common in the higher dislocations from pressure on the medulla.

eatment.—Reduction is indicated even when the cord is not
ed, and may relieve all symptoms.

III. Caries of the Vertebræ.

iology and Pathology.—Pott's disease (Pott, 1779), spondylar-
ace, is the most common vertebral disease involving the cord.
es is almost always tuberculous. It is most common after the
h year and in adolescence, but may be seen at any age. Tuber-
sis in other organs is frequent in Pott's disease; it usually begins
ntaneously," in one or several vertebral bodies or more rarely in
rticulations. It may follow trauma. The spongy portion of the
bral body is first involved. Granulation tissue is the earliest
ng; later caseation occurs with extension to contiguous parts,
onnective tissue, ligaments, processes and cartilage. The collapse
e altered body usually produces an angle and prominence in the
e called *Pott's boss*, and the diseased body slips backward, some-
s compressing the cord. By extension the nerve roots may be
lved; they are bathed in pus, sometimes show connective tissue
ration and, in cases of long standing, atrophy. The outer surface
e dura is very frequently inflamed (peripachymeningitis or pachy-
ingitis externa); collections of thick, caseous pus gather in the
lar tissue between the dura and the posterior surface of the body
e vertebra; the cord is more injured by caseous or purulent
ymeningitis than by deformity of the spine. It was once thought
the cord was not only compressed but also inflamed, whence the
e, compression myelitis.

mpression paralysis (so-called compression myelitis) may result
(*a*) vertebral disease, tuberculosis, cancer, or exostoses; (*b*)
se of the spinal cord and its membranes, as tumors, pachy-
ingitis hypertrophica, syringomyelia, or hydromyelia; (*c*) com-
sion by aneurysm, or echinococcus. Pathologists differ as to the
al changes in the compressed cord. Myelitis may develop in
e cases, according to the older pathology (*a*) from compression by
fragments or exudate, or acute extension by contiguity of the
mmation to the cord; (*b*) from slower inflammatory reaction,
wing degeneration of the nervous structures, or (*c*) rarely, from
culous myelitis. "Compression myelitis" is probably nearly
ys softening (myelomalacia). Œdema of the cord results from
anical lymph stasis in the dura (Kahler and Schmaus) and
ends but does not necessarily destroy its functions. Anæmia of
ord (Ziegler) is of less importance. Vessel thrombosis may cause
ning. In some cases with marked paralysis, the postmortem
ination shows no changes in the cord, whence it is certain that
ure may interrupt nerve impulses without structural cord disease.
her cases all gradations of alteration are found, from a few granule
ing cells or swollen axis cylinders to marked disintegration, corpora
lacea, neuroglia overgrowth and induration.

Symptoms.—The symptoms usually develop in the order of grouping given below.

1. **Spinal Column.**—Angular deformity (*kyphosis, gibbus,* Pott's boss) is common but not invariable; it is most marked in upper dorsal disease, and is at times accentuated by periosteal induration. Quite frequently there is only lateral deviation (scoliosis) in which connection it must be remembered that ordinary lateral curvature of the spine practically never affects the cord or its nerve roots. *Local tenderness* over or beside the affected vertebræ and *pain* are usually localized and develop early. The application of heat, which is not felt over the normal vertebræ, may cause pain over the diseased focus (Copeland). Pain and tenderness are very common in the neuroses, but the hyperalgesia is largely cutaneous, is usually diffuse, and varies more than in caries. *Rigidity of the spine* is most conspicuous in disease of the cervical or lumbar regions, where movement is normally most free; it is usually an effort to spare pain. *Gravitation abscesses* are fairly frequent and follow along the planes of least resistance in the tissues. Dysphagia from retropharyngeal abscess or low compression of the œsophagus may cause errors in diagnosis.

2. **Nerve Roots.**—Eccentric pain is usually the earliest symptom; it radiates into the trunk or extremities and indicates involvement by pachymeningitis of the posterior nerve roots. It is much less acute than in cancer and is especially elicited by movement. Sensory are more common than motor root symptoms, and irritative symptoms, as hyperæsthesia, are more common than anæsthesia. Root symptoms are most pronounced in lumbar and sacral disease, and least pronounced in dorsal disease.

3. **Spinal Cord.**—Compression (*a*) interrupts function at its level, disturbs reflexes, impairs rectal and vesical evacuation and (*b*) interrupts upward and downward cordal conduction, producing spastic paresis or paralysis, anæsthesia affecting all varieties of sensation, and increase of skin and tendon reflexes, whereas root disease does not interrupt conduction. Cord symptoms develop slowly; Oppenheim placed the average time at one year; weakness of the legs is followed by paresis, this in time by paraplegia, which, however, may develop suddenly. Anæsthesia and other sensory manifestations are less common than are motor symptoms, because the sensory tracts are more resistant or more protected, since the greatest compression is exerted on the outer lateral surface of the cord. Ataxia is usually obscured by the paralysis. Marked anæsthesia, paralysis and atrophy are usually correctly referred to the cord rather than to the more resistant nerve roots. The reflexes are increased (unless their arc is destroyed at the level of the lesion) since cerebral inhibition is interrupted; Sternberg and Sherrington believe there is also a spinal inhibition.

Special Localization.—1. **Dorsal Caries.**—This is the most frequent type. Gradual deformity is usual, with formation of a sharp or obtuse

gle. Acute deformity, such as might result from a fall or lifting a
avy burden, is much less common. Paraplegia develops in varying
grees of intensity with increased muscle tonus (spasticity), increased
; reflexes, or even clonus, or paralysis of the bladder and rectum;
ometimes develops with contractures but rarely with marked sensory
anges, other than intercostal root pains in the chest wall, which
netimes produce precordial oppression or a girdle sensation.
rpes zoster may develop. The level of the lesion may be marked
a zone of hyperæsthesia or hyperalgesia. Trophic changes in the
n are common, as glossiness, smoothness or dryness. There is no
ential atrophy save from disuse, nor is there any reaction of degen-
tion, unless the anterior horns are diseased. In cases of partial
alysis, the tendon of the tibialis anticus becomes prominent (tibialis
enomenon). The tendon reflexes may be increased, even when the
alysis is apparently flaccid. They are almost always increased
ow the lesion. Exaggeration of the patellars is an early symptom,
en antedating the paralysis. The skin reflexes below the lesion are
en increased, but may be decreased in cases of severe compression,
ugh they are seldom absent. The Babinsky reflex (see BRAIN
MORRHAGE) is often found.

2. **Cervical and Upper Dorsal Caries.**—This form ranks next in
quency; radiating pains reach the shoulder, arm and hand, and
y be felt in or about the joints on one or both sides. Anæsthesia
nore common, along the ulnar nerve, over the sternum and scapula,
cause the posterior roots are more often affected. Paralysis may
ult from compression of the cord, or from disease of the anterior
ts, and involves the small muscles of the hand, forearm and triceps.
e fore-finger and thumb often cannot be apposed (the ape hand),
d from atrophy of the interossei, the *main en griffe* (*v. i.*) results.
e forearms are flexed and pronated, while the arms are adducted
hoburn). The tendon reflexes usually persist. In other cases,
rked atrophy occurs instead of paralysis and the reaction of degen-
ation sometimes develops. Spastic para-paresis or -plegia in the
gs from compression of the pyramidal tracts usually accompanies
e diplegia (paraplegia) brachialis, which, however, sometimes occurs
one, when the anterior horns or roots are diseased. Myosis is com-
on from injury to the oculospinal centre (Rollet, 1864), and there
ay be narrowing of the eye fissure and less often retraction of the
eball, which is more frequently unilateral than bilateral, or differs
degree on the two sides. Flattening and reddening of the cheek
e less common. These are less symptoms of disease of the cord
an of the anterior roots. The reflexes of the legs are increased;
tal compression of the cord may destroy the tendon reflexes; this
caused by secondary changes in the lumbar cord; when the paralysis
flaccid, the urine is retained at first, and later there is incontinence.
here is usually constipation.

3. **Disease of the First Two Cervical Vertebræ.**—(Rust's Disease).—
ı disease of the atlanto-occipital articulation, nodding is impossible,

while the involvement of the odontoid-atlas articulation prevents turning of the head. In body-movements the patient supports the head with the hands (Rust). The head may be held as it is in torticollis, and induration under the occiput is common. Pains radiate to the eye, ear, and occiput. The typical paralysis in disease of the two upper cervical vertebræ is brachial paraplegia, which usually occurs without atrophy. Spasms in the face, anæsthesia in the neck and upper chest, aphonia and other symptoms indicating involvement of the medulla oblongata may be present. Charcot observed a slow pulse with attacks of syncope. The spinal accessory supply to the head and palate and the hypoglossus supply may be affected. The course is usually more rapid (one to two years) than in carcinoma; recovery is possible (v. Bergmann). It has long been held that degenerated axis cylinders do not regenerate, but Fickler made the interesting observation that the fibers of the severed lateral pyramidal tract grew around the old scar to reach the anterior horns below it. Gravitation abscess may reach the neck or pharynx. Sudden death is not unusual, because dislocation readily occurs, since the disease is in the articulations rather than in the vertebral bodies.

4. Lower Dorsal and Upper Lumbar Caries.—This type produces atrophic flaccid paraplegia, destruction of the lumbar reflexes, urinary incontinence, anæsthesia of the legs, pain in the abdomen or possibly in the groins or anterior thighs. When it occurs in the lower *lumbar region* there is flaccid atrophic paralysis, absence of the patellars, and spastic paralysis of the legs, with clonus of the Achilles tendon (see CONUS AND CAUDA LOCALIZATION).

Diagnosis.—Cases may be classed under three groups (*a*) those with spine symptoms, (*b*) those with spine and spinal cord symptoms, and (*c*) those with cord symptoms only. Cases are almost unmistakable in which the paralysis follows signs of bone disease in early life. Repeated examinations usually determine the nature of doubtful cases in which bone-symptoms develop after compression of the cord or nerve roots, and in which there are early increase of the skin or tendon reflexes, root-symptoms, which are usually bilateral, as cervical neuralgia, lumbago (Janeway), anæsthesia, and muscular atrophy or paralysis. The Roentgen rays may aid in diagnosis. Gravitation abscesses, fever, and tuberculosis elsewhere are significant. In persons of advanced years, cancer, tumor within the spinal canal and aneurysm must be considered (see DIAGNOSIS OF CANCER OF THE SPINE).

Prognosis.—The prognosis is unfavorable, since about 50 per cent. of cases die. Relative recovery with deformity is possible, especially in children, but even then the process may break out anew. A rapidly fatal course is rare. Absolute paralysis may disappear after months or even years. The prognosis is best in diseases of the lower dorsal region and is least favorable in disease of the atlas and axis. Gowers remarks that recovery or improvement is more frequent than in any other disease with symptoms of equal severity. There is hope of recovery in most cases, and yet there are few in which it is sure. Cystitis ion, and leads to pyelonephritis; bed-sores are frequent, and esults from sepsis, mixed infection, amyloid disease or lung osis.

ment.—(*a*) *Medical* treatment includes administration of iron, and cod-liver oil; fresh air and a full diet are equally impor-he skin and bladder should receive special attention. Elec-contra-indicated. (*b*) *Orthopedic treatment* produces the most ul results. Rest in bed on the back should be enforced for Extension, fixation by braces of the neck and back, suspen-d for lighter cases or those involving the lower spine, the ast, are described in works on orthopedic surgery. (*c*) *Sur-tervention* is indicated only when other treatment has been orough trial. The results are not encouraging and disease of cted bodies is difficult of access. The dura should never be According to Chipault, the *indications* are (i) disease restricted rches, (ii) cold abscess, (iii) flaccid paralysis, since Babinsky hat this occurs in moderately severe dorsal disease, and (iv) of the bone focus and replacing of granulation tissue by con-tissue, as in the cases first operated on (Macewen, 1888). The *idications* are (i) multiple vertebral disease, (ii) lung tubercu-d (iii) disease of the cord itself. Victor Horsley advises ortho-eatment for two months in children, and then, in case of laminectomy. In adolescents and in those of advanced years, ders that laminectomy is indicated practically at once.

IV. Carcinoma of the Spine.

noma is almost always secondary to carcinoma of the mamma often of the digestive tract, uterus, prostate or thyroid; it y involves the bodies, usually of several vertebræ.

toms.—Angular deformity is less common than in caries. A nay be felt. Bruns considers a sinking in of the vertebræ or g of the spinal column as rather characteristic. Rigidity is but is less conspicuous than in caries. Tenderness is common. *nal symptoms* closely resemble those of compression by caries, present in 60 per cent. of cases (Schlesinger). Compression posterior roots is most common and produces neuralgic hich is at first paroxysmal and later constant, lancinating, ercible; it is located along the spinal column, while in caries elt over the spine or in the side of the chest (Gull). Closely ed are hyperalgesia and anæsthesia, tenderness on pressure, reflex muscular spasm and herpes zoster. Compression of the ess common and occurs late; if paralysis develops it is usually nd painful. The veins of the lower extremities thrombose ly and fatal pulmonary embolism may result (Schlesinger).

osis.—Diagnosis depends upon (*a*) recognition of a primary which is sometimes latent or overshadowed by the secondary deposit); (*b*) local signs (tumor, spinal irregularity, arcuate kyphosis); (*c*) cachexia; (*d*) advanced age, for caries commonly occurs in young individuals; (*e*) pain, which is usually bilateral and is increased by movement; paraplegia dolorosa (Cruveilheir) is much more common and incomparably more intense in cancer than in caries, from infiltration of the sensory roots; anæsthesia with pain (anæsthesia dolorosa) is also common; and (*f*) rapid onset and course. *Sarcoma* may be primary or secondary. Cachexia is less common; it is more often seen in youth than in elderly persons, and is more frequently multiple. *Vertebral syphilis* is very uncommon, as gummata in the bone or periosteum, but responds to antisyphilitic treatment. Syphilis simulates caries rather than carcinoma. Actinomycosis rarely causes compression of the cord; it is recognized by its presence in other organs and by the sulphur bodies in the sinuses and aspirated fluid. *Arthritis deformans* (*q. v.*) is recognized by exostoses, tenderness, ankylosis, in rare cases by crepitus, by alterations in other joints and by root-symptoms caused by narrowing of the foramina (radiating pains, vasomotor symptoms and atrophic paralysis). *Aneurysmal erosion* of the spine, cervical pachymeningitis, hydatids, exostoses, tumors within the spinal canal, glands compressing the intercostal nerves and tabetic pains in the trunk must be considered. The *prognosis* is absolutely unfavorable, and the *treatment* is directed chiefly against the pain.

(C). DISEASES OF THE SPINAL MENINGES.

Few diseases of the vertebræ and cord do not to some degree affect the meninges but, with few exceptions, the meningeal symptoms have little independent clinical interest.

I. Hyperæmia.

At autopsy the blood gravitates to the dependent parts, and therefore congests or discolors the meninges. At the present day but little importance is attached to the symptoms referred by Frank, Oliver and Brown-Séquard to meningeal congestion. Stasis, congestive diseases as tetanus, eclampsia, and chorea, or asphyxiating poisons, as carbon monoxide, or strychnine, doubtless congest the membranes.

II. Hemorrhage.

(*a*) Between the vertebræ and dura, hemorrhage is frequent from rupture of the subdural plexus of veins. It surrounds the nerve roots and is most severe over the posterior cord; it results mostly from trauma and to a lesser extent from convulsive affections, as tetanus, or strychnine poisoning, vertebral disease, stasis or aneurysmal rupture.

(*b*) In the arachnoid sac hemorrhage (hæmatorrhachis) is equally frequent and results from the above named causes, and from asphyxia, extraction by the foot during labor, hemorrhagic diseases, or virulent leptomeningitis. Severe hemorrhage may follow brain hemorrhage, especially at the base (from rupture of aneurysm, or apoplexy) and the entire spinal canal may be filled with blood. (*c*) In the pia, hemorrhage rarely attains either pathological or clinical interest.

Symptoms.—Small hemorrhages cause no symptoms, and meningeal apoplexy may easily be overshadowed by brain or traumatic cord symptoms. Pain, localized in the small of the back or sometimes generalized, rigidity, radiating pains from compression of the posterior roots, hyperæsthesia or hyperalgesia, painful reflex muscular spasms, motor weakness (and paraplegia), anæsthesia, sphincter disturbance, and abolition of the tendon reflexes are the common symptoms. They develop suddenly, affect the lower more than the upper cord, increase for a day or so, produce inflammatory reaction for two or three days and usually subside in from four to eight weeks. Physical signs vary with the location; for instance, brachial paraplegia is present in cervical hemorrhage, and involvement of the legs in lumbar or dorsal hemorrhage. There are no cerebral symptoms, except those resulting from the initial shock in traumatic cases.

In making a *diagnosis* the most important symptoms are those of irritation, which is sometimes followed by paralysis, while in hemorrhage of the cord (hæmatomyelia) paralysis prevails and pain is less frequent. In non-traumatic cases, exact diagnosis is impossible. The *treatment* is symptomatic. Absolute rest in the lateral decubitus or on the face should be enjoined.

III. Pachymeningitis.

(*a*) This may be *external* (peripachymeningitis), as described under caries. It may follow penetrating bed-sores, deep cellulitis, psoas or retropharyngeal abscess. Its symptoms are those of compression by vertebral caries (*q.v.*). Root symptoms and fever are frequent. The prognosis is unfavorable, and the treatment is that of the cause, or is expectant or surgical. (*b*) *Internal pachymeningitis hemorrhagica* is a second form. Its causes are those of the cerebral form, as paretic dementia, alcoholism, trauma and sometimes tuberculosis. (*c*) Charcot (1871) and later Joffroy described a form involving chiefly the cervical region (*pachymeningitis cervicalis hypertrophica*), in which the inner surface of the dura is thickened by annular concentric fibrous deposits. The dura adheres to the vertebræ and leptomeninges and compresses the nerve roots, which indurate. Several cervical segments are involved. The process is most marked on the dorsal aspect of the cord, which is flattened anteroposteriorly and shows peripheral induration by extension of the process to it through the pial vessels (meningomyelitis); this causes the frequent ascending and descending degeneration. Cold, syphilis, alcoholism and trauma are probable causes.

Symptoms.—Charcot described *three stages;* (*a*) the *neuralgic stage,* which lasts two to three months, and corresponds to involvement of the posterior nerve roots, in which pains radiate from the nape of the neck or occiput into the ulnar and median nerve supply. They are continuous, with paroxysmal exacerbations, with localization at times in the arm joints, with rigidity as in caries, paræsthesia in the arms, some motor weakness and sometimes vesicular eruptions. Hyperæsthesia is common. (*b*) The second stage is that of *atrophic paralysis* of the arms in which the process involves the anterior nerve roots. The pains are replaced by atrophy and paralysis in the ulnar and median nerve areas, while the radial area is usually unaffected; this produces flexor paralysis in the forearms and hands, and the claw hand (*main en griffe*) results from contracture; the wrist is overextended, the first phalanges are extended, the second and third are flexed; ulnar and median anæsthesia and the reaction of degeneration are found. The shoulder and elbow muscles may be involved. Muscular atrophy is not constant and other deviations from Charcot's complex are at times noted, as absence of contracture or extension to the lower cord. (*d*) In the *third stage,* the legs show a *spastic paralysis* without atrophy, and the bladder and rectal functions and sometimes the sensory conduction are disturbed from secondary changes in the cord.

Diagnosis.—The process is usually localized with ease as a disease in the cervical region. The marked pain is characteristic but may also occur in tumors, cervical spondylitis, syphilitic meningomyelitis and at times in syringomyelia. Some cases result from syphilis. Von Leyden and others maintain that diagnosis is usually impossible and that the process is not a unit but only a cervical localization of a meningomyelitis. Amyotrophic lateral sclerosis (*q. v.*) and progressive spinal muscular atrophy have no such severe pains, and syringomyelia (*q. v.*) is characterized by dissociated anæsthesia as well as by muscular atrophy, painless felons and trophic changes in the joints. Tumors (*q. v.*) are difficult to differentiate. Neuritis causes symptoms which are usually limited entirely to the limbs, and over whose nerve trunks there is often tenderness.

The **prognosis** is usually unfavorable, and chronic progression for two or more years, cystitis, etc., usually develop. Instances of recovery are recorded.

Treatment.—Warm baths, cautery for counterirritation, sedatives, narcotics, mercury, iodides, and care of the skin and bladder are indicated.

IV. Spinal Leptomeningitis.

This closely corresponds to the description given under cerebral meningitis, as to the spinal, pathological and clinical findings. It ay occur (*a*) in the acute form, from trauma, regional extension from euritis, lung cavities, bed-sores or other adjacent infections, from neral infections, and from epidemic, suppurative or tuberculous eningitis; or it may occur (*b*) in the chronic form, which has little dependent clinical interest, which results from tabes, trauma, myelitis, philis (*q. v.*) or alcoholism. The symptoms are pain in the back, gidity, hyperæsthesia, and radiating pains; the course is afebrile.

V. Syphilitic Affections. (See Syphilis.)

(D). TUMORS OF THE SPINAL CORD AND ITS MEMBRANES.

These are rare; about 200 cases are reported. Brain tumors are x times as frequent (Schlesinger).

I. Tumors of the Membranes.

These may arise from the dura or leptomeninges. In a collection of) *extradural tumors*, Horsley found five sarcomata, four lipomata, ur tubercles, three echinococcus cysts, and one each of myxoma, brochondroma, carcinoma, and fibrosarcoma. Cysticercus often uses no symptoms. These growths are more likely to cause erosion the vertebræ than compression of the cord. *Intradural tumors* are early twice as frequent. Horsley collected 38, of which there were velve myxomata, seven sarcomata (endotheliomata), seven fibromata, ur psammomata, four tubercles, two parasitic, and two syphilitic mors. Lipomata, echinococci, cysticerci, neuromata and angiomata re rarer. Though they are usually single, multiple occurrence may e noted in sarcomata, parasitic tumors and neuromata. This group ompresses the cord or its nerve roots. The cord is altered as in caries; here is œdema, flattening, discoloration, peripheral hardening and entral softening, and secondary ascending and descending degenerations. The membranes thicken and the nerve roots flatten and atrophy.

Symptoms.—Extradural growths produce more vertebral than cord ymptoms. The first symptoms are usually those of compression of he *nerve roots*, *e. g.*, rigidity; hyperæsthesia; radiating pains, first on ne side, then on both, which run into the arms, trunk or legs, according their localization, and advance from one root to another; paræsesia; anæsthesia which is less common since it implies involvement at least three roots (Sherrington's law); atrophic paralysis with somotor symptoms; and spontaneous muscular contractions. Local derness is rare. Early symptoms sometimes date from trauma. e symptoms recall those of carcinoma of the spine aside from its rtebral signs. Later, evidences of *compression* of the cord appear, paresis perhaps of one side first (Brown-Séquard's type, *v. i.*) or raparesis with atrophy if the anterior horn is involved at that level, with increased reflexes (unless the arc is broken or total compression results), with spasticity and contractures; and with sensory changes. In this type, as in carcinoma of the vertebræ, the paraplegia dolorosa of Cruveilhier is common, as is anæsthesia dolorosa (the "eccentric projection" or reference of pain to the anæsthetic areas). Finally, there is total paraplegia, anæsthesia, bladder and rectal paralysis and decubitus.

II. Tumors in the Cord Substance.

These are six times less common than tumors in the membranes; they include *glioma* (which is most frequent in the cervical and upper dorsal regions), sometimes gumma, sarcoma (in the pia), tubercle (in the gray substance or posterior horn) and cysticercus. Most of them are circumscribed, though glioma tends to vertical diffusion. Unlike meningeal tumors, they are rarely metastatic.

Symptoms.—In intramedullary growths, root symptoms are absent, especially in gliomata and, since there is gradual compression of the cord, the clinical picture may resemble chronic myelitis and syringomyelia. The cord symptoms are disturbed conduction; spastic paraplegia; marked increase of reflexes (unless the arc is broken or absolute cord compression develops); bilateral symptoms, or if unilateral, they soon reach the other side; extensive muscular atrophy, if the cervical or lumbar enlargement is diseased, often then with the reaction of degeneration; and sensory changes involving all varieties of sensation or certain varieties only, as pain or temperature (dissociated or partial anæsthesia). Some writers, as Bruns, maintain that the lesion and symptoms are at first unilateral. Then the Brown-Séquard complex is found; on the side of the tumor there is paralysis with increase of the reflexes, loss of muscle sense, a zone of anæsthesia just below and one of hyperæsthesia just at the level of the lesion, and on the other side there is total anæsthesia save that the muscle sense is preserved. As the tumor grows, total paraplegia and anæsthesia may, in rare cases, develop below the level of the lesion aided perhaps, by softening, œdema, or actual myelitis.

Localization.—Accurate determination of the localization is most important but this can rarely be done until the cord itself is compressed. The most common error is in locating the tumor too low. Atrophic paralysis, in the cervical or lumbar regions, is the most certain sign, and sensory changes are less reliable. The actual lesion is one or two (three or four) segments higher than the anæsthesia (Sherrington's law) because anæsthesia results only from involvement of at least three roots. The hyperæsthesia usually represents the level of the lesion, and local spinal tenderness, though rare, is very important. Finally, the spinal segment involved must be considered in terms of the corresponding vertebræ (*v.s.*, page 1061 Gower's cut). (*a*) When the tumor is in the *upper cervical* cord, there are pains in the cervical plexus and perhaps anæsthesia; at first after the Brown-Séquard syndrome, there ay be a spinal hemiplegia differing from cerebral hemiplegia in that e face and hypoglossus escape and the anæsthesia is contralateral the paralysis; then there is spastic paralysis of all four extremities, hich rarely lasts long, since affection of the phrenic nerve causes rly death. (*b*) In tumor of the *cervical enlargement* there is atrophy, accid paralysis and pain in one arm, spastic paresis in the leg of the me side, anæsthesia of the entire opposite half of the body and pposite limbs; and then paralysis of both arms and legs with anæs- esia. (*c*) Localization in the *dorsal cord* often produces unilateral gns, as paresis of one limb with its half of the trunk, and anæsthesia the corresponding parts of the other side; spastic paraplegia of gs and abdomen, retention of urine and later incontinence; and en girdle pains. (*d*) In tumors of the *lumbar enlargement* there are nilateral pains in the lumbar plexus and atrophic paralysis in the eopsoas, quadriceps, and adductors; there is total anæsthesia when e cord is involved in the region of the lumbar plexus; later partial æsthesia (affecting the sense of temperature and pain) develops in e region of the sacral plexus; still later there is complete paralysis d anæsthesia in the region of the lumbar plexus, with atrophy, sence of the patellar reflexes, with the reaction of degeneration, while e legs and feet are spastic, and with clonus of the Achilles tendon. (*e*) the localization is in the *sacral cord*, there is atrophic paralysis of e leg and foot, of the posterior muscles of the thigh, of the gluteal d perineal muscles, anæsthesia, total paralysis of the bladder and ctum, impotence, loss of the Achilles tendon reflex (while the patellars ay remain), early decubitus and cystitis. (*f*) If the tumor is in the *uda*, the symptoms are usually bilateral from the beginning; the rown-Séquard complex cannot occur; paraplegia is rarely as symetrical as it is in cord lesions; typical reaction of degeneration is dicative of caudal disease; pain is violent, since many sensory fibers re in close contact; Minor described bilateral and even unilateral ciatica as an early symptom. The sacral plexus is chiefly or first nvolved, while growths of the lumbar enlargement affect both the acral and lumbar plexus.

Diagnosis.—The diagnosis of meningeal tumor, which is always ost difficult, is based on (*a*) local pain and rigidity, which is especially arked in the extradural type; (*b*) hyperalgesia; (*c*) radiating pains, e., root symptoms which later, by gradual transition, give way to d) cord symptoms, which are usually or often unilateral at first and en bilateral, as interrupted sensory and motor conduction, and crease of the reflexes. *Carcinoma* of the vertebræ produces more in on movement than other tumors. In *caries* there is less pain, al vertebral changes eventually appear, and the course is usually ore rapid. In *myelitis* the focus is less commonly near the cervical largement, and therefore the arms are less often paralyzed. In yelitis, radiating pains may occur, but are much rarer than in tumor, d a girdle sensation is more common. The local pain sometimes ggests *aneurysm*, lung disease or peripheral neuralgias, the tender points of which are always absent. Other symptoms in the sequence above given must be present for diagnosis. A quiet course, root symptoms, early interruption of cordal conduction and absence of trophic disturbance in the bones and of dissociated anæsthesia argue for tumor and against *syringomyelia.* A diagnosis of *tuberculosis* or syphilis is fortified by their presence in other organs. Age is of diagnostic importance: in youth, tubercle; at puberty, sarcoma and glioma; in the third and fourth decades, glioma, tubercle or sarcoma; and in advanced years gummata are most frequent. *Diffuse sarcomatosis* is usually cerebrospinal, rarely invades the cord substance as do gummata and tubercles, affects the posterior cordal surface most frequently (the lateral surface little, and the anterior very rarely) and generally in the lumbar and lower dorsal regions; it occurs largely in the young and its course is rapid. Abscess of the cord is most rare. *Tabes* in one remarkable case was simulated by multiple tumors of the posterior roots. *Tumors of the cord* are more difficult of differentiation, for root symptoms are often lacking.

Course and Prognosis.—Slow development, progression which is either steady or "by starts," sometimes remissions, and eventual compression of the roots or cord are usual, with death from cystitis, or decubitus. Sudden paralysis is less common. The average duration is one to three years, but a longer course is possible (even ten to fifteen years).

Treatment.—1. **Therapeutics.**—Narcotics must be employed to relieve pain, but should be used sparingly in the early stages, because if given freely, their effect may fail later if the course is protracted. Syphilis is always a possible factor; absence of evidence or history of the disease counts for little, and the patient should receive the benefit of antisyphilitic treatment.

2. **Surgical Treatment.**—Gowers in 1887 localized a case of fibromyxoma which Horsley removed successfully; this was the first case on record. Bruns collated twenty cases of operation; 33 per cent. of them recovered or improved. Putnam and Warren collected 33 operated cases, with over 50 per cent. recovery. Tumors of the cord are less favorable than those of the meninges. Intramedullary tumors may only crowd the nerve paths, but their removal is often attended by danger of laceration or acute myelitis, which may possibly result in damage equal to that of the tumor. Accurate localization is most important, because, in one case, after removing parts of several vertebræ in vain, further search for the suspected growth had to be abandoned. Therefore, Urban's osteoplastic resection is valuable; it consists of folding back the cut muscles and bone that they may be replaced and the strength of the spinal column may not be impaired. Death may result from shock, escape of cerebrospinal fluid (Chipault) and less frequently, with modern methods, from infection. Starr records 16 complete recoveries. Harte in 1905, collected 92 operations; the operative mortality was 47 per cent.; of the balance, 59 per cent. were cured, 34 per cent. improved and 7 per cent. not improved.

RCULATORY DISTURBANCES; HEMORRHAGE; TRAUMA OF THE CORD.

I. Hyperæmia of the Spinal Cord.

the spinal meninges, postmortem hypostatic congestion is com-
pecially in the dorsal aspect of the cord. Sometimes the mem-
are hyperæmic and the cord anæmic. The white matter is
pale. The cord may be passively congested, as in cardiac
iency. Vascular variability was formerly considered causal of
rritation which, however, is functional (hysterical and neuras-
No definite clinical picture can be constructed.

II. Anæmia of the Spinal Cord.

lyses referred to anæmia of the cord are without doubt mostly
eurosis, neuritis, hemorrhages in the cord or nerve sheaths, and,
icious anæmia, to degeneration of the dorsal columns. Paralysis
ng profuse hemorrhage from the stomach or uterus may be pro-
eferred to cord anæmia. Stenson (1667) demonstrated experi-
ly that compression of the aorta produced paraplegia due to
of the cord, the motor cells of which appear to be especially
ible to ischæmia. The symptoms are referred by others to
of the peripheral nerves. Ligature or compression of the
fter ligature of both subclavian arteries has identical effects
aul and Tenner).

nbolism and Thrombosis (Myelomalacia) of the Spinal Cord.

olism of the spinal arteries is extremely rare, but has been
d in endocarditis (v. Leyden, Weiss.) No separate clinical
ms can be recognized, although experimentally Money observed
orm movements. Thrombosis is very rare. Venous throm-
rare, and is secondary to other lesions of the cord. Arterial
osis is rather more common. It has been found in syphilis,
e sclerosis, senility and perhaps bears some relation to acute
es in the cord, as disseminated or focal myelitis. It is often
ible to decide whether an area of softening is ischæmic (myel-
ia) or inflammatory (myelitis).

IV. Hemorrhage in the Cord (Hæmatomyelia).

logy and Pathology.—Most cases of this rare lesion occur in
etween 20 and 40 years of age. (*a*) Capillary hemorrhages
ause symptoms; they occur about larger extravasations of blood

in foci of softening, in inflammation, stasis, intoxications, infections or blood diseases. (*b*) Hemorrhagic infiltration occurs diffusely, and chiefly in areas of softening or near larger foci of hemorrhage. (*c*) The larger hemorrhagic focus may result from trauma, muscular effort, caisson-disease, etc; it compresses and destroys the cord substance; it rarely exceeds the size of a hazelnut (round or oval form) but may extend through the entire length of the cord (tubal form of Levier), following exactly the less resistant gray matter, while the firmer white columns (pyramidal tracts) usually resist its lateral diffusion; if it invades the white matter, hemorrhage usually occurs in the posterior columns; it occurs mostly in the cervical cord; all large non-traumatic hemorrhages occur in the gray matter. Effused blood undergoes the same fate as in other parts; it turns brown, then yellow-red, and ultimately deposits dark pigment and hæmatoidin crystals. The nervous tissue is disorganized by larger hemorrhages, which form in it a cavity. Eventually a pigmented apoplectic scar or cyst results.

Symptoms.—The symptoms vary with the different forms. (*a*) In the accessory form there is bleeding into a tumor, cavity, softening or inflammation (Charcot, Hayem, Köster); punctate hemorrhage may accompany other affections, as hemorrhagic purpura, stasis or convulsive disorders, and few or no symptoms develop. (*b*) The traumatic (see TRAUMA) form is less important in spinal fracture than concussion; it may develop during dystocia or fœtal extraction. Trauma causes 90 per cent. of cases of hæmatomyelia (Oppenheim).—

3. The *spontaneous* form is rarer than it is in the brain, because the cord is firmer, subject to lower blood pressure, and most rarely the seat of miliary aneurysm; it results from sudden exertion in man (and more often in the horse), from repeated coitus, suppressed menstruation or rarely from arteriosclerosis.

The onset is sudden, without prodromes or loss of consciousness. Previous symptoms may suggest a hemorrhage into an inflammatory focus. Local symptoms must vary with the structures involved, and their level; they embrace paraplegia, monoplegia, often the Brown-Séquard syndrome, paralysis with or without atrophy or spasticity, anæsthesia either total or partial (*i. e.*, dissociate, as in syringomyelia, Minor), as might be anticipated from its location in the gray substance and posterior horns; pain at the level of the lesion, perhaps from involvement of the posterior horn; less often eccentric pain, girdle sensation and muscular rigidity. The reflexes and sphincters vary with the level. In general the reflexes are increased in small and abolished in large hemorrhages. The symptoms advance rapidly and may suffer an accession from secondary reaction or myelitis. Early death is rare. Late death from cystitis or decubitus is more common. Recovery is rare, compared with recovery in cerebral apoplexy; it occurs most often in the punctate type; it is rarely absolute and some disturbance in motility or sensation usually remains.

Diagnosis.—The diagnosis is usually only probable; it is based on the sudden onset of paresis and anæsthesia and their rapid advance

ntiation from meningeal hemorrhage (*hæmatorrhachis*), see
; in the latter, meningeal irritation is more common, as
d radiating pains, rigid spine and muscles, hyperæsthesia of
d muscles; paralysis is absent or incomplete; and recovery
mplete. From *acute myelitis*, differentiation is often uncer-
equently of only academic interest, since each affection may
the other and the prognosis and treatment of each are
n myelitis and anterior poliomyelitis elevation of temperature
ive.

V. Caisson or Divers' Paralysis.

ng to Pol and Watelle (1854), this occurs in workers who are
pressure of at least 2, and usually 3 or 4 atmospheres.
appear when the individual comes suddenly into the open
y vary, and include transient pain in the ears, sometimes
nd vertigo, headache and pains in the limbs, joints and epi-
in 15 to 50 per cent. there is paraparesis or paraplegia,
with anæsthesia, and usually with urinary retention; much
there is hemiplegia or involvement of both arms; epistaxis,
mptoms; loss of consciousness, and symptoms of brain-
s vomiting or slowed pulse. Men of heavy build, alcoholics,
hands" seem to be predisposed; hard exercise after coming
r produces unhappy symptoms, which are not severe or are
he interval of work does not exceed one hour.

gy and Pathogenesis.—The pathology and pathogenesis
rmined. Hoppe-Seyler (1885) and Bert found that lessened
ric pressure liberated oxygen, nitrogen or carbon dioxide in
and tissues, which ruptured the vessels and tissues and
punctate hemorrhage. In the first autopsy made, v. Leyden
ts in the cord substance, particularly in the posterior and
lumns, surrounded by round cells (reactive disseminated
most changes were in the dorsal cord, the tissues of which
ompact; secondary degeneration is found (von Rensselaer)
ges in the gray substance (Schultze) which are thought to
m hemorrhage; Catsaris actually saw gas bubbles in the
nder moderate pressure the gas is given off to the lungs,
treme instances it is given off to the tissues. The symptoms
y result less from the gas bubbles (air embolism) accumulat-
blood under high pressure, than from its sudden reduction
eactive apoplective myelitis. Andrew H. Smith holds that
is cordal congestion and stasis.

sis.—A few subjects die at once; others die from cystitis, as
s; about 50 per cent. recovers completely and 50 per cent.
with spastic paresis. The mortality ranges from 2.7 to 15.8

Treatment.—The return to normal atmospheric pressure should be made gradually; recompression is successfully employed. Smith recommends ergot.

VI. Trauma of the Cord.

Compression by caries, tumor and carcinoma have been considered; other surgical lesions can only be touched on here. *Crushing* of the cord may be associated with hemorrhage, myelitis and secondary degenerations. *Total division* of the cord may result from fracture, gun shot or stab wounds; it is extremely rare; early death is usual; its symptoms are absolute paraplegia, anæsthesia, bladder paralysis, and abolition of the patellar reflexes. A recently reported case of total section with operation and recovery was received with some skepticism. *Gun shot* wounds usually result in fracture, crushing and sometimes actual division; as a rule the prognosis is unfavorable. *Stab* wounds may produce bilateral symptoms, or unilateral signs, as the Brown-Séquard paralysis; 20 per cent. recover; 60 per cent. improve and 20 per cent. of cases die (Wagner and Stolper). *Restitution of function* in lesser injuries results not from regeneration but from compensation by other paths of conduction. Laminectomy is indicated only in crushing injury of the lower half of the spine, because cervical cases almost never recover (Hahn).

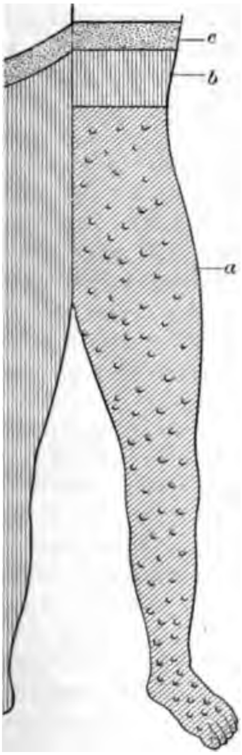
VII. Brown-Séquard's Paralysis.

This was described by Séquard and Türck simultaneously in 1850. According to Brown-Séquard's original proposition, *on the side of section* (cervical cord) there occurs (*a*) paralysis of voluntary motion, muscle sensibility and vasomotor tonus; (*b*) hyperæsthesia of trunk and limbs, to touch, pain, heat and cold; (*c*) vasomotor paralysis of face and eyes (higher temperature, narrow pupils, and moderate contracture of certain facial muscles). *On the contralateral* side there is anæsthesia of all varieties of sensation, except muscle sensibility.

Symptoms.—Exact hemisection of the cervical cord produces a *spinal hemiplegia* on the side of the section, but in clinical injuries and experimental section, the trauma is rarely wholly complete or strictly unilateral. (In some cases decussation of the uncrossed pyramidal tracts may occur in the cord lower down than the usual crossing point in the medulla [Flechsig] and spinal hemiplegia with contralateral monoplegia may result from unilateral lesion. Déjérine and Thomas speak of a third "homolateral pyramidal" tract, which may cause restitution of motor function. Edinger mentions a "reserve" innervation by which decussation of motor fibers occurs lower than the pyramidal decussation in the medulla). The most characteristic clinical type is the mid-dorsal lesion, but in cervical section or in

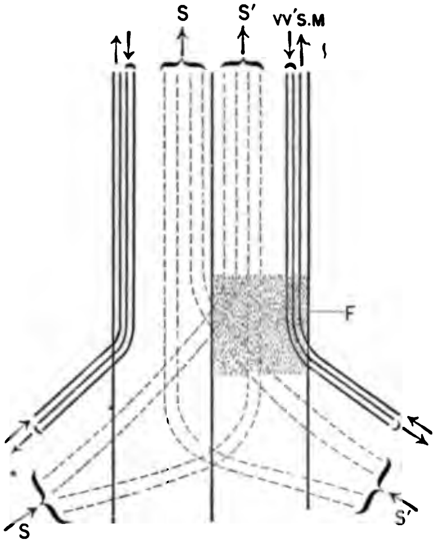
;ical foci, paresis of the arm with paralysis of the leg is more
ecause the cervical motor tracts are less compact than they
ıe lower cord; *i. e.*, paralysis is more frequently limited to a
f muscles than to an entire upper extremity. "Conversely,
ı of the leg may be incomplete, while that of the arm is com-
ring to the escape of fibers for the leg which cross lower down
ırd" (Gowers). The paralysis often decreases with surprising
, and if the anterior cells are intact it gradually becomes simple

FIG. 74.



ıard's paralysis from a left-
ı A, vaso-motor and motor
B and D, cutaneous anæs-
hyperæsthetic zone. (Erb.)

FIG. 75.



(Combined from Brown-Séquard.)—F, Focus in left side of cord. V, Pyramidal fibers having crossed in medulla. V', Vasomotor fibers, not crossing in cord. *Sm*, Fibers for muscle sense, not crossing in cord: SS S' S', other sensory fibers crossing in the cord. The focus explains the classical signs and also how few of the crossed sensory fibers to the left side are involved (merely a zone of anæsthesia), while all sensory fibers to the right side are severed (hemianæsthesia).

s. The motor are restored more readily than the sensory
s. There is an inactivity atrophy of the muscles without
ı of faradic irritability and without reaction of degeneration;
lysis may be spastic or flaccid. Respiration is rarely affected,
erfect movement of the chest has never occurred. Involvement
horacic and abdominal muscles usually indicates a bilateral
Swelling and œdema in the paralyzed members have been
l, and also swelling with pain in all the joints on the paralyzed

side. The *deep reflexes* are usually exaggerated on the side of the lesion; they are abolished in a sudden total transverse lesion of the lower cervical or upper dorsal cord. The skin reflexes are abolished or decreased on the side of paralysis, and on the opposite anæsthetic side they may be normal, absent or increased.

Hyperæsthesia (Fodera, 1823) occurs on the paralyzed side and also as a zone above the anæsthetic area and near the level of the lesion. If the lesion be cervical or lumbar, the anæsthetic or hyperæsthetic areas above are not zonular but irregular. Hyperæsthesia has been referred to the wound itself or to paralysis of inhibition. Hyperæsthesia or hyperalgesia concern tactile, pain, and heat and cold sensations, and their absence infers incomplete section (Kocher). It usually disappears rapidly. The fibers, the section of which results in hemianæsthesia, lie in the lateral tract near the cerebellar and pyramidal tracts.

The *zonular anæsthesia* on the side of the injury and of motor paralysis is readily explained since the lesion involves not only the sensory fibers (which are ready to decussate to the other side and produce the crossed hemianæsthesia), but also implicates sensory fibers from the contralateral side, which have just decussated to the side of section. Abolition of *muscle sensation* on the side of the lesion is fully in accord with the uncrossed course of the muscle sense fibers in Burdach's column, entering Goll's column higher up. Muscle sense is preserved on the side opposite to the lesion. Vasomotor paralysis occurs on the side of section, as the vasomotor fibers course in the anterolateral columns. "*Sensation is affected on the opposite side*, but not quite up to the level of the lesion, because the decussation of the sensory tract is not immediate but occurs somewhat above the entrance of the nerve" (Gowers). Complete crossed hemianæsthesia occurs clinically in about 60 per cent., and partial anæsthesia (analgesia and thermo-anæsthesia) in about 40 per cent. of cases.

Prognosis.—In clinical and experimental cases almost complete restitution to normal is possible; it is not due to reëstablishment of conduction in the severed tracts, in which ascending and descending degeneration occurs, but rather to assumption of function by the uninjured side of the cord (Kocher). The prognosis and *treatment* of Brown-Séquard's paralysis vary with the etiology of this symptomatic syndrome, which may be syphilis, tumor, sclerosis, myelitis, tuberculosis, trauma, hæmatomyelia, fracture or luxation.

VIII. Concussion of the Cord.

Gussenbauer referred the severe symptoms, as paralysis, to hemorrhage, which is produced in turn by movement of the cerebrospinal fluid. Long ago Ollivier explained the symptoms as being due to molecular alteration in the nervous tissue; this was confirmed by cases of experimental concussion (Schmaus and Bikeles) which resulted n degeneration of the cells and cylinders. Myelitis and chronic legeneration may follow concussion. The "railway spine" (Erichsen) s one of the traumatic neuroses. Obersteiner considers that long ontinued moderate jarring, as in the case of tramway drivers, may njure the cord. *Symptoms* develop at once or sometime later, as paraparesis, paraplegia, retention of urine and anæsthesia, less often than paræsthesia. Some cases recover after months to a year or two. In others the symptoms are permanent; in still others death, usually early, occurs. In *diagnosis*, fracture, hæmatorrhachis, hæmatomyelia and traumatic neurosis should be considered. Simulation, in order to obtain damages for accidents, may be difficult to exclude, but some railway surgeons and medical experts in corporation employ, err in discounting the importance of concussion and traumatic neurosis (*q. v.*). Paraplegia and retention of urine are indicative of structural alteration of the cord. *Treatment* consists of rest, local application of cold and avoidance of neurotic tendencies.

(F). INFLAMMATION OF THE CORD.

Myelitis is inflammation of the cord. It was described by Oliver and Abercrombie a century ago; the term has long been made to cover many affections which are not properly inflammatory, as compression paralysis. Oppenheim and Marie have vigorously disputed the opinion of Gowers, v. Leyden, and Erb, that myelitis is a frequent affection.

I. Acute Myelitis.

Etiology.—There are two prominent causes, infections and intoxications. The affection occurs largely in males between ten and forty years of age.

1. Acute Infections.—Experimental myelitis has been produced by the B. pyocyaneus, B. diphtheriæ, B. coli, B. typhosus, strepto- and staphylococci, avian and human tuberculosis. French writers have contributed most to this subject. It has followed epidemic meningitis, variola, typhus, measles, rheumatism, and other infections, caused (*a*) by the germs themselves or secondary infection (focal myelitis resulting) or (*b*) by their toxins (disseminated myelitis resulting).

2. Intoxications.—These include poisoning by alcohol, ergot, and lead, and perhaps auto-intoxications, as in cancer, or severe anæmia.

All other causes are doubtful; trauma (fracture, concussion, compression by caries or cancer) is often considered of etiological importance but probably causes softening or hemorrhage, to which infection may of course be added; exposure to cold and dampness most likely reduce the physiological resistance, thus indirectly favoring localization of bacteria; syphilis (*q. v.*) may affect the meninges and cord (meningomyelitis), but the so-called syphilitic myelitis is usually ischæmic softening from vessel changes. The etiological importance of sexual excesses and emotional shocks is at present sharply questioned, as is von Leyden's theory that infection from the bladder may reach the cord by ascending neuritis.

Pathology.—On macroscopic examination we find redness and swelling; on cross section, blurring of the cord tissues, and decreased consistency, even to fluidity; as in the brain, "red softening" (hemorrhagic myelitis), and in older cases "yellow softening" from fatty change, or "white softening" and cavity formation occur. Myelitis can usually be seen to best advantage after hardening in Müller's solution, the normal tissue then appears green and the diseased, yellow. In the ultimate stages, atrophy, cyst formation, induration and very rarely abscesses are found. *Microscopically* there are, *in fresh cases*, degenerated swollen axis cylinders, the disintegrating medullary sheaths of which may form myelin drops; granules and granule corpuscles (leukocytes or vessel endothelial cells); extravasated red and white cells; turgid vessels the nuclei of which proliferate; choking of the perivascular lymph spaces with cells; degeneration, blurring and vacuole formation of the nerve cells the processes of which shrink and the nuclei vanish; granular amorphous deposits of coagulated albumin in and about the cells; in some cases the parenchymatous far exceeds the interstitial alteration, and often parenchymatous myelitis were better called parenchymatous degeneration. The gray matter (poliomyelitis) is involved much more often than the white (leukomyelitis). In *older cases*, there are corpora amylacea, empty spaces corresponding to destroyed nerve fibers; large "spider" cells with many processes (Deiter's cells); and there is disappearance of the vascular changes and substitution of destroyed tissue by focal interstitial proliferation. In rare instances bacteria are found, as streptococci, or the colon or typhoid bacilli, but probably the toxins are more important than the organisms. The microscope differentiates between the ischæmic inflammation and postmortem softening. The *degree and the extent* of inflammation vary greatly; *transverse myelitis* may destroy an entire cross section; or *disseminated* foci may involve at random various structures at different levels. *Meningomyelitis* (perimyelitis, annular or cortical myelitis) chiefly affects the periphery of the cord, whence concentric invasion along the septa may split the cord structures; this is frequent in the syphilitic form. *Poliomyelitis* (inflammation of the gray substance) is really a type of myelitis, but involving as it does only the anterior horns, is usually classified by itself. Degeneration and inflammation of the motor and sensory *roots* may follow myelitis in the cervical or lumbar enlargement; secondary degenerations are common; the inflammation may ascend or descend the cord, but usually affects only one or two segments.

Symptoms.—The symptoms vary with the extent, form and localization. The localized form, limited to a small segment, is called *transverse*, while the diffused form is known as *disseminated myelitis*.

Acute transverse myelitis is the prototype. The cord symptoms re usually first in time and always first in importance, and constitutional symptoms as fever, chills, malaise, and in children convulsions, ometimes may precede but usually attend the cord symptoms. The ymptoms resulting from *involvement of the cord* are (*a*) *paralysis*, hich is usually early, rapid but not apoplectic in onset, and is prominent, paraplegic, progressive, flaccid and rather complete. In omplete paralysis, the flexors are usually weaker than the extensors. he toes alone may be moved; twitching of the paretic muscles is ually associated with pain. Clonic muscular spasms are sometimes resent, inappropriately designated by Brown-Séquard as spinal pilepsy. The paralysis is often preceded for a short time by sensory henomena, as numbness, or by darting pains in the limbs, joints or ack, which are usually neither conspicuous nor severe; it is less often receded by hyperæsthesia, local muscular contractions, or general onvulsions, especially in children. The paralyzed limbs show a ransient rise of temperature, followed by somewhat subnormal emperature. The limbs are often dry and may not sweat even after njection of pilocarpin (Brieger). (*b*) *Sensation* is frequently involved. he initial tingling pain has been considered. Anæsthesia may be omplete to the level of the lesion, but more often incomplete (hypæsthesia), and rarely partial, according to the cordal tissues involved. It higher anteriorly than posteriorly; a zone of hyperæsthesia or "girdle ain" marks the upper level; it is caused by early irritation or late catrization. Severe pain suggests vertebral or root disease rather han myelitis; gastric crises like those of tabes, dysæsthesia (Charcot), *e.*, diffuse vibrating sensations over the entire limb from local imulation, as by a needle, and ataxia (which is usually obscured y the paraplegia) are much less common. (*c*) At the onset all *reflexes* ay be lost, but they are usually increased below the focus, because f lessening of the cerebral inhibition traveling in the lateral yramidal tracts, the skin reflexes being increased early and the muscle reflexes later; involvement of their centres causes permanent oss of the reflexes; for instance, they are lost in the arms and increased n the legs in cervical myelitis; lost in the trunk and increased in the egs in dorsal myelitis; lost in the legs in lumbar myelitis. (*d*) The *sphincters* are affected almost constantly and early, perhaps even before he onset of paraplegia; initial difficult urination (ischuria) or urinary etention, possibly with sphincter spasm and later with incontinence, s usual; when the bladder is full it may dribble from relaxation of he sphincter: this is known as ischuria paradoxa or retention with ncontinence; the patient may or may not be conscious of the retention, epending on whether the afferent sensory fibers are involved or not; lumbar myelitis incontinence prevails from the beginning. The rine is at first alkaline and later ammoniacal on the advent of cystitis. he sphincter ani acts involuntarily, *i. e.*, is spasmodically contracted; he paresis of the bowel and abdomen are factors in constipation; in umbar myelitis the sphincter is paralyzed. (*e*) *Trophic changes* may occur. Muscular atrophy and the reaction of degeneration are caused by disease in the anterior horns and the muscles also waste from inactivity. The skin is usually dry, sometimes glossy or the seat of papular or vesicular eruptions, shedding or over-growth of the hair.

Cord Symptoms Grouped According to Localization.
(Amplified from Morton Prince.)

In order of frequency.	*Dorsal myelitis* most frequent.	Then *Lumbar myelitis.*	Lastly, *Cervical myelitis.*
Paralysis.	Trunk, back, intercostal and abdominal muscles (favoring mucus stagnation in lungs). Arms normal. Paraplegia, first flaccid then spastic (lateral columns involved).	Trunk and arms normal. Paraplegia flaccid throughout (ant. horns involved).	Neck muscles, diaphragm, arms, trunk and legs paralysed. Typically, atrophic flaccid paralysis of arms (ant. horns involved), with flaccid, later spastic paralysis of legs (lat. columns involved): however, paraplegia brachialis with paralysis of legs is very rare. In rare lesions high in the cervical cord, the neck muscles atrophy and both arms and legs show simple, then spastic paralysis, without wasting.
Atrophy.	In trunk; difficult to elicit. In legs; only from inaction.	None in arms or trunk. Atrophy in legs: (ant. horn involved).	Atrophy of neck muscles if high lesion (rare). Usually of arms only (ant. horns involved at level of lesion). In legs only from inaction.
Reaction of degeneration.	Present in trunk muscles; absent in legs (or only quantitative reduction).	Present in legs or in mild cases quantitative reduction.	Present in arms; (rarely in neck).
Sensation.	Girdle pain and hyperæsthesia, between ensiform and navel. Anæsthesia corresponding with motor paralysis.	Pain in loins or legs; hyperæsthesia in loins. Anæsthesia of legs.	Pain and hyperæsthesia in arm nerves (with highest localization); anæsthesia in arms (lower localization); and the trunk and legs.
Reflexes.	Superficial reflexes, initial loss, rapid return and increase.	Lost.	Superficial reflexes; initial loss, rapid return and increase.
	Deep reflexes, initial loss, slow return and increase.	Lost.	Deep reflexes; initial loss, slow return and increase.
Sphincters.	Bladder, initial retention, later intermittent overflow or incontinence. Cystitis common.	Incontinence from beginning (sphincter paralysis).	Same as in dorsal myelitis.
	Bowels, usually spasm of sphincter ani; constipation.	Incontinence (sphincter paralysis) disguised, perhaps, by constipation.	Same as in dorsal myelitis.
Trophic changes, etc.	Decubitus from neglect, etc.	Decubitus, cystitis, etc., from neglect and trophic alteration.	As in dorsal lesions. (Occasionally very high temperature.)
Pupils.	Klumpke's paralysis of pupil (if in upper dorsal).	Absent.	Paralysis of pupil (lowest cervical region). Optic neuritis in isolated cases.
Priapism.	Often present and painful; sometimes ejaculation at onset.	None. Impotence.	Priapism often present.
Mode of death.	Cystitis, decubitus.	Decubitus, cystitis.	Medulla symptoms, vomiting, slow pulse; usually by involvement of phrenic nerve with early death.

ıe joints are sometimes swollen and the limbs œdematous. Bed- es are frequent over the sacrum, trochanters, heels, between or even er the knees, from the bed covers; they result from neglect or in nbar myelitis from trophic disturbance (disease in the gray matter posterior horns). The same factors are said by some writers to use cystitis, ulcerations in the bladder or urethra, and the tendency cellulitis or phlegmon.

Course and Prognosis.—The onset may take place in a few hours ıe apoplectic form), or in several days to a week (the subacute form). *ur stages* are described, which however, blend: (*a*) the more or less ute *initial stage;* (*b*) the *advancing degeneration;* (*c*) the *restitution*; d (*d*) the *stationary* (residual or chronic) stage. Complete para- egia and anæsthesia may persist, but more often sensation returns ter some weeks, while movement returns far more slowly and incom- etely, usually with some residual spastic paraplegia. Contractures ay develop, especially in the adductors and the knee flexors, in which se the prognosis is particularly unfavorable, for profound disease is dicated. Contractures, muscular contractions, and increased re- xes are caused by secondary degeneration. The immediate outlook myelitis is always uncertain, and ultimate recovery is usually incom- ete; it depends (*a*) on the etiology, the prognosis being best (Oppen- im) in acute infections; (*b*) on the intensity of inflammation; (*c*) on its nsverse or vertical dimensions, and (*d*) on the rapidity or tardiness signs of improvement. Complete recovery in mild circumscribed rms is barely possible, since indirect symptoms, due to collateral dema may disappear. Death results from extension to the medulla ausing slow pulse, and vomiting), phrenic nerve phenomena, and psis following cystitis, pyelitis, or pyelonephritis. A form of recur- nt myelitis is said to exist (*myelite à rechutes*).

Diagnosis.—The early onset of structural cord symptoms, as reten- n of urine, followed by paraplegia and by anæsthesia, is especially nificant after acute infections. Very rarely is there local tenderness. he upper level of the myelitis corresponds to that of the paralysis or æsthesia. The lower level may be determined by the condition of e reflex centres. Application of a weak faradic current shows to at extent the gray matter is involved. *Anterior poliomyelitis* (*q.v.*) es not involve sensation, and but rarely extends back of the anterior rns. Compression by *caries*, or cancer usually causes local vertebral anges and root symptoms precede cord symptoms. The so-called *philitic myelitis* is usually ischæmic myelomalacia; it begins acutely subacutely without pain, and is rarely relieved by antisyphilitic medies. *Hemorrhage* in the cord produces most sudden symptoms, d acute spinal pain without prodromes; a precipitate onset may use confusion instead of being of diagnostic value, in case of apoplec- myelitis. For differentiation from *Landry's paralysis*, see page 02, and from *multiple neuritis*, see page 1145. *Meningeal hemorrhage* d meningitis produce unmistakable root symptoms. *Abscess of the rd* is rare, for few more than a dozen cases are reported; its symptoms are essentially those of myelitis, plus meningeal symptoms. *Hysterical paraplegia* has a psychical etiology; it is characterized by other stigmata of hysteria, motiveless variability of the symptoms, little myotatic irritability, little extensor spasm (and then both legs move together when one spasmodically extended leg is lifted); rarely by incontinence of urine or fæces, no trophic changes, no cystitis, no reaction of degeneration, and no decubitus.

Gower's scheme for diagnosis from the *method of onset* is here given:

DISEASE.	ONSET.	DISEASE.
	Sudden (few minutes).	Vascular Lesions.
	Acute (few hours or days).	Inflammation.
Pressure and Growths.	Subacute (1 to 6 weeks).	
	Subchronic (6 weeks to 6 months).	Degeneration.
	Chronic (more than 6 months).	

In the *chronic stage* the diagnosis of what has gone before must be uncertain, unless the history is most definite; the diagnosis is usually provisional, and atypical cervical pachymeningitis, tumors, and most often multiple sclerosis of the spinal type, should be considered.

Treatment.—This is symptomatic. In the *acute stage*, the indications are (*a*) absolute rest in bed for at least two weeks, keeping the patient on one side or on the face, with possibly local abstraction of blood and local warmth. Blisters should be avoided lest they develop into decubitus. (*b*) Gowers recommends spt. etheris nitrosi with digitalis to equalize the circulation and promote diuresis. (*c*) When hemorrhagic myelitis is suspected ergotin (grs. iij to v) may be given three times daily. (*d*) Mercury and iodides are usually without effect in syphilitic myelitis (softening) or are possibly directly injurious, although some rationally recommend their use unless unfavorable effects appear. (*e*) Care of the skin is necessary. Though bed-sores may be trophic, practical experience shows that care greatly reduces their frequency and extension. Pressure and heat should be avoided by change of posture, by use of rubber cushions, soft thick pads of cotton or the water bed. As in typhoid, the use of two beds is very beneficial, and the sheets should be stretched smooth. Alcohol rubs are antiseptic and harden the skin. Moisture and filth are most dangerous; the bowels should be irrigated if they move involuntarily; incontinence of urine is more dangerous, especially in women; hard urinals, if left between the legs, may cause equal damage; the urine should be gathered in bags of oiled silk filled with cotton; in women mild antiseptic gauze should be disposed about the genitalia. The author has used successfully the rubber dam used by Dr. Murphy in laparotomies, by spreading it over the sacrum and buttocks after carefully cleansing and drying the skin and washing it with ether, before applying the dam. (*f*) Care of bladder is imperative. Cystitis develops in most cases in which the catheter is used, or in which there is incontinence with retention; residual urine is easily infected, probably by infection ascending the dribbling, patent urethra; the urethra should e irrigated before the absolutely clean catheter is introduced; lavage ith Thiersch's solution in cystitis with retention or incontinence, d grs. v to x, each of urotropin and salol t.i.d. are indicated. (*g*) he paralyzed muscles should be gently massaged from the very onset. In the *subacute or chronic stage,* tonics, massage after four weeks, ild faradism of the muscles of the bladder and rectum (not those of e cord, and only when there is muscular wasting or incontinence) e indicated. Bruns considers spinal galvanization most efficacious. ontractures are modified or prevented by warm baths and massage. ot baths are directly dangerous.

II. Acute Multiple Disseminated Myelitis.

This affection is relatively infrequent, although of late more cases re reported. It is usually subacute in onset; foci develop irregularly, gether or successively in different parts of the cord or perhaps of the rain (*disseminated encephalomyelitis*), with acute ataxia, scanning eech, dysarthria, tremor, nystagmus, optic neuritis, spasticity, or eakness of the limbs. Sensation and the sphincters are usually not fected. The clinical picture is necessarily irregular, because the inflamation may occur anywhere; von Leyden distinguishes (*a*) the atactic, d (*b*) the paraplegic form. A myelitis which spreads after the first o or three days is of the disseminated type, and may be fatal in a w days to a few weeks; though it was once thought very fatal, numers of recoveries have been reported lately. Treatment is as in the ute transverse variety.

III. Chronic Myelitis.

This may be the stationary stage of a passed acute myelitis, or in ery rare cases, myelitis may be chronic from the beginning, *i.e.*, it ay have a gradual onset and slow progression. Many deny the xistence of this form and classify it under the paraplegic state, muliple sclerosis, or chronic system-disease. If myelitis, chronic from the nset, is to be recognized, its symptoms differ from the acute form only or chiefly in the mode of onset.

IV. Acute Poliomyelitis of Children.

Acute poliomyelitis, also known as atrophic paralysis, was first described clinically by v. Heine (1840), and pathologically by Cornil (1863), although Prévost and Vulpian (1865) first accurately defined the changes in the ganglionic cells of the anterior horn. The famous ontribution of Charcot and Joffroy (1870) fixed the trophic relation f the special cells involved to the atrophy of the nerves and muscles supplied, and stated that the disease was due to primary inflammation of the cells themselves. Archambault and Damaschino found the primary inflammation in the vessels (interstitial myelitis).

Definition.—An acute, probably independent, infection, characterized pathologically by acute myelitis of the anterior horns, and clinically by the incidence in children of an acute flaccid paralysis with wasting in the muscles, loss of reflexes, the reaction of degeneration and later, contractures.

Etiology.—In some statistics it constitutes as high as 8 per cent. of the diseases of childhood.

1. *Age.*—The disease largely occurs between the first and fourth years of life (mostly in the second and third); 60 per cent. of cases occur in the first three years, and it is ten times as frequent in the first decade as in all the others. Its occurrence in youth is difficult to explain although Lange refers it to functional activity of the cord, as learning to walk, etc. It has no causal connection with dentition, as Underwood thought, although often synchronous with it.

2. It may follow such *infections* as measles or scarlatina, and Marie thinks that it may result from many others, (*infectiones innomminées*). In some localities it has occurred as an independent epidemic, as Caverly's series of 140 in Vermont, or Medin's series of 44 in Sweden. Alice Woods describes an epidemic of 60 to 70 cases in San Francisco, in May and June, 1901.

3. Eighty per cent. of cases occurs in the *summer months* (Sinkler). Cold and trauma are improbable factors. Sex is of no importance, save after the tenth year, when the disease is almost entirely confined to males (Gowers).

Pathology.—The affection is called poliomyelitis (Kussmaul), because it is an inflammation of the anterior gray matter, which is supplied by the central arteries of the anterior median artery of the anterior longitudinal fissure. A single vessel does not supply all the anterior gray matter, but branches up and down, supplying a cylindrical vertical area which measures two inches or more; this is the usual dimension of a poliomyelitic focus. The inflammation may extend somewhat back of the anterior horns into the white matter of the antero-lateral columns. Fresh cases are rarely seen, since death is unusual; to the naked eye, the focus is red, soft, juts above the cut section and is mostly confined to the anterior horn, *i.e.*, it involves the beginning of the second (or lower) motor neurone. Microscopically, the lesion is inflammation in and about the vessels above named, in the anterior roots, and in the vessels even before they enter the cord. The vessels are engorged; their perivascular spaces are full of cells and granule-cells; there is exudation of red and white cells with actual hemorrhage in the severe cases, as shown by blood pigment; the ganglion cells in the anterior horn are swollen, glistening, granular, pale, distended, decreased in number, and surrounded by leukocytes; their dendrites may shrink, their nuclei disappear, or the whole cell may disintegrate, while here and there some cells escape; no definite cells are involved, e the process depends solely on the vessel affected. The involve-ıt is sometimes bilateral, and in severe cases is distributed through ral segments, especially in the lumbar cord. As may easily be cipated, disease in the trophic cells of the lower motor neurone must ollowed by degeneration of their axis cylinders, nerve roots, peri-ral nerves and muscles (which become granular, fatty, and later ırated). In *old cases*, macroscopic inspection shows shrinking of anterior horns, perhaps also of the contiguous white matter, with of differentiation between the gray and white matter. Micro-ically, the (formerly inflamed) vessels are thickened and pig-ıted; groups of ganglion cells have disappeared, and their nerve fibers degenerated or wholly wasted, which led Charcot to hold that the ess was essentially in the ganglionic cells, but the connective ıe also is rich in cells and stains deeply with carmine, because it is ker than normal; in other words, a cicatrix remains from the pri-y inflammation. In *severe cases*, identical changes have been ıd in the medulla, pons, and centrum ovale. Acute poliomyelitis rs from acute myelitis and disseminated myelitis only in its locali-on, for which there is no universally accepted reason; the gray tance is more vascular, and Lamy's experiments, in which he cted powder into the abdominal aorta, always showed embolic ening of the gray matter first.

ymptoms.—There are *three clinical periods:* 1. The acute stage luding prodromes): 2. The stage of diffuse paralysis, when degen-ion appears and regeneration occurs: 3. The stationary stage, ı some improvement and determination of the paralysis to given ıps of muscles.

The Acute Stage.—The typical onset is acute, appearing in a d previously well, with fever (75 per cent.), often with vomiting, lache, somnolence, and sometimes with convulsions or coma, ch are followed by a sudden complete and flaccid paralysis in one or more than one limb; it corresponds to the acute inflammation ıe anterior horn. The fever averages 101°, may reach 105° or more, lasts about 24 hours. Convulsions are not as common as in the bral type; they occur mostly in younger subjects and are attributed ıaps to teething, which was once thought the cause of the paralysis. simple motor paralysis usually reaches its maximum in twenty-to forty-eight hours; it is not progressive; its distribution will be sidered under the next stage. In some cases *prodromata* exist, ch are not characteristic; they are malaise, anorexia, pain in the k and limbs or diarrhœa; their average duration is one to two days. *usual variations* in the onset are possible; the fever may last one wo weeks before the paralysis develops, it may be short, lasting a few hours only, or may be absent throughout the entire course. paralysis may develop during the night. Abortive cases are most ptional and are probably often due to neuritis; the paralysis may w some acute infection, as measles or scarlatina. Sometimes par-is is not noticed for a long time, until, for example, the child fails to walk. In some cases pain is the first symptom in older children, and is referred to the back or extremities. Convulsions involve principally the extremities, and sometimes occur in the form of clonic muscular contractions; Strümpell observed them for more than a month before the paralysis developed. The onset may simulate acute rheumatism.

2. **The Stage of Degeneration.**—This results from localization in the anterior horn, which produces (*a*) paralysis, (*b*) disturbed nutrition, (*c*) vasomotor changes and (*d*) loss of reflexes. (*a*) The *paralysis* is motor only; it is sudden, complete, flaccid and nonprogressive; though at first diffuse, it usually regresses, although incompletely, leaving some permanent paralysis. *General localization*, according to Starr's combined statistics of about 600 cases gives: (i) Paralysis, most frequently of one leg, then (ii) of both legs, and (iii) and (iv) of all four limbs or of one arm (with equal frequency), (v) of arm and leg of same side (spinal hemiplegia), (vi) of arm with opposite leg (crossed or alternating paralysis), (vii) of three limbs, and least often, (viii) of both arms. The trunk muscles may be involved, but the cranial nerves are almost always exempt, save in severe and diffuse foci. The bladder is usually unaffected, except in the early stage, from general toxæmia, or in severe cases. As to its *special localization*, the *leg* is paralyzed in 75 per cent. of cases (E. Remak); in one form, the lasting paralysis affects the quadriceps femoris with the adductors (upper lumbar lesion), but the sartorius is not involved; in another form, the muscles below the knee supplied by the peroneal nerve, are involved, as the tibialis anticus, extensor digitorum communis, ext. longus or brevis, ext. hallucis longus, peronei (lower lumbar and upper sacral lesion) of which some muscles may escape as the tibialis ant., since total paralysis is rare. If both legs are involved, the paralysis is rarely symmetrical. Poliomyelitis usually involves several segments, because of the arterial distribution. The arm is paralyzed in 25 per cent., chiefly in the form of the combined shoulder paralysis of Remak, involving the deltoid (sometimes alone), biceps, brachialis, supinator longus with the supra- and infra-spinatus and teretes (lesion in upper cervical enlargement, fifth to sixth segments); in some cases the forearm type may be seen, with involvement of the flexors or possibly of the extensors, while the supinator longus often escapes (lower cervical lesion, seventh and eighth segments). The trunk or neck muscles usually show improvement in this stage. (*b*) As to *disturbed nutrition*, the muscles and nerves corresponding to the level of the poliomyelitis rapidly degenerate as shown by early reaction of degeneration and muscular atrophy The reaction of degeneration consists of failure of the nerve and muscles to respond to the faradic current and usually appears in one or two weeks (Duchenne) or rarely earlier; it also includes a lack of response of the nerve to the galvanic current, and finally too prompt a response of the muscles to the galvanic current and more quickly to the positive than to the negative pole; this latter reaction lasts for two, three or six months and then gradually disappears in a year or two. Muscular

atrophy may be apparent in two weeks and is more clearly seen in the arm than in the leg or trunk, whose adipose deposit may obscure it. Fibrillary contractions are rather frequent. The wasted flaccid member hangs and swings in a lifeless fashion. (*c*) *Vasomotor paralysis* causes the low temperature (1 to even 10° below normal) and the livid cyanotic or sometimes clammy skin. (*d*) The skin and tendon *reflexes* are abolished in the limb at first; the skin reflexes usually return later. The *sphincters* are almost always intact and sensation is very rarely affected. True trôphic changes are almost unknown.

3. **Stationary, Chronic, Residual Stage.**—Some improvement may occur, but this stage is generally marked (*a*) by accentuation of *muscular atrophy*, connective and sometimes adipose tissue replacing the contractile substance; it is most marked in the legs in old cases, and involves also the nerves, arteries, and tendons. (*b*) The *bones* become thin, flexible, friable, arrested in development and therefore shorter, since the bone cortex wastes and its medulla becomes more spongy; scoliosis may result. (*c*) There may be pronounced vasomotor lividity. (*d*) In the *joints*, nodules may appear as in cerebral hemiplegia; the joint structures are relaxed, and the "flail joint" may develop with luxation or subluxation. (*e*) *Contractures* are due principally to the mechanical influence of the unopposed antagonist muscles on the posture of the joint; if all the muscles are paralyzed they sometimes result from static causes, as from standing or attempting to walk, *i. e.*, from neglect; trauma and malposition of the luxated joints or tendons may also cause contracture or actual deformity; the arm, the shoulder, wrist and fingers are usually flexed and the elbow usually escapes involvement. In the leg, deformity (pes equinovarus) is more common than in the arm.

Diagnosis.—The initial vomiting, fever or convulsions are usually misinterpreted until the diffuse purely motor paralysis appears, and consequently, the physician is unjustly blamed for what he cannot anticipate. The age, acute paralysis, rapid atrophy, reaction of degeneration and loss of reflexes are positive diagnostics, together with the absence of sensory and sphincter disturbance. Bedsores "practically never develop."

Differentiation.—(*a*) The various forms of *muscular atrophy* in children are gradual in onset, progressive, symmetrical, and often familial. The atrophy precedes and exceeds paralysis, and there is no arrest in development. The neural progressive muscular atrophy of Hoffmann, due to disease of the nerves or nerve roots, may produce paralysis, atrophy and the reaction of degeneration, but the affection is progressive, familial and involves sensation. (*b*) Birth palsies are distinguishable by their history, and more favorable course. (*c*) *Multiple neuritis* (*q.v.*) has several distinctive features. Poliomyelitis is due to a local inflammation, is unilateral and spinal in type—or if bilateral, it is asymmetrical. Multiple neuritis is hæmatogenous and toxic, and therefore affects the spinal, and often the cranial nerves symmetrically and bilaterally, and usually involves the arms and legs; ataxia, sensory disturbance and local tenderness over the nerve trunks are common, its onset is more subacute and convulsions are rare; the reaction of degeneration is less intense and the issue is much more favorable. (*d*) The *cerebral palsies* of children (*q. v.* for table) affect the upper motor neurone and therefore never produce marked degenerative atrophy, or loss of faradic irritability. Its hemiplegic form involves the face and its paraplegic form is spastic. Disturbance of the intellect, focal or general convulsions and hemichorea are common. In rare cases, the cerebral and spinal forms of infantile paralysis may co-exist. (*e*) *Myelitis* also begins acutely, but usually in older subjects. Poliomyelitis may be confused only when it is bilateral, located in the cervical or lumbar enlargement, when sensory changes occur (2 per cent.), when the sphincters are involved or when the poliomyelitis reaches back into the white matter, producing a flaccid atrophic paralysis of an arm and a spastic paresis of the leg, the latter of which is only transitory. Bed-sores, which are common in myelitis, are "almost unknown" in poliomyelitis. (*f*) Painful affections causing immobility of a joint, hip-joint disease, Parrot's syphilitic pseudoparalysis and infantile scurvy bear a superficial resemblance to poliomyelitis. Differentiation is easy. Westphal noted that in the new-born the electrical reaction normally resembles that of the reaction of degeneration. (*g*) Hysterical contractures, sometimes with atrophy, disappear under anæsthesia and show no reaction of degeneration.

Prognosis.—(1) *Regarding life;* there is little danger after the fever subsides; life is very rarely threatened from extension to the medulla oblongata. In exceptional cases, death may result from antecedent pneumonia. (2) *Regarding relapses;* second attacks almost never occur. (3) *Regarding recovery;* improvement is the rule. The diffuse initial paralysis usually becomes localized, but complete recovery is so infrequent that it may be disregarded. A localized initial paralysis may show but little improvement, and an extensive initial paralysis may diminish greatly. A definite prognosis of the ultimate extent of the paralysis cannot be made for two weeks and then only by the electrical examination. Muscles which show no reaction of degeneration after fourteen days will probably recover; those showing slight loss of faradic irritability will recover in great part; after six weeks only those muscles are paralyzed which will remain paralyzed; if there is no recovery in three months, there will be none or it will be slight.

Treatment.—In the *acute stage* rest, a light diet, laxatives, diaphoretics and diuretics are indicated. After the fever, the patient should be kept in bed for weeks, and should be given luke warm baths. Electricity is contra-indicated. As the paralysis begins to recede, a fuller diet and careful passive movements are beneficial. In the *chronic stationary stage*, with its inevitable and permanent paralysis, fresh air and liberal nourishment are important. Proof is lacking that strychnine is valuable, but it probably is because of its general tonic action, and its almost specific physiological action on the motor elements of the cord. It should be given three or four weeks after the paralysis becomes stationary. Full doses may induce muscular spasm, pain and irritating paræsthesia. Electricity cannot restore totally destroyed tissue, but it hastens recovery in partial lesions; if applied to the spine, it is useless, but the constant stream to the muscles and the faradic current to the skin are helpful. Contractures may be modified as well as deformities due to neglect, to which gymnastic movements, massage and orthopedic apparatus are essential. The child should be kept in bed for a long period. In some cases tenotomy, arthrodesis and tendon transplantation, by attaching the tendons of sound muscles to those of paralyzed muscles (Vulpius, Nicolodoni) are indicated. Nerve transplantation is in its infancy, but is proving very successful.

V. Acute Poliomyelitis in Adults.

This is rare in comparison with the infantile form. It was established by Vogt in 1858, Moritz Meyer, in 1861, and anatomically by Gombault, in 1873. The *etiology* is practically the same as that of the infantile variety; most cases occur between the twentieth and thirtieth years. The *symptoms* and course are essentially the same, with the following variations: fever is almost always present, which with the prodromes lasts longer than in children; it often lasts a week. There is the same flaccid paralysis with atrophy and the reaction of degeneration, but it regresses less and results less frequently in contractures. The types of Remak are more clearly developed in the adult. Pain is a more prominent symptom. Friedländer observed mixed cases in which there was early although transient anæsthesia. Though the *diagnosis* is easy in children, it is more uncertain in adults, chiefly because multiple neuritis and to a lesser degree myelitis and hæmatomyelia are more difficult to exclude but in all three affections sensory alterations are usual.

VI. Subacute and Chronic Poliomyelitis.

Many reported cases are probably multiple neuritis, although Oppenheim has published perfectly clear cases. *Etiology.*—Metallic poisons and syphilis are important factors. *Pathologically,* two main types are distinguished; in the larger group there is no inflammation, but rather a seemingly primary atrophy of the ganglionic cells, which heightens its resemblance to spinal progressive muscular atrophy, than which, however, it runs a more rapid course; in a smaller group of cases there is early perivascular exudation in the anterior horn and later sclerosis, wherein it resembles acute poliomyelitis, although it is slower in its course. The anterior roots are involved and in most cases there is degeneration in both pyramidal tracts, possibly also in the posterior columns or roots, in Clarke's columns or rarely in the medulla. The *course* is gradual, for one extremity becomes slowly affected (in one to three years), sometimes with extension to others after months or years. There is paresis with atrophy, fibrillary tremors, partial or complete reaction of degeneration, decrease or absence of the reflexes and normal sensation and sphincters. Life is rarely immediately threatened, but recovery is most unlikely; if it occurs, neuritis, not poliomyelitis, is probably the actual lesion. The ultimate outcome is unfavorable. *Differentiation* is, as follows: (*a*) Multiple neuritis (see ACUTE POLIOMYELITIS); (*b*) from *progressive spinal muscular atrophy*, even an anatomical differentiation is not easily made. As a rule, the following clinical distinctions hold; in progressive muscular atrophy, atrophy precedes and exceeds the muscular weakness, fiber after fiber of muscle very slowly wastes, the atrophy is especially marked in the distal parts as in the hands and fingers and the reaction of degeneration is usually less complete. In chronic poliomyelitis, atrophy always rapidly follows paralysis; it is an outright atrophy (*atrophie en masse*), it occurs more often in the proximal parts, as in the shoulder, and the reaction of degeneration is marked. (*c*) In amyotrophic lateral sclerosis the paralysis is spastic with increased reflexes, contractures and often with bulbar symptoms. *Treatment* is the same as that of the chronic stage of acute poliomyelitis.

VII. Landry's Paralysis.

Landry's paralysis (acute ascending or centripetal paralysis) was described by Landry and independently by Kussmaul in 1859. Landry's description included five cases of his own and five others; the famous Cuvier died of this disease, in 1832. Acute ascending paralysis is a symptom complex rather than a disease in itself and may be conveniently classified under inflammatory affections of the cord. The *etiology* is vague. The affection occurs mostly between twenty and forty years of age and three times more frequently in males than in females. It has followed acute infections, as diphtheria, typhoid, influenza or variola and also chronic infections as syphilis and tuberculosis.

Pathology.—In the early and in many recent cases no anatomical findings existed, and on this Westphal (1876) founded his three criteria: (*a*) that the disease is a progressive, ascending and finally fatal affection with (*b*) normal electrical reaction and (*c*) negative findings at autopsy. Though negative findings are frequent, recent cases have given definite though varying postmortem results, as structureless exudation in the cord, disseminated foci of inflammation in the cord, medulla and pons, changes in the pyramidal tracts, anterior horns and in the spinal and sometimes in the cerebral peripheral nerves. By some writers two forms are distinguished: (*a*) the *bulbar or medullary form*, in which sensation and electrical re-

tions are normal, and (*b*) the *peripheral neuritic form,* with sensory
nd electrical alteration. The parenchymatous swelling of the liver,
pleen, kidneys and lymphatics indicates a general infection or tox-
mia. Bacteriologically, an anthrax-like bacillus (Baumgarten), the
neumococcus, typhoid bacillus, bacilli in all the peripheral nerves
nd streptococci (Œttinger and Marinesco) have been found.

Symptoms.—The onset is acute with short prodromes, as head-
nd back-ache, muscular spasms, and paræsthesia, or it is without
odromes. Paralysis usually commences in one foot, then in the
her, and develops in the legs, thighs, abdomen, back, arms, even in
e face or the eyes, possibly with optic neuritis; the paralysis is pro-
essive and ascending; when the medulla is invaded, dysarthria,
sphagia and paralysis of the tongue occur and finally death results
respiratory paralysis or in atypical cases by circulatory paralysis.
he proximal parts of the limbs are more involved than the distal and
me muscles may be unaffected. Fever is usually absent; the sen-
rium, sensation, sphincters and electrical reaction are usually normal.
here are no tremors, no twitchings, no convulsions, no contractures,
trophic disturbance. The reflexes are variable, but are usually
creased or absent. In rare cases there may be partial reaction of
generation (which usually has not time to develop), atrophy, and
oderate disturbance of sensation or the sphincters. The paralysis
ay be of the descending type, in which event the bulbar involvement
fatal before the paralysis in the legs is well developed.

Course and Prognosis.—The disease lasts two or three days to one
two weeks, and usually terminates with a respiratory paralysis.
ulbar symptoms are ominous, but not necessarily fatal. The central
rm of paralysis is fatal and the peripheral form probably includes
ost of the recoveries. In rare cases, death may occur in a few hours
only after months, with remissions and exacerbations. In the
ll rarer cases of recovery, chronic paralysis of some part may re-
ain. If recovery ensue, the paralysis regresses in an order inverse to
at of development. A *diagnosis* is usually easily made, because of
e ascending motor paralysis with loss of reflexes and without marked
ental, sensory, sphincter or electrical alteration. The *treatment* is
at of myelitis or multiple neuritis. Administration of strychnia and
her stimulation is indicated if the bulbar symptoms are manifested.
L. Greene reports a case which was kept alive by artificial respira-
n forty-one days after respiratory failure set in; he advises calomel,
lonic flushings and oxygen inhalations.

MULTIPLE SCLEROSIS.

Insular or disseminated sclerosis, *sclérose en plaques disseminées,* was
escribed pathologically by Cruveilhier and Carswell (1838); Frerichs,
in 1849, made the first diagnosis, and Charcot (and Vulpian), in 1863–64,
gave a description of the typical case.

Etiology.—In fully 50 per cent. of the cases no cause is found, and little is known of its etiology. It occurs equally in either sex and usually commences between the twentieth and thirtieth years. Cases resembling multiple sclerosis (pseudosclerosis) occurring after the fortieth year are thought to be arteriosclerotic. Two main factors are thought to exist: (*a*) *Acute infections*, as typhoid, pneumonia, measles, smallpox, etc. Marie adopted this theory, but Oppenheim finds that many cases can be traced back to infancy, a view vigorously contested by Strümpell as is (*b*) the idea that chronic intoxications with alcohol, lead, etc., are causal.

Pathology.—Macroscopically, the *sclerotic foci* are usually seen with ease as irregular grayish-red areas as large as a pea or walnut; they are multiple (numbering even over 100), are distributed at random and occur largely *in the white substance*, although recent studies by Oppenheim and Taylor have revealed equal involvement of the gray matter. In most cases their distribution is *cerebrospinal*, in rarer cases in the cord alone, and most rarely in the brain only; the brain cortex is less frequently involved than the centrum ovale, central ganglia, corpus callosum, and the anterior under surface of the pons and the crus. The medulla is quite often and the cerebellum rarely involved. The white matter in the cord is more frequently affected than in the brain. *On section*, the focus is a trifle sunken and transparent, is fairly well demarked, firm save in the freshest foci, and on hardening in Müller's solution, appears light green, while the normal tissue is a darker green color. Microscopically, the medullary sheaths in the white substance are thinner or absent and though the axis cylinders are somewhat wasted and at times varicose, their remarkable persistence, first noticed by Charcot, causes the incomplete character of the symptoms and the usual absence of ascending and descending degenerations. The ganglion cells suffer some atrophy and pigmentation, though in general they resist the sclerosis fairly well. The foci stain deeply with carmine, are not sharply demarked from the healthy tissue and show corpora amylacea, granule-bearing cells, increase of the fine meshed neuroglia fibrils, nuclear proliferation and sometimes Deiter's cells. In contrast with the system-degenerations, the primary change is interstitial with secondary changes in the nervous tissue. Some pathologists, as Ribbert, see significant changes in the bloodvessels of early cases, as arterial thrombosis, followed by exudation, and finally neurogliar proliferation; this theory is vigorously opposed by Strümpell. The cerebral nerves suffer often, especially the optic nerve, in which proliferation of connective tissue is followed by loss of the medullary sheaths, while the axis cylinders are unaffected. The nuclei, roots and nerve trunks of the third, eighth, seventh and other cranial nerves may suffer similar alteration. The spinal nerve roots are not infrequently involved; the peripheral nerves have not been systematically studied.

Symptoms.—The clinical picture is so variable that a definite description cannot be made to cover all cases. Charcot's description uded the intention tremor, the nystagmus, scanning speech, motor kness and rigidity, and apoplectic insults, although he recognized pical forms (*formes frustes*).

. **Motor Symptoms.**—These are the most important. (*a*) *Mus-* *r weakness* is very common. Paresis is more frequent than paraly- and the gross muscular power is lessened, though rarely abolished, e some axis cylinders remain intact. The movements are slow fatigue rapidly follows their repetition; this statement applies to ated rather than to synergistic movements. The paresis is most arent in the legs, there is usually some in the arms, and frequently e in the musculature of the eyes, head and speech. It sometimes s not develop until late in the disease, may at no time be conspicu- and is commonly associated with increased reflexes and spasticity. gait is spastic, scuffling, and the feet seem to stick to the ground; his is sometimes added a cerebellar, atactic or paretic element. scular atrophy is rare but may result from an adventitious focus he anterior horn. The electrical reactions are practically normal. *Muscular rigidity* is the most frequent finding, is referred to ase of the lateral columns and is most developed in the legs. It reases with successive movements; for example, each step becomes re stiff in walking. It is sometimes slight in degree, and amounts y to a sense of stiffness; in other cases the spasticity is so extreme, the muscles so hard that the limb cannot be moved even with at effort. Contractures develop, which usually affect the adductors extensors first and the flexors later, and draw the heels up to the tocks. Contractures often affect the muscles of the neck. (*c*) *mor* is one of the most striking phases of the disease, being present 5 per cent. of cases. It is caused as is the paresis, by the loss of the lullary sheaths (Charcot) which allows diffusion of nerve impulses, foci in the pons or thalamus (Erb), or by lack of coördination ween antagonistic muscles (Strümpell, v. Leyden); the axis cylinders ain intact. There is no tremor during rest, but it appears when ntary efforts are attempted, whence the name *intention tremor;* levelops as the patient reaches for an object, which is grasped y after jerking and unsuccessful efforts. The hand carrying a ss of water to the mouth shakes so much that all the water may be lled by the rhythmic tremor, which possesses a wide range, and urs five or six times to the second. It is apparent in the head when patient sits, because the muscles of the neck are in constant action upport the head; it disappears in the recumbent posture. Similar mor occurs in the face, larynx, trunk or legs on changing posture, as ng; in the fingers, it produces handwriting which becomes more atic with each word written; and in the thoracic muscles, it causes a cies of cog-wheel respiration. (*d*) *Nystagmus* occurs in 50 per cent. cases, and is elicited by having the patient follow with his eyes index finger when moved to one side of his field of vision, his head aining stationary. His eyes show short, jerking lateral movements. s most common on lateral or vertical movements. Spontaneous nystagmus is rarer, as nystagmus on attempting to focus the eyes. must be remembered that it may occur without organic disease, as general weakness or convalescence. There is no satisfactory explanation; it is considered a tremor or an incoördination. (*e*) *The speech scanning* (54 per cent.) *syllabic* or *staccato*; it is slow, monotonous, unmodulated and sometimes explosive, especially for the letters c, p, and d. The speech muscles become fatigued easily and the end of sentence is often elided; the sounds are often nasal, interrupted by yawning inspiration, or by expiration at the beginning of each syllable. Leube observed a tremor of the vocal cords, which oscillated during their movement. The cause of the peculiar speech is not clear; it may be due to tremor, or ataxia from foci in the pons or medulla. (*f*) *Paresis of the eye muscles* (in 17 per cent. Uhthoff) affects the third or sixth nerves, convergence or associated movements. About 50 per cent. is nuclear. In rare cases the paralysis is progressive. Transient diplopia, unequal pupils, rhythmic spontaneous widening and narrowing of the pupils (hippus) and strabismus are less common. (*g*) *Apoplectiform seizures* (in 20 per cent.) develop without prodromes, and are marked by partial, rarely by complete coma, increased pulse, high temperature, aphasia and hemiplegia, which is usually fugitive and flaccid, rarely enduring or spastic. They may recur. In most cases negative pathological findings exist, but Leube and Leyden refer them to acute encephalomyelitis. Though usually hemiplegic, they may exceptionally produce paraplegia, hemianæsthesia or ataxia, or monoplegia. Less frequently, unilateral convulsions occur.

2. **Sensory Symptoms.**—These are less frequent (30 per cent.) and important, because the sensory fibers are more resistant to disease, although they are rarely wholly absent. Freud found sensory symptoms in 80 per cent. of his cases. *Subjectively*, dull pains, formication, vertigo, the importance of which is "frequently overestimated," and paroxysmal moderate headache are frequent, and lightning pains, crises, girdle sensation, trigeminal neuralgia, palpitation and dyspnœa are very uncommon. *Objectively*, any species of sensation may be involved, especially in distal parts of the extremities, where it is usually transient. *Ataxia* is usually referred to disease of the posterior columns or sensory tracts of the pons or medulla; it is marked in 50 per cent. of cases and may produce a typical tumbling cerebellar gait.

3. **Special Senses.**—The optic nerve is affected in 45 per cent., usually as a pallid partial atrophy involving its temporal side, and less often as neuritis or complete atrophy with total blindness. The change is more conspicuous pathologically than clinically. Optic neuritis is transient and runs a rapid course; in the majority (60 per cent.) of cases, it produces central scotomata, although at times it narrows the field of vision. It may be the first sign of the disease. Hearing and taste may be affected.

4. **Psychical Symptoms.**—Slight mental impairment is common; the patient is indifferent and singularly complacent; the memory is weak; psychoses are rare. Compulsory or forced laughing and weep-

are partly without motive and partly emotional; they are sometimes rred to mental alteration but probably should be classed among the or symptoms and be referred like the analogous compulsory yawn- or swallowing to lesions in the medulla, pons or crus.

. **Bulbar Symptoms.**—These may, in rare cases, resemble the ptoms of bulbar palsy, as dysphagia, anarthria, and tongue atrophy.

. **Reflexes.**—The tendon reflexes are increased, most markedly in legs. The patellars are exaggerated, there is ankle clonus, and ling of the sole produces dorsal flexion instead of the normal plantar ion of the foot (Babinsky's sign) showing disease of the pyramidal ts. The skin reflexes are usually said to be normal, but Strümpell nd the abdominal reflexes absent in 70 per cent. of his cases. The illary reflexes are usually normal or but slightly slow.

. **Sphincters.**—Lasting or severe symptoms in the bladder or rectum uncommon, though slight disturbance and weakness occur in most es. The bladder is often hypertonic and irritation is frequent. The ual power is variable.

. **Trophic Symptoms.**—These are rare; they consist of skin erup- s, œdema, low temperature, sweating, chronic arthritis and ery- melalgia.

ourse and Prognosis.—The onset is usually gradual; the spinal ptoms are most prominent and in most cases the weakness in the appears first and for a long time is hard to interpret; it occurs out pronounced sensory symptoms, bladder disorder or atrophy. etimes the onset is acute, with loss of consciousness, transient iplegia, vertigo, headache, eye symptoms, vomiting and optic ritis. In the average case the leg paralysis is followed by scanning ech, nystagmus, and tremor, which covers a chronic course averag- three to six, or in some cases even more than twenty years. A id course, as twenty-three months, may occur in the bulbar form. e symptoms remit and recur. Progression to a fatal ending is vitable. Charcot distinguished *three stages* (*a*) the period from the elopment of the first symptoms to the *tremor and paresis*, (*b*) the iod in which the patient is *bed-ridden*, and (*c*) the *terminal stage* with mentia, decubitus, sphincter paralysis, cystitis and septicopyæmia.

iagnosis.—The typical case is unmistakable, because of the cardi- symptoms, which consist of (1) intention tremor; (2) spastic esis; (3) nystagmus; (4) scanning speech; (5) ataxia; (6) reased reflexes; (7) optic atrophy; (8) the slow, uneven course h remissions and recurrences; (9) the variability of the symptoms;) apoplectic insults; and (11) moderate involvement of the mental- sensation, bladder and rectum.

ifferentiation.—Transient hemiplegia may suggest hemorrhage, hilitic thrombosis, embolism or tumor (*q. v.*). If these can be luded, multiple sclerosis or paretic dementia must be differentiated v.). *Paralysis agitans* is usually but rather unnecessarily differen- ed; the absence of the cardinal signs of sclerosis multiplex and the sence of a constant fine tremor of the hands and fingers (not of the large joints, and much less in the head) which is not increased by movement, its persistence during rest, the mask-like facies, festinating speech, peculiar attitude, gait, propulsion, retropulsion, muscular rigidity without increased reflexes, and the advanced age are clear and characteristic points.

Multiple Sclerosis.——*vs.*	——Syphilis.
Occurs in the medullary substance.	Occurs in the periphery of the cord (meningomyelitis and root symptoms).
A focal disease.	Diffuse.
Nystagmus, scanning speech, tremor.	More frequent mental obscuration, insults, hemiplegia, etc.; speech may be disturbed or slow in pons lesions but is not scanning.
Transitory amaurosis, hemianopsia, complete optic atrophy, choked disk or neuritis are very rare.	Amaurosis, etc., are frequent.
Partial optic atrophy occurs.	There is more disturbance of vision
Mydriasis 1 per cent., myosis 4 per cent. Argyll-Robertson pupil practically never present.	Mydriasis is very common.
	Cortical symptoms may be present; rectum and bladder more frequently involved; results may be obtained in early stages from antisyphilitic remedies.

Hysteria more than any other disease simulates early multiple sclerosis, the two affections often occur together, and in both the symptoms are changeable. In hysteria sensory symptoms are much more prominent, there is often limitation of the visual fields, monocular diplopia, contractures, and convulsions, but there is no optic atrophy, nystagmus is extremely rare (Sougues) as is the scanning speech; and intention tremor may occur, but is not so wide. The movements in hysteria are often accompanied by contraction of the antagonist muscles, which is a most valuable diagnostic aid. In extremely rare cases, described first by Westphal as *pseudosclerosis*, similar symptoms prevail though the autopsy shows nothing; the tremor affects the arms chiefly; there is no ataxia or nystagmus; the facies is somewhat rigid and outbursts of anger are frequent; syphilis is a possible cause. Birth *palsies* may be accompanied by jerky movements, incoördination of the legs and even scanning speech, but the history of very early onset is conclusive.

Strümpell groups the *atypical forms* as (*a*) the latent group; (*b*) those resembling chronic myelitis, or (*c*) pure spinal spastic paralysis, maybe resembling amyotrophic lateral sclerosis if the anterior horns are involved, or chronic bulbar palsy if the medulla is affected; optic atrophy in such cases indicates multiple sclerosis. (*d*) Cases resembling tabes with ataxia, and pains, which are probably cases of combined system disease (*v. i.*); (*e*) the hemiplegic form; and (*f*) the psychical form.

Treatment.—*Treatment* is futile. Drugs as silver and arsenic are inert, and hydro- and electro-therapeutic measures are useless. The patient should live in a warm climate during the winter months.

(H). SYRINGOMYELIA.

The term refers to cavities in the cord, and was first employed by Ollivier (1824). The affection was first described by Morgagni. Syringomyelia must be distinguished from hydromyelus (hydrorrhachis interna), which is a dilatation of the central canal due to developmental anomalies, possibly to stasis of cerebrospinal fluid resulting from pressure; it is usually an accidental finding at necropsy, or if symptoms are present they are those of syringomyelia.

Etiology.—Sixty-three per cent. of cases occur between the tenth and thirtieth years and 70 per cent. of them in males. Other factors are wholly uncertain, as trauma, manual occupations, or cold.

Pathology and Pathogenesis.—**Gross Pathology.**—A cavity may be seen from without. The cord is often lax, sometimes fluctuating or flat. The cavity is usually localized but may sometimes extend from the medulla to the filum terminale; it is most often found in the lower cervical or upper dorsal regions, less frequently in the dorso-lumbar segments or in the lower brain; in size it ranges from a narrow slit to a cavity as wide as the cord itself; it is not absolutely symmetrical and may be unilateral; it may communicate with the central canal, may be multilocular from septa or strands of tissue, and diverticula may be found. Its wall is smooth, sometimes pigmented from old hemorrhages. Sometimes the cord is normal, but it usually suffers thickening, and sclerosis develops from the ependyma, posterior commissure or posterior horns, in the form of hard or soft, tumor-like, somewhat asymmetrical neurogliar proliferations, especially in the cervical region; these later break down and form secondary cavities. The posterior horns and columns are often affected, the anterior horns and columns less frequently and the lateral columns very rarely. Hemorrhage into the gliomatous tissue or the cavity may result from rupture of the new formed bloodvessels.

Minute Pathology.—The essential finding is overgrowth of the neuroglia, which in part breaks down and forms cavities. This hyperplasia is most probably due to some congenital anomaly, which also explains its frequent association with dilatation of the central canal (hydromyelus). The neurogliar overgrowth is rich in fibers, whereas the ordinary glioma (gliomatosis) especially abounds in cells; syringomyelia and gliomatosis may co-exist. The proliferation is seemingly embryonic in character, contains some large sharply nucleated cells, here and there a few epithelial cells, is firm as a whole, but somewhat softer towards the posterior horns, is often hyaline nearest the cavity from compression, and abounds in bloodvessels, which are often hard and hyaline. The cavity is usually lined with cylindrical epithelium like that of the central canal, sometimes only with neuroglia; its contents resembles cerebrospinal fluid, but is occasionally blood tinged or less often gelatinous, mucoid or milky. *Degenerations* occur in parts actually involved and at times in distant parts as in the medulla. The nerve fibers are not much involved, although secondary degeneration may occur in the posterior and less frequently in the lateral columns.

The exact *pathogenesis* is obscure. Congenital hydromyelus (with primary neurogliar hyperplasia and secondary cavity formation), inflammation and hemorrhage are not causal factors.

Symptoms.—There are *three cardinal symptoms.* (1) *Partial or dissociated anæsthesia* occurs in the typical case, in which the sensation of touch, pressure and locality is preserved while heat or cold and pain are not recognized as such but as simple pressure. *Thermo-anæsthesia* usually concerns perception of both heat and cold, sometimes of only one of them, or one in one place and the other in another; *analgesia* is frequently preceded by hyperalgesia; it concerns the superficial or deeper structures and usually corresponds with the thermo-anæsthesia which however may exist alone. Thermo-anæsthesia and analgesia usually begin in the hands and fingers and are later found on the upper trunk; sometimes the entire trunk, the legs, mucous membranes, and face may be affected, depending on the location of the cavity. The patient may suffer burns or injuries in these parts without pain. Dissociated anæsthesia is most probably caused by disturbance of conduction of temperature and pain in the gray substance of the cord, which is frequently invaded by neurogliar proliferation and the cavity formation; tactile sensation traverses the white columns. In some cases the partial anæsthesia is unilateral. Anæsthesia may involve all variaties of sensation. Subjective sensory disturbances include paræsthesia and burning, tabes-like pain in the neck, arms or trunk, and sometimes in the joints, spine or legs.

2. *Muscular atrophy* is due to involvement of the anterior horn and affects the arms mostly, because the disease is largely in the lower cervical and upper dorsal cord. It is slow in onset, more marked than the attendant flaccid paresis, degenerative, and often observed with fibrillary muscular contractions and increased myotatic irritability. It usually involves the small muscles of the hand as the interossei, produces flattening of the thenar and hypothenar eminences and the claw-like hand as seen in the Duchenne-Aran type. The radial supply is first affected, then the ulnar and median supply. Sometimes the scapulohumeral type is observed, especially with bulbar symptoms. The muscles of the trunk, and much less often of the legs, may be affected. From cervical compression of the pyramidal tracts, paraplegia results, with increased reflexes and spasticity, in 12 per cent. of

s. Other motor symptoms occasionally occur, as tremor, contures, spasms, opisthotonos or choreiform movements. The gait ormal, spastic and paretic or sometimes of the cerebellar type. iberg's sign and ataxia result from involvement of the posterior mns or coincident tabes.

Trophic, secretory, or vasomotor symptoms may occur. Trophic ptoms are quite frequent. (*a*) Phlegmons and *felons* develop. icularly on the fingers. They are painless, chronic, often recurrent, lead to necrosis of the bone, ankylosis of the joints, and to con:able deformity or mutilation. (*b*) The *joints* suffer in over 10 :ent., especially in males (75 per cent.), and this is probably due iss of articular sensation (whereby the joints are used inappro:ely) or to lack of the nutritional reflex. These arthropathies are of hypertrophic form, with thickening and bony formation, like ritis deformans or of the atrophic form with relaxation of ligaments ail joint. There is little exudation, no fever, no pain. On the e, they are similar to the tabetic joints but are more chronic and often involve the upper extremities (shoulder 32 per cent., elbow er cent., hand 16 per cent., hip 8 per cent., ankle 8 per cent., knee r cent.). (*c*) *Painless bone necrosis* and spontaneous fractures may r late in the disease; moderate scoliosis is frequent, usually ined to the dorsal spine, sometimes with kyphosis. It is probably to trophic vertebral changes or possibly to atrophy of the back cles; perforating ulcer, thick, friable nails and clubbed fingers are common. Morvan (1883) described an affection as *paresie analjue avec panaris*, endemic in Brittany, which is clearly syringomy- "Morvan's disease" is marked by trophic disorders, excessive or :ient secretion of sweat, œdema especially on the dorsum of the ls, erythema, urticaria, more rarely local asphyxia, and herpes ɔr.

here may be *other symptoms*. (*a*) The *reflexes* of the skin are lly normal, although the abdominal, cremasteric and plantar xes are sometimes increased; the tendon jerks are variable, being decreased in the upper and increased in the lower extremities. The *sphincters* are not involved, or may be only late in the disease. ntion of urine and constipation are more frequent than incon-ıce. (*c*) *Bulbar or pontine symptoms* are not infrequent, are usually ateral, and appear in the later stages. They include facial hemiphy or paresis, paresis of the tongue with atrophy, partial dysphagia, sis of the palate (nasal speech), paralysis of the vocal cords, of the rnal branches of the spinal accessory nerve (trapezius paralysis) and minus (sensory symptoms), eye paralyses (abducens), unequal ils, nystagmus, tinnitus, salivation, apoplectiform attacks, polyuria, osuria and vagus symptoms (disturbed pulse or respiration, vomitand singultus). *Complications* include hysteria, paretic dementia, s, pachymeningitis, Graves' disease, and epilepsy.

iagnosis.—An almost positive diagnosis can be made from the e combined cardinal symptoms; (1) the dissociated anæsthesia (thermo-anæsthesia and analgesia with practically normal tactile sensation) is the earliest, most constant and widely distributed sign. Grasset has collected cases of twenty or more nervous diseases (functional, traumatic, neoplastic, specific) in which the dissociation of syringomyelia has been found; but it remains highly distinctive in conjunction with (2) *muscular atrophy* of the *upper extremities* with paresis and (3) *trophic disorders, felons, arthopathies,* and scoliosis; to which three cardinal manifestations may be added the spastic paresis of the legs and bladder symptoms. Besides the classical form, various varieties are described by Schlesinger; (*a*) the latent form, (*b*) the motor form, simulating amyotrophic lateral sclerosis, (*c*) the sensory type, possibly resembling hysteria, (*d*) the trophic and (*e*) the tabetic types.

Differentiation.—*Progressive spinal muscular atrophy* and *amyotrophic lateral sclerosis* may resemble syringomyelia with poorly developed sensory symptoms (Clarke and Gull, 1862) but they possess very few sensory signs, no anæsthesia, no trophic disturbance and bulbar symptoms are bilateral. *Juvenile muscular dystrophy* may be simulated in the scapulohumeral type of syringomyelia. *Peripheral neuritis* rarely produces dissociated anæthesia but all variations of sensation are disturbed; the distribution of the paralysis is peripheral, not spinal or segmental, and there is the reaction of degeneration; neuritis of the upper brachial plexus (Duchenne-Erb paralysis) produces paralysis of the shoulder, arm and supinator longus, a combination not found in syringomyelia. *Focal disease* of the anterior and posterior gray substance, as multiple sclerosis, hemorrhage, or myelitis may simulate syringomyelia, but their course is less chronic and there is less trophic disorder. The signs of irritation produced by *tumor* of the cord are lacking. Located in the dorsolumbar region, syringomyelia may closely simulate *tabes*, from the lancinating pains, crises and ataxia, but the Argyll-Robertson pupil is lacking and the sensory impairment is typical. The anæsthetic form of *leprosy* has caused difficulty but its anæsthesia is peripheral, not spinal, in type; in the lesions of the skin, ulcers, and nasal secretions, the Henssen bacillus is often found; if there are no lesions in the skin, thickness and tenderness of the nerves indicate leprosy.

Course and Prognosis.—The onset is very gradual and the course chronically progressive, for years or even for decades. When recognized, the affection is well advanced and recovery is impossible. The first symptoms are pain and dissociated anæsthesia, followed by atrophy and paresis, with trophic changes in the advanced course. Death occurs from intercurrent disease, cystopyelitis, septicæmia and bulbar complications.

Treatment.—Treatment is unavailing, being confined to hygienic and symptomatic measures. The danger of self injury, as a result of the peculiar anæsthesia, should be borne in mind.

I. SYSTEM DISEASES.

These constitute a small group of very important diseases and involve *structures or systems having a common function,* thus differing from such indiscriminate or random disease as myelitis or multiple sclerosis. The essential change is a *primary slow degeneration of the nerve elements,* followed by *secondary proliferation of connective tissue* (sclerosis). The pathogenesis of these degenerations is not clear, although *hereditary weakness* causing nutritional failure at a certain age (the familial form), and the *selective action of toxins* in the blood acting on certain nervous structures, have been thought causes.

System Disease of the Sensory Tract.

The sensory type of system disease is locomotor ataxia in which the peripheral sensory neurone is affected, in the posterior roots and columns, while the motor types are spastic paraplegia, involving the upper motor neurone (pyramidal tracts), and muscular atrophy due to lesions in the lower neurone (anterior horns and roots).

Tabes Dorsalis, Locomotor Ataxia (Duchenne).

Definition.—A disease characterized (*a*) etiologically, by syphilis; (*b*) pathologically by lesions in the posterior spinal roots and posterior columns, in the cutaneous and muscular sensory and also the cerebral nerves; and (*c*) clinically by incoördination (ataxia), sensory symptoms (pains and defective sensation), loss of myotatic irritability (of the tendon reflexes), loss of the pupillary reflex to light and trophic disturbances.

It is the best known and most frequent chronic disease of the cord. Horn in 1827 partly described the affection, although Todd in 1847 gave the first accurate account, eliminated paralysis, and found the posterior columns diseased at autopsy. Stanley first (1840) referred the affection to the posterior columns and peripheral sensory nerves. Türck first examined the tissues microscopically and Romberg (1851), and especially Duchenne (1858), popularized the disease; it is still known in France as Duchenne's disease.

Etiology.—*Syphilis* is the essential causal factor (Fournier and Erb) in 75 per cent. (Gowers), 90 per cent. (Erb and Strümpell), or 100 per cent. (Möbius). In about 10 per cent. of cases the syphilitic history and stigmata are lacking, whence it is sometimes attributed to other causes, as trauma, heredity, concussion, exposure, toxins of soft chancres (Hitzig and Buzzard), and ergotism (Tüczek); but it is most probable that these are only apparent or incidental factors and that syphilis is the sole cause. The following etiological data of tabes are practically those of syphilis; *age,* 50 per cent. in the thirtieth to fortieth year; 25 per cent. in the fortieth to fiftieth year; less than 25 per cent. in the twentieth to thirtieth year (Gowers); cases may occur in persons over fifty, under twenty, or even ten years of age, the latter of which are probably due to hereditary syphilis. Forty-seven juvenile cases were collected by Hirtz (1905); of these, the parents in thirteen instances had syphilis, tabes or dementia. *Sex*: as in syphilis and paretic dementia, ten cases occur in males to one in females. Syphilis is more difficult to diagnosticate in women and more often runs a latent course. Like syphilis, tabes is sometimes familial; Nonne (1904) saw tabes in a mother and two daughters. It prevails largely in cities, and among the higher classes ("syphilization and civilization"). It is uncommon in the negro though Hecht has seen cases. The reason that syphilis is not considered causal by some is the fact that tabes does not occur during its florid or even its tertiary stage; tabes is meta- or parasyphilitic, a degenerative lesion occurring on an average six to twelve years after the chancre or even later. Dalous (1904) collected 21 cases with active syphilis.

Pathology.—The *gross changes* are usually visible to the unaided eye; the posterior columns are smaller, outwardly flattened instead of rounded, and appear gray from loss of the medullary sheaths and translucent from primary loss of nerve fibers and secondary increase of connective tissue. The pia may be secondarily opaque and thickened. The degenerated areas are somewhat sunken on section. The posterior horns and roots are always somewhat atrophied and gray or grayish-red in color. As to *minute changes*, the toxins (Strümpell) bring about a very *slow degeneration of the peripheral sensory neurone*, its fibers, and the processes of the spinal ganglia cells, which themselves suffer much less and later obvious changes than their processes. These processes run in two directions: to the periphery and to the posterior roots and posterior columns. (*a*) The *peripheral spinal sensory nerves* are often degenerated (Westphal, Pierret, Déjérine), less in large trunks (sciatic or crural nerves) than in the peripheral filaments of the skin, joints and muscles which are important in the sensory, trophic and atactic symptoms of tabes; their white substance wastes and then their axis cylinders. The trophic centres for these fibers are largely in the root-ganglion but there is some evidence that there are also some peripheral trophic cells. (*b*) The *fibers running from the ganglia to the posterior roots* are more early, regularly and obviously changed. In the cord the degenerated tracts stain deeply with carmine and include only structures derived from the posterior roots and their collaterals, all others escaping involvement. The posterior root fibers run (i) to the posterior columns, whence there occurs, low down in the cord, degeneration in Burdach's and then higher up in Goll's columns, into which the root fibers are pushed as new and higher root fibers enter the cord. The location of the degenerated fibers therefore differs at successive levels and the degeneration is an *ascending* one. In the *lumbar cord* the posterior columns are most intensively and extensively diseased, especially in their middle and posterior parts, nearest the osterior roots and surface of the cord. Early and marked degenertion is found in the fine fibers of Lissauer's zone which lies just back f the posterior horn. The anterior part of the post. ext. column and the entral part of the post. int. column are slightly, if at all, diseased as ey are not derived from the post. ganglia. In the *dorsal cord* the post. olumns are degenerated save in the post. ext. part and in the ant. part. n the *cervical cord* the post. int. columns are diseased (representing the ot fibers of the lumbodorsal cord) and also some areas in the post. ext. lumn where degenerated root fibers have just entered. There is me pathological as well as clinical variation in different cases; Marie lds that one set of sensory fibers after another is involved, and that egeneration develops by segments. Tabetic changes are usually most eveloped in the lower cord, but in the rare "cervical tabes" the upper rd suffers severely and perhaps exclusively. (ii) From the post. ots a second series of fibers run to Clarke's column. They and their llaterals are degenerated but the cells of Clark's column (and their cending fibers to the cerebellum) usually remain intact. (iii) The post. rns degenerate, because they contain fine root fibers. (iv) The reflex llaterals to the ant. horns also degenerate.

As already stated, pronounced or early atrophy in the cells of the st. ganglia is rare. Degenerative changes may involve other analous neurones, as the *auditory*, trigeminus, *optic*, visceral, sympathetic ocular nerves, or less often the peripheral motor nerves, the pyraidal tracts, the muscles themselves, the medulla and the brain cortex. old cases, corpora amylacea are found.

The exact *pathogenesis* is still disputed: (i) Obersteiner believes the itial changes are in the pia through which the dorsal nerve roots pass; i) Marie contends that there is a lymphangitis of the post. columns; ii) Edinger believes the toxins (admittedly those of syphilis) attack ose nervous structures most in use, and therefore most easily fatigued; ese are the sensory tracts of the lumbar cord, pupil, bladder and testine.

Symptoms.—The symptoms vary chiefly as to their intensity, uration and order of succession. In general *three stages* are described. . The *initial*, neuralgic or pre-atactic stage, lasting sometimes for years usually 6 to 8, or even 10 to 20). The earliest symptoms are usually ensory and irritative, as *pains* which are either lancinating or rheunatic, perhaps neuralgic or like migrane or paræsthesia. The *knee rk* is *lost* in nearly all cases, usually very early and the *pupil reacts* to ccommodation but *not to light*. 2. These symptoms usually exist years efore the ataxy of the second stage appears. Much less often optic trophy, sexual, sphincter, ocular, articular or visceral symptoms first ttract attention. The *atactic stage* may last many years; muscular ovements become incoördinate, especially in the legs, but sometimes, ough later, in the arms. The tabetic gait develops, the patient sways hen his eyes are closed (Romberg's sign), sensation is obtunded, pecially the muscular sense, urinary and sexual disorders develop nd in some cases diplopia. The gross muscular power is unimpaired.

Trophic, joint, bone and skin lesions and visceral disturbances (crises) occur. 3. The *terminal stage,* improperly called paralytic, is much like that of other chronic affections of the cord. The patient is bed-ridden, helpless from extreme ataxia and rarely from motor weakness, and bedsores, lesions in the medulla, and cystitis lead to a fatal issue.

SPECIAL SYMPTOMS IN DETAIL.—1. Of the *motor symptoms,* (*a*) *ataxia* (incoördination) is of such importance that the disease is named *locomotor ataxia.* It is present in 75 to 88 per cent. of cases but may develop slowly, only after years or perhaps not at all; it is less common at the present time than formerly (Brissaud); it occured in barely 10 per cent. of Babinsky's cases, who explains this decrease by his use of mercury. Ataxia is a disturbance of harmonic efficient muscular contraction and arises not only from tabes but also from lesions in the upper sensory neurones of the cord, medulla, cerebellum, pons and cerebrum. The cause of tabetic ataxia is probably disease of the afferent muscular fibers (muscular anæsthesia, Reynolds 1855), intensified by sensory impairment, disturbance in the reflex collaterals and sometimes by atrophy of the optic nerve. Duchenne first made the valuable distinction between ataxia and loss of muscular power which is preserved until late in the disease. Ataxia is frequently first observed by the patient when he walks in the dark or closes his eyes, as in washing the face. When he closes his eyes or looks well upward or when the heels and toes are brought closely together, the body sways (*Romberg's sign*) and he perhaps falls, because vision is one of the sensory factors in equilibration. Later, incoördination occurs even when the eyes are open, and the patient can walk only with the support of a cane. It is often increased by removing the shoes, which support the feet. The ataxia is marked when the patient attempts to "about face," to walk backward (Althaus), to stand on his tiptoes (Rosenbach) or to stand with the knees half flexed (Brissaud). In women marked ataxia is less frequent than simple awkwardness. Ataxia is also seen when the patient, with the eyes closed, attempts to place his heel on the opposite knee, or perform with one limb those movements made by the physician in another limb. The gait is atactic, the feet are lifted too high, are thrown too far forward and outward (*wide-legged gait*) and are brought suddenly and forcibly down in a "*stamping*" fashion. It has been called the "rooster gait." A tumbling cerebellar gait is less frequent, observed later in the disease and referable to involvement of the trunk or hips. Movement in tabetics is characterized by excessive excursion, rapidity, irregularity and uncertainty of execution. The arms usually escape or are involved less markedly and later, save in cervical or brachial tabes. The patient cannot execute finer movements, such as writing or buttoning the clothes. Ataxia is rare in the head, eyes, face and tongue, although in exceptional cases it has been observed in masticatory movements. (*b*) *Muscular power* may be normal even with the extreme ataxia. The muscles are hypotonic, and this loss of tone allows hyperextension and hyperflexion of the joints; it is due to loss of centripetal sensory conduction. There is a feeling of weakness in the legs in two-thirds of the cases. Attacks of transient muscular weakness are frequent (62 to 92 per cent.) as in the eye muscles. Paralysis, though usually transient, may be permanent from disease of the pyramidal tracts, ant. horns or peripheral nerves. (*c*) *Atrophy* is not common, but may affect the arm (type of Duchenne-Aran), the leg (the tabetic club foot, usually due to neuritis), or most rarely the face (hemiatrophy). (*d*) The *electrical reactions* are usually normal. Sometimes muscular contractures occur, due to abnormal sensation. Associated movements, in which one leg moves when its fellow moves, and spontaneous movements, taking place without volition or consciousness of them and due to the slight unnoticed reflexes, may occur.

2. *Sensory symptoms* are prominent. In the majority of cases the disease begins with irritative sensory phenomena. (*a*) Spontaneous *pains* occur in 80 to 90 per cent., are sudden, paroxysmal, lightning-like, lancinating, and often nocturnal. They are usually severe, but may be agonizing, though sometimes only dull, burning or like those of muscular rheumatism; they may be increased by damp and cold. They last from a few hours to a day or two. The legs are the most common seat of pain, then the back, trunk, arms especially in the ulnar distribution, and sometimes the head (occipital nerves) and face (trigeminus, whose ascending branch represents the sensory parts of most of the other cranial nerves); Collet found migraine-like attacks in 40 per cent.; their bilateral occurrence is very suggestive. The pains are more often deep than superficial and are more often irregular than exactly conforming to a nerve's course. The pain in the trunk may assume the "girdle form" (31 per cent.) over a narrow or broad zone. Sometimes the area affected shows local trophic changes, as herpes, ecchymoses or friable hair. (*b*) *Paræsthesiæ* are common, as creeping sensations (formication), "pins and needles" sensations, a sense of heat or especially of cold. They occur in the legs in 66 per cent. of cases. If in the arms, the ulnar distribution is especially involved (17 per cent.). The ulnars at the elbows are often insensitive to pressure (*Biernacki's sign*) and show lessened appreciation of pain, temperature and tactile impressions. In the face a perverted feeling, as of a mask, may be noted ("*Hutchinson's mask*"). (*c*) In the early stages hyperæsthesia (probably from nerve irritation) may be noted. In 86 per cent. of developed cases *sensation is reduced;* it is most often of the spinal or segmental type, though sometimes peripheral. *Tactile sensation* is often disturbed, especially in the legs and soles of the feet. Hypæsthesia may be observed on the buttocks, scrotum, perinæum, penis (the cause of early decreased sexual power) or on the trunk as a zone (80 per cent.) which is hypæsthetic especially to light touch (Hitzig and Lähr). It is not usually found higher than the third rib, although there may be occasional occipital or trigeminal anæsthesia. If the arms are affected, it is mostly in the hands and palms. Sometimes a touch to one member is referred to another (allocheiria). Occasionally a single contact feels like several (polyæsthesia).

associated with analgesia, tabetics are often oversen
The *muscle sense* is disturbed not only as regards the lo
as to active or passive movements of the muscles, especi
A change of angle of one or two degrees in the joints
noted by normal individuals, while in tabetics much wi
are not perceived, especially those in the small distal j
may be moved unnoticed by the patient, perhaps in the
fingers also.

3. *The reflexes* are also involved. (*a*) The skin r
increased early in the disease. Later the plantar and
are somewhat decreased. The abdominal and cremas
remain normal. (*b*) Of the tendon reflexes, the *knee j*
(*Westphal's sign*); this is a most constant finding in t
per cent.) and is due to disease of the afferent nerves
reflex paths in the cord). These reflexes should be exam
the patient sit on the edge of a table, with his eyes closed
cerebral inhibition of the reflexes by having him pull,
direction, the locked fingers (Jendrassik's reinforcement)
reflexes are rarely present save in exceptional cases of
They are sometimes simulated by a reflex movement whi
tapping the patellar tendon but in this case the interval i
the reaction, and the same reaction may follow a sin
The knee jerk has been known to return after a cereb
The *Achilles reflex* is often lost, though less frequently th
(or equally frequently, according to Fränkel). The ten
the arms are sometimes decreased.

4. *The Eyes and Special Senses.*—(*a*) The *pupils contr*
dation but not to light in 85 to 97 per cent. (Argyll-Ro
In testing, the patient must look to a distance, and on
tested at a time lest accommodation be confused with
light. The cause of the Argyll-Robertson pupil is not

the pupillary changes, especially when they are painless wherein they differ from the rheumatic and syphilitic forms; he holds that the majority of all eye paralyses in the adult are tabetic. Diplopia (in 16 per cent.) is common and usually unilateral. The order of frequency of involvement is the sixth, third (lev. palpebrarum), sup. rectus and fourth nerves. When one muscle is paralyzed the others are usually weak. The early form of paralysis is usually transitory; the late form is often permanent. Nuclear degeneration is the most probable cause. (*c*) *Optic atrophy* (10 per cent.) is generally an early symptom. The disk is pale and shrunken, especially on its temporal side and at the edges, which are sharp. The process is always bilateral; central scotoma and actual neuritis are rare. The process is usually progressive and blindness results. When optic atrophy occurs early, ataxia may not appear; this is known as "tabes arrested by blindness." When it develops later it has no effect on the ataxia. (*d*) *Deafness* may develop gradually or suddenly, sometimes with vertigo or Ménière's complex. It may be permanent or transient. Auditory defects are present in 80 per cent. of cases. Atrophy of the auditory and olfactory nerves has been found.

5. *The Sphincters and Genital Sphere.*—(*a*) The *bladder* is frequently affected (45 to 80 per cent.), possibly as the first symptom of the disease. The bladder is slowly or imperfectly evacuated, because its centripetal sensory fibers are diseased. There may be overflow, incontinence, and in the later stages cystitis frequently results from decomposition of the residual urine, followed in turn by pyelitis, nephritis and septicæmia. (*b*) The *sphincter ani* is somewhat weak; there is rarely incontinence. Constipation is the rule. (*c*) The *sexual* appetite and power are reduced (58 to 76 per cent.), often early. Labor is not interfered with and is sometimes painless.

6. *Trophic Disorders.*—The general nutrition sometimes suffers, but local trophic changes are much more common. (*a*) Trophic alterations in the *bones and joints*, described by Cruveilhier and Mitchell, were first thoroughly studied by Charcot, after whom they are named, Charcot's disease. They usually appear late and develop in 2 per cent. of cases. The onset of articular changes is usually gradual, sometimes acute with marked swelling, or may seem to be traumatic. Two forms are observed; first and more frequently the atrophic form, especially in the hip and shoulder, in which the cartilages are eroded, the articular surfaces waste and crepitate, foreign bodies sometimes develop, and the joint becomes too lax, and dislocation of the hip or knee (genu recurvatum) results; second, the hypertrophic form with bony formation, ossification of the ligaments, and rigidity resembling arthritis deformans but more acute, extreme and effusive. The adjacent muscles atrophy—the arthropathic muscular atrophy. The large joints are usually involved, as the knee (45 per cent.), hip (20 per cent.), shoulder (11 per cent.), elbow (5 per cent.), or ankle (4 per cent.), but smaller articulations may be affected, as the tarsus (8 per cent.) or less often the jaw, spine, thumb, etc. The tabetic joint is *painless*. The

cause is the same as in syringomyelia; the anæsthetic joints are moved inappropriately and the nutritional reflex (which normally depends on centripetal influences) is lacking (Marinesco and Serieux). The *tabetic foot* is characterized by thickness and roundness of its inner border and arch, flattening of the sole, decreased motility and pathologically by erosion and atrophy of its bones. It must be distinguished from the tabetic club foot due to muscular atrophy of the calf muscles with contractures. The *bones are friable* and break easily but painlessly, as first described by Wier Mitchell. Fracture is due to rarefying osteitis, which renders the medullary substance more porous, the cortex thinner and the inorganic base less in amount. If a callus develops, it is often exuberant. The tendons sometimes rupture, probably from trophic changes. (*b*) The *perforating ulcer* of the foot (*mal perforant du pied*) is more frequent. It is "punched out" in appearance, and thickening precedes ulceration; it is usually painless, secretes but little pus and is due to nerve degeneration. Similar ulcerations resembling syphilis may occur in the nose, ear, buccal mucosa, palate or uvula. (*c*) In the *skin*, local sweating, ecchymoses, herpes, blebs, alteration of the hair or nails, gangrene and ichthyosis may occur. Decubitus is not common save in the last stages, and it may not develop even after years in bed. There may be painless falling out of the sound teeth with analgesia of the gums or there may be dental caries, referred to changes in the fourth ventricle (Démange).

7. *Of the visceral symptoms*, the most important are the *painful crises* described by Delmarre but first correctly interpreted by Charcot. (*a*) *Gastric crises* occur in 5 per cent. of the cases. There is severe sometimes excruciating epigastric pain, which often radiates into the back, vomiting (with or without nausea) of food, then fluid, finally bile or even some blood; this is often attended by restlessness, epigastric retraction, hiccough, fever and a slow or rapid and irregular pulse. A test meal gives varying results; hyperacidity is less frequent than hypacidity. The urine is alkaline, contains indican, and the chlorides are decreased. The crisis lasts several hours to several days (even 8 or 10) and may greatly reduce the general nutrition; it recurs and is sometimes the first evidence of tabes. The cause of the crisis is hard to explain; it is usually considered nervous, but Pal thinks it is due to increased arterial tension. (*b*) *Laryngeal crises* are next in frequency but are shorter in duration. They are due to spasm of the laryngeal muscles, and are often dangerous. In some instances only a hoarse cough like that of pertussis is noted; in others there is collapse or death (ictus laryngis). Anatomically, the ascending branch of the fifth nerve and the glossopharyngovagus nucleus or its branches are degenerated. (*c*) Other crises are far less common, as *intestinal* crises, which are characterized by colic, diarrhœa and sometimes rectal tenesmus; the author saw one case with severe constipation lasting 12 days, which was immediately relieved by morphine gr. ¼; renal crises with scant urine, transient albuminuria or even hæmaturia, and less often nasal, pharyngeal, hepatic, cardiac, bronchial, vesical,

urethral or vulvovaginal crisesoccur. (*d*) The *pulse* in tabes frequently reaches a rate of 100 or 120. Lachrymation, salivation, polyuria or glycosuria are sometimes noted.

8. *Cerebral symptoms*, other than the eye changes noted above, are not frequent; they are unilateral atrophy of the tongue; paralysis of the vocal cords, especially of the abductors; vagus and accessorius symptoms other than those already noted, such as rapid pulse, palatal weakness or rarely paralysis affecting the trapezius and sternomastoid, which is probably neuritic; and very rarely facial or bulbar paralysis.

Complications.—These include hysteria, neurasthenia, Graves' disease, paralysis agitans; true cerebrospinal syphilis; other meta-yphilitic maladies, as arteriosclerosis, aneurysm (10 to 20 per cent.), valvular disease, arteriosclerotic aortic regurgitation (2 to 9 per cent.) nd especially *dementia paralytica*, which is maintained by some to be cerebral tabes but more correctly held to be an independent although losely correlated affection; myelitis; general or local muscular trophy, particularly the symmetrical form (Déjérine) due to neuritis; erebral thrombosis due to concomitant arterial disease; and apoplec-tiform seizures which occur as in paretic dementia.

Course and Prognosis.—The typical progress of the disease by stages *v. s.*) may be altered. According to Sarbó, pain is the first symp-tom in 68 per cent. while no other single early symptom is the first in more than 3 per cent. The disease in rare cases begins acutely. Not over 50 per cent. are progressive (Gowers). Optic atrophy or insanity may arrest tabes. In the early stages or in rudimentary forms, there is a slight chance that the symptoms may become stationary, but not after ataxia fully develops. Tabes is incurable. Leube mentions an instance of a man who worked hard for forty years after the onset of tabes; tabetics may live for twenty years in the last stages of the disease. Crises, especially the gastric crisis, alcoholism, traumatism, acute onset, intercurrent affections which may be unnoticed because of their painlessness, typhoid, and affections of the bladder and kidney with sepsis are unfavorable prognostics. In 1,960 well-observed cases (out of 4,000) 40 per cent. followed a long course with exacerbations, 30 per cent. were steadily progressive, 19 per cent. halted for three years or more, 6 per cent. ran a severe rapid course, and 5 per cent. almost recovered (Belugou and Faure, 1903).

Diagnosis.—The diagnosis is usually very easily made and is based on the following signs in various combinations:—(*a*) The *Argyll-Robertson pupil*, which according to Möbius is practically indicative of tabes, paretic dementia or rare lesions in the corp. quadrigemina. *b*) The most common and early symptom, *Westphal's sign*, loss of the patellar reflex (which most rarely fail in health, marasmus, old ndividuals or as an hereditary lesion). (*c*) *Lightning pains*, "tabetic neuralgia," girdle sensation, *gastric and other crises*. (*d*) *Sensory changes*, subjective and objective, including bladder disturbance. (*e*) *Optic atrophy*. (*f*) *Painless ocular paralysis*, particularly in the adult and of the nuclear form, producing ptosis, and diplopia. (*g*) *Ataxia*, which usually does not develop early, with *Romberg's sign* and the *tabetic gait*, is practically pathognomonic after exclusion of peripheral neuritis.

In less than 1 per cent. of cases (Déjérine) the disease is located in the cervical region; and in this *cervical tabes*, the sensory changes, pains, and ataxia are most manifest in the arms, or even in the face. The arm reflexes are sometimes abolished, and the patellars may rarely be normal or increased. The characteristic pupils are almost always present, and gastric crises appear early and very frequently. The diagnosis of *atypical tabes* may long remain uncertain, as in cases in which ataxia, Westphal's sign, and the Argyll-Robertson pupil are absent, or in those with unusual symptoms, as marked motor weakness, atrophy, or paralysis.

DIFFERENTIATION.—In the earliest stage, confusion with neurasthenia, hypochondriasis and nosophobia is possible, from common symptoms, as pains, paræsthesia, girdle sensation, and weakness of the legs, but close continued observation usually determines the diagnosis; the tabes of Hippocrates was probably neurasthenia. One of the forms of *peripheral* (*multiple*) neuritis, (*q. v.*), *viz.* acute ataxia, or pseudotabes, may require differentiation. *Cerebrospinal syphilis*, especially of the roots and the membranes, may sometimes cause confusion.

TABES——*vs.*——SYPHILIS.

Gradual onset; very slow progress.	More rapid.
Blindness often complete; marked ophthalmoscopic changes. Hemianopsia not common; rarely central scotoma. Argyll-Robertson pupil.	Blindness rarely total; ophthalmoscopic changes may be few or none, yet with great disturbance of function. Amaurosis, etc., acute or subacute, "in installments"; central scotoma with free periphery. No Argyll-Robertson pupil.
Symptoms bilateral.	Often unilateral.
Oculomotor nerve symptoms may cause the greatest difficulty, since the closest relation (Erb) exists between the two diseases as regards this nerve, yet painless in tabes and nuclear in origin.	Oculomotor symptoms with headache (meningitis and nerve trunk involvement).
Crises and anæsthesia of the fifth nerve, laryngeal paralysis.	Crises, anæsthesia of the fifth nerve, etc., rare. Anæsthesia of the fifth is for all varieties of sensation, while tabetic anæsthesia is for pain.
Sensory nerves most involved.	Motor nerves mostly.
Paralysis very rare.	Paralysis frequent at some period.
Knee jerk absent.	Knee jerk variable, increased.
Ataxia.	Rare.
In syphilis and parasyphilitic diseases (tabes), lumbar puncture shows predominance of mononuclear cells.	Remissions frequent with greater variability and brusqueness than in tabes.

Tabes and *paretic dementia* have a common etiology but in the latter e pyschical alteration is characteristic. For differentiation from *riedreich's ataxia*, see page 1135. In *cerebellar tumor*, the ataxia sually ceases when the eyes are closed, and is not characterized by the cessive excursion or rapidity of tabes; in most cases the patellar flexes are increased, there is no anæsthesia, and signs of tumor are ten present. In *multiple sclerosis* there may in exceptional cases be ises, pains, and sensory disturbance, but the Argyll-Robertson pupil, axia, marked sensory and bladder disturbances are most rare. The flexes are increased, there is nystagmus, scanning speech, intention mor and central scotoma, and functional oculomotor involvement more frequent. *Paraplegias* are not difficult to differentiate, since e motor disturbance is rare and of late occurrence in tabes; the flexes are increased and spastic rigidity is marked, even in atactic raplegia. *Tabetic pain* is often misinterpreted; pain as a rule ggests disease of the vertebræ and membranes rather than of the rd itself, but in vertebral disease or cord tumor the pain is fixed, d is often increased on pressure and movement. Tabes may be nfused (but only on hasty or partial examinations) with *rheumatism*, ercostal or trigeminal neuralgia, and gastric, renal or other *visceral ease*. Bilateral sciatica always suggests tabes, diabetes, or nephritis. rümpell describes, among workers in tobacco, a *nicotine tabes*, in hich pain, pupillary alteration, atactic gait and absence of knee jerks e observed, but which presents a characteristic tremor and increased taneous reflexes in the legs.

Treatment.—Since the disease is incurable, no therapy is especially ficacious.

1. Etiological Treatment.—Antisyphilitic remedies are at first dicated, but their frequent and almost regular failure has led some riters to erroneously deny the causal connection between syphilis and bes. Tabes is not active syphilis; it is a late degenerative paraphilitic affection. Mercury and iodides are therefore advantageous iefly either in early cases or in those in which gummatous changes -exist with tabes. They apparently arrest some cases of optic atrophy. ercury is thought by Erb to be beneficial if it is used carefully; it ay be administered, but should be discontinued at once if untoward mptoms develop.

2. Hygienic Treatment.—Overwork, overexercise, worry, fatigue, uma, exposure, and excess in tobacco, alcohol or in sexual indulgence ould be avoided. Tabetics should not marry. The diet should be gulated, since dyspepsia promotes pain. If possible, the patient ould live in a warm climate and at a moderate altitude.

3. Drug Treatment.—There is no specific remedy. Silver, strych-, and nitroglycerine give uncertain results. Gowers considers senic beneficial.

4. Balneotherapy.—Warm baths are injurious; lukewarm baths e best, but exert no specific influence on the disease. The same is ue of (5) *electrotherapy*, and (6) *massage*.

7. SYMPTOMATIC THERAPY.—(*a*) If *pain* is superficial, sinapisms, chloroform liniments, cocaine (hypodermatically) or the galvano-cautery should be tried; if moderate, acetanilide or cannabis indica, are beneficial, but morphia should be avoided as long as possible, because the habit is readily acquired. Peripheral faradization is often efficacious. Gowers recently described cases in which pain was a salient symptom and existed without ataxia or loss of the patellar reflexes: for this "tabetic neuralgia," differing from the ordinary pains of tabes, he recommends salicylates and aluminium chloride, gr. v-x t. i. d. (*b*) *Crises* resist milder therapy and usually necessitate the use of hypodermics. Laryngeal crises often respond to nitroglycerine hypodermatically, chloroform by inhalation or cocaine locally. Stretching the spine and cord probably relieves pain; the patient sits on the floor with the legs extended; without other movement, the body is carefully pushed forward as far as possible. (*c*) For *vesical* affections, catheterization for residual urine is indicated, but under most rigid antisepsis; belladonna and faradization are often beneficial. Cystitis is treated by lavage. (*d*) Secondary infection should be avoided. Corns should never be cut but always rubbed off.

8. ATAXIA.—This may be relieved by H. G. Fränkel's systematic exercises, "a reëducation of the central nervous system by repeated exercises," which teaches that carefully and persistently executed muscular movements, not forceful movements, are desirable. (The nerve stretching of Langenbuch and the suspension method of Mitchell are useless and even injurious).

II. System Diseases of the Motor Tract.

The centrifugal or motor paths may be diseased (1) in the upper neurone, as in spastic spinal paralysis; (2) in their entirety; this affects both neurones, as amyotrophic lateral sclerosis; (3) in the peripheral neurone, as spinal muscular atrophy or its cerebral analogue, progressive bulbar paralysis; (4) in the peripheral motor nerves, as the neuritic muscular atrophy and finally (5) in the muscles themselves, as the muscular dystrophies. These forms are probably more or less correlated and some of the types blend, but separate descriptions are necessary in order to bring out clearly the classical symptoms and physiology (though the classification of cases into those with and without disease of the spinal gray matter, is more simple).

1. Spastic Spinal Paraplegia.

Lateral sclerosis, *tabes dorsal spasmodique*, was described first by Erb in 1875, and later by Türck and Charcot. Though amyotrophic lateral sclerosis affects both neurones and the muscular atrophies the lower neurone in the cord, nerves or muscles, this malady concerns only the upper motor neurone, especially that part which is in the cord, although it has been traced at times to the cortex.

Etiology and Pathology.—The disease represents a clinical symptom complex rather than a pathological entity. The causal factors are uncertain, as syphilis, or occasionally an hereditary tendency. In 75 per cent. of cases it occurs between the twentieth and fortieth years. It was thought that there was a primary sclerosis of the lateral pyramidal tracts, especially in their lower parts, for which unequivocal confirmation is still lacking, for the cases of Erb, Charcot, Strümpell, Dreschfeld, and Minkowski were not wholly confined to these tracts and there were some changes in other neurones, as in the post. columns.

Symptoms.—There are *three cardinal findings.* (*a*) Motor weakness, which is usually paraplegic; (*b*) muscular rigidity, and (*c*) increased reflexes. (*a*) *Motor weakness* is usually the first sign; it begins slowly and is evidenced by weakness, fatigue or stiffness in the flexors of the hips especially but also of the knees and ankles. Every gradation from paresis to paralysis may be noted in different cases or in different stages. The arms rarely are first affected. Unilateral paresis, as of one arm and one leg, is very exceptional. Strümpell attributes the loss of finer synergistic movements to involvement of the lateral pyramidal tracts; he holds that the coarser movements for groups of muscles are possible, *via* the anterior pyramidal tracts. The muscles are large and well nourished. (*b*) *Muscular rigidity* (hypertonicity) results also from disease of the lat. pyram. tracts, which lessens the inhibition and is proportionate to the loss of power. The muscles resist passive efforts at flexion, especially quick efforts, but yield to slow, gradual flexion and remain in the position given them; this is the "*lead pipe contraction*" of Wier Mitchell. The knees are rigidly extended, even when the patient sits on the edge of the bed; the great toe is strongly flexed dorsally; when one leg is lifted as he lies in bed, the other comes with it; the specially tense adductors prevent abduction of the thighs. The muscles are stiff in the morning; painful spasms may occur during the night; hypertonicity may lead to contractures which are largely active in character and patients sometimes complain of the "tendons getting too short." In active movements rigidity is manifest; the gait is paretic and spastic, the knees are extended and closely apposed, the steps are short, the balls of the feet seem to cleave to the ground, and the feet scuffle. (*c*) *Increase of the reflexes* is also due to lateral pyramidal sclerosis. The *knee jerks* are most *excessive*; ankle clonus and Babinsky's reflex are present; a strong clonic contraction of the quadriceps follows depression of the patella, and the gait may be hopping; in short, this is the so-called "spinal epilepsy," (Brown-Séquard). The *arms* are often normal but may show weakness; there may be rigidity like that of a late hemiplegia, although less in degree, in which the arms hug the chest, the forearm is pronated and partly flexed and the fingers are flexed; there may be clonic spasm of the fingers, and increased tendon and periosteal reflexes. In the *trunk*, there sometimes is weakness in the back and painful contractions of the chest and back muscles with dull aching. The cranial nerves are rarely diseased, although the jaw-jerk may be increased. Sensation, the electrical reactions, the sphincters, muscular nutrition, and the eyes are usually quite normal. If there are sensory symptoms they are rarely objective; the sphincters are usually affected late.

Clinical Forms and Diagnosis.—Diagnosis concerns (*a*) the paresis, (*b*) hypertonicity, (*c*) increase in the reflexes, (*d*) gradual onset, and (*e*) absence of sensory, trophic and other symptoms. Six clinical forms may be described. (1) There is rigidity and spasticity in the legs, arms and perhaps the face, sometimes with compulsory laughing or weeping, as in amyotrophic lateral sclerosis, but without muscular atrophy.

2. The *hereditary or familial form* (Erb, Bernhardt, Strümpell, Spiller) commences in persons between twenty and thirty years of age; the paresis develops late; this form is characterized anatomically by sclerosis of the (congenitally weak) lateral pyramidal tracts, and also by sclerosis of the cerebellar and Goll's columns (this would be more properly classified under combined system disease).

3. In *infantile forms* (see CEREBRAL PARALYSIS IN CHILDREN), the rigidity is less; foot-clonus less common; tonic spasms are rare; athetoid movements, deficient mentality and epileptiform convulsions are common.

4. In the *syphilitic form of Erb*, there is paresis and slight spasticity, plus urinary incontinence, retention, and impotence.

5. There are *forms which are symptomatic of brain disease* (tumor, syphilis, paretic dementia, encephalitis, hydrocephalus), *cord disease* (tumor, myelitis, compression, syringomyelia, multiple sclerosis), and *hysteria* (which may closely simulate spastic paraplegia); hysteria has not the peculiar extensor spasm, loss of power is rarely complete, and ankle clonus is rare, although a spurious half voluntary contraction of the calf muscles may be noted.

6. Westphal, Charcot, Schachnowitch, but especially Goldflam, have described *periodic familial paralysis*, which affects the legs and less often the arms, is flaccid, and results in decrease of the patellar and absence of the plantar reflexes, decreased faradic irritability, sleepiness, but without sensory symptoms; it lasts one to three days. The prognosis is good.

Prognosis.—The disease may be stationary for years and its course is chronic. "It is perhaps the least dangerous to life of any chronic spinal lesion." Recovery or arrest is rare.

Treatment.—Much exercise, fatigue, electrotherapy and strychnia are to be avoided. Massage and warm baths relieve the spasticity. Otherwise the therapy is expectant.

2. Amyotrophic Lateral Sclerosis.

This was often called the *Maladie de Charcot*, because Charcot (1865) with Joffroy first described it.

Etiology.—It occurs mostly between the twenty-fifth and forty-fifth years, more frequently in males, but its exact causes are not known, although cold, trauma, and overexertion are possible factors.

Pathology.—Changes may be found from the cerebral cortex to the muscles themselves, but the name amyotrophic lateral sclerosis, left to us by Charcot, designates the leading changes, which are, *amyotrophy*, muscular wasting, from atrophy of the anterior horns and *sclerosis of the lateral columns*. (*a*) The *lateral pyramidal tracts*, and to a lesser extent the anterior, show symmetrical primary degeneration of the nerve fibers, followed by interstitial overgrowth (secondary sclerosis) which is most marked in the cord, *i. e.*, the lower part of the upper neurone, but extends upward (Charcot, Marie) in some cases to the medulla, pons, crus or even to the very central convolutions, the ganglionic cells of which may be atrophied. Charcot's belief that the disease starts in the pyramidal tracts and is followed by changes in the anterior horns (*v. i.*) is not proven. In some cases similar but unmportant changes may be found in the cerebellar tracts or indeed in the posterior columns. (*b*) The *ganglionic cells of the anterior horn* are degenerated, so that the anterior motor part of the cord is distinctly maller and flatter, particularly in the cervical region. Likewise the nerve fibers from the pyramidal fibers, anastomosing with the proesses of the ant. horn, are degenerated. With these changes in the rophic centre of the second motor neurone, the atrophic changes in the anterior roots, motor nerves and muscles, are easily explained. Certain muscles are wasted to a varying degree, and show degeneration or complete atrophy, as in the purely spinal progressive muscular atrophy, next to be described. (*c*) The *bulbar nuclei*, with their nerves and muscles, show identical changes, for they are the analogues of the ant. horns.

Symptoms.—The symptoms, like the lesions, are strictly motor.

Stage 1 of Charcot.—The disease begins with weakness and atrophy of the *muscles* of the hand, first in one and then in the other; they are most marked in the thenar, hypothenar and interosseal muscles, and are followed by wasting and weakness in the *forearm extensors, deltoid* and *triceps*, while other muscles of the arm and forearm escape or are involved later. There may be some tingling in the parts affected, but sensory symptoms are absent. The decreased power of unwasted muscles is often manifest before they atrophy. Other symptoms of disease of the lower neurone appear, as *fibrillary contractions*; the *reaction of degeneration* is present in those muscles which are wholly atrophied, while in others the response is reduced proportionately to the atrophy; the nerve trunks respond normally. Evidences of involvement of the upper neurone also appear; paralysis often precedes the wasting and accentuates the weakness from ant. horn involvement; there is *increase of the reflexes* (which never occurs in muscular atrophy due to disease of the ant. horns alone), increased biceps, triceps, and periosteal forearm reflexes, and there is often *rigidity and contracture* in which the arm hugs the chest, the forearm is flexed and pronated, and the wrist and fingers are flexed. Sometimes the neck is rigid and even trismus is observed.

Stage 2.—In less than a year the *legs are involved*; indeed they show symptoms during the first stage. The legs are weak, especially in the flexors of the hip, knee and dorsal flexors of the foot. Spastic symptoms prevail. The legs resist passive movement, because of increased muscle tonus and the increased reflexes. The *gait is spastic* and *paretic* (see above, Spastic Spinal Paralysis) and walking finally becomes impossible because of the rigidity and the *twitching tetanic movements* of the *extensors*. The *patellar reflexes* are *exaggerated*; ankle clonus is frequent; when the patient attempts to flex the thigh while the examiner holds the ankle, there is a visible contraction of the tibialis anticus (the tibialis phenomenon); *Babinsky's reflex* is present (dorsal instead of plantar flexion on stroking the sole); there are evidences, in short, of pyramidal disease, which dominate the leg symptoms, because symptoms referable to the lower neurone, as atrophy, are much less frequent, less marked and less early in the legs than in the arms. In exceptional cases the leg symptoms develop first.

Stage 3.—This stage occurs in one or two years with bulbar symptoms, dysphagia, atrophy of the lips and tongue with fibrillation, as in primary bulbar paralysis (*q. v.*). On tapping the jaw, a marked jerk is elicited, analogous to the increased reflexes in the arm and leg. A tonic tension of the facial muscles may occur as in spastic spinal paralysis. Spasmodic laughing or weeping almost always occurs and is due to loss of inhibitory innervation. The mind is clear. Death results from respiratory failure, aspiration pneumonia or intercurrent disease. In rare cases the bulbar symptoms antedate atrophy in the arms and spasticity in the legs.

Course and Prognosis.—In some cases the spinal, in others the atrophic, symptoms develop first and are dominant. The entire course is rapid compared with other diseases of the group, covers one to four years, and is always fatal.

Diagnosis.—The involvement of both motor segments, muscle atrophy with increased reflexes, rigidity, absence usually of sphincter or sensory alteration (except rarely some pains) and the final bulbar symptoms make the disease easy of diagnosis.

The *treatment* is that of spinal muscular atrophy (*v. i.*), but avails little.

3. Progressive Spinal Muscular Atrophy.

Duchenne and Aran (1849–50) gave us the first full clinical description of this disease, while Clarke, Luys, (1860) and then Charcot, discovered changes in the anterior horns.

Etiology.—The ultimate causes are not known. Seventy-five per cent. of cases occur in males, mostly between the twenty-fifth and forty-fifth years. Hereditary factors, which are often observed in the nuscular dystrophies (*v. i.*) are infrequent, although Werding (1891), Ioffman (1894), Bruce, Osler and Thompson have described familial nfantile forms. Hard work, mental distress, syphilis, trauma, acute nfections and acute poliomyelitis are possible factors.

Pathology.—In the cord, especially in the cervical region, (*a*) *the nt. horns are small and flattened*; their ganglion cells are shrunken, leformed, their processes lost; they are wholly wasted in advanced ases. Any interstitial changes are wholly secondary. (*b*) The *nterior roots*, especially the cervical, are somewhat *degenerated*, and ften also the peripheral nerves, although changes in them are most lifficult to distinguish. (*c*) The *muscles are small, stringy* and pale, aving lost their normal redness. Microscopically their individual bers are small, although a few normal or even hypertrophic fibers may e seen. Their transverse striation persists remarkably, and waxy, atty or granular degeneration and longitudinal splitting are not ommon; sometimes the empty sheaths alone are seen. The muscle uclei multiply, round cells may wander in, and there is usually increase f connective tissue and sometimes fatty deposit. Just where the process egins is disputed. No close distinction can be drawn between this isease and amyotrophic lateral sclerosis, although changes in the pper neurone are absent in spinal muscular atrophy. Gowers and . Leyden consider them identical; Kahler and Pick held that both nese diseases and bulbar paralysis are one affection.

Symptoms.—(*a*) The essential symptoms are those of the motor erves, and all others are accessory. The earliest symptom is *muscular trophy* with *weakness* in *the upper extremity* (90 per cent.), especially n the muscles of the (right) *thenar* and *hypothenar eminences*. The bductor and opponens pollicis are first involved, then the flexor brevis nd adductor, the wasting of which brings closer together the metaarpi of the thumb and index finger (the "*ape hand*"). It is finally npossible to appose the thumb to the fingers. The interossei on the orsum waste and leave marked furrows, producing the "claw hand," *nain en griffe*, from contraction of antagonist muscles, the ext. igitorum communis, the tendons of which stand out prominently. The wasting lumbricales make the palm more hollow. Wasting as bove described is known as the Duchenne-Aran type, in which individual muscles" are selectively involved (Charcot). The onset nd progress are always very gradual, and the muscles become lax, oft, thin and flat. The atrophy long exceeds the paresis. The *forearm* nay be next involved, first the extensors, especially those below the int. ondyle and later the supinators and flexors. A deep depression etween the ulna and radius eventually occurs. Later, often after everal years, the *shoulder* muscles, the posterior and middle third of he deltoid, the biceps and brachialis waste, and much later the triceps. ometimes the wasting of the muscles of the shoulder and arm immeditely follows the involvement of the hand, or perhaps the disease egins in the shoulder. The bones of the shoulder stand out clearly, overed only by the skin; these cases are "the living skeletons" seen in museums. The trunk muscles are involved in this order: trapezius in its middle and lower parts (rarely in its upper third, which is the *ultimum moriens* of Duchenne); pectoralis, latissimus dorsi, rhomboidei (causing the shoulder to droop) and extensors of the head, (allowing the head to fall forward). Scoliosis may develop, lordosis is common in involvement of the extensors of the trunk and hip, and the spine stands out distinctly in cases of extreme atrophy. The platysma and face almost always escape. In most instances the respiratory muscles, intercostals or even the diaphragm are involved, and this is a not infrequent source of danger to life. The abdominal muscles are sometimes weakened. In the leg the muscles are affected late and to a lesser degree, as the glutei, quadriceps, and the peronei. In very exceptional cases the disease first develops in them. In the fully developed case there is extreme helplessness in which an added misery in the form of the sister-process, bulbar paralysis, may develop.

(*b*) In the wasted and weak muscles there are usually *fibrillary contractions*; they are involuntary twitchings, tremor- or even wave-like in character, which are sometimes incessant, and again are initiated only by cold, or tapping; they show increased mechanical irritability. They may appear in muscles not yet wasted and are not to be confused with the similar neurasthenic fibrillation. (*c*) The *electrical reaction* depends on the degree of wasting. In the nerves it is normal. In the muscles there is usually only quantitative reduction, and the voltaic excitability remains long after the faradic is lost; in the last stages there is a partial reaction of degeneration. (*d*) The *reflexes* are *weakened* or abolished; they are roughly proportionate to the atrophy and due to disease in the motor side of the reflex arc. They are never increased. The sphincters are intact, but sexual power is often lost. (*e*) *Sensation* shows no objective change, and even subjective paræsthesia is rare; in some instances there are dull aching pains in the muscles or joints. (*f*) Vasomotor, secretory and trophic symptoms are lacking. The coolness of wasted parts is due to lack of motility.

Diagnosis.—The diagnosis is based on (*a*) the slow course; (*b*) the involvement of the muscles of the hand and its slow "individualization"; (*c*) fibrillation; (*d*) partial reduction in electrical irritability or partial reaction of degeneration; (*e*) absence of increased reflexes and (*f*) of any sensory signs.

Differentiation is necessary (1) from disease in which muscular atrophy is but one among several symptoms. (*a*) *Syringomyelia* is marked by its trophic characteristics, its analgesia, thermo-anæsthesia and less symmetrical wasting. (*b*) *Meningitis*, pachymeningitis, spondylitis, tumors and myelitis, are manifested by anæsthesia, pain, tenderness, and rigidity. (*c*) In *root neuritis* or *multiple neuritis* there is a clearer reaction of degeneration, more sensory signs and more tenderness in the nerve trunks. (*d*) The *atrophies* caused by an individual's occupation are distinguished by the history and slight sensory disturbance.

(2). *Other muscular atrophies* must be differentiated. (*a*) Subacute and *chronic poliomyelitis* causes paralysis first and atrophy afterward; it affects groups of muscles at once, its course is not gradual but rapid and fibrillary contractions are infrequent. (*b*) The *myopathic atrophies* must be distinguished (see p. 1135). (*c*) *Amyotrophic lateral sclerosis* is an allied, perhaps an identical affection; Charcot held that the atrophy is not "individualized," but occurs *en masse*; the distinguishing features are the increase of the reflexes, spasticity, and a much more rapid course. Gowers has never seen a case of progressive spinal atrophy without anatomical change in the pryamidal tracts; in describing the two affections together, he distinguishes three varieties (i) extreme atonic atrophy, (ii) muscular spasm and weakness with little or no atrophy, and (iii) moderate tonic atrophy with increased myotatic irritability. (*d*) *Arthritic muscular atrophy*, probably due to reflex influences, or direct extension of inflammation to the muscles or nerves, may be acute or chronic, and involves chiefly the extensors in a moderate wasting; diffuse wasting suggests a nervous cause; the history or evidence of joint disease is distinctive. The electrical reactions are normal or show only slightly decreased excitability. There is usually distinct increase in myotatic irritability and reflex action.

Course and Prognosis.—The onset between the twenty-fifth and forty-fifth years is gradual, the course is slowly progressive, remissions infrequent, arrest very rare, and death usually results from intercurrent disease, respiratory affections (tuberculosis) or bulbar palsy. Gowers observed arrest but rarely, and then chiefly in cases marked by symmetry and synchronous involvement of both hands; arrest occurs, unhappily, chiefly late in the disease.

Treatment.—Drugs are of uncertain value. Gowers gives strychnia hypodermatically; he has seen 7 consecutive cases of arrest of which 6 were permanent; it has little effect by mouth. Arsenic and iodides are employed. Massage, active and passive gymnastics and electricity are always indicated but do not stay the disease.

4. Neuritic Muscular Atrophy.

Charcot-Marie type. Peroneal family type (Tooth). Neuritic type (Hoffmann).

Etiology.—It is often hereditary, even through five generations; it is usually familial. It occurs in the second half of childhood, and rarely after the twentieth year. Sixty-six per cent. of cases are in males.

Pathology and Symptoms.—The pathological characteristic is *neuritis*, which Hoffmann thought might ascend to the post. roots; secondary changes have been seen in Goll's column. Early atrophy occurs in the *small muscles of the feet* and may not attract attention until bilateral symmetrical peroneal paralysis and atrophy develop, together with paralysis in the long toe extensors, and club feet (*pes equinus* or *varo-equinus*). The calf muscles are the next to suffer and the thigh is involved still later. Then after years, paralysis and atrophy of the hands, as in progressive spinal atrophy, develop, with the "claw hand"; in involvement of the forearm, the extensors suffer more than the flexors. The shoulder, neck, trunk and face are seldom diseased. Fibrillation is infrequent; the reaction of degeneration is generally present, sensory manifestations are common as pains or anæsthesia (especially plantar), and reflex action is decreased or abolished, though the patellar reflexes may long persist.

The *diagnosis* is based on the hereditary or family history, the age, localization and signs of slow neuritis. *Treatment*, as in other forms of muscular atrophy, is unsuccessful.

5. Muscular Dystrophy.

The progressive muscular dystrophies (Erb) or myopathies (Charcot) are often classed with diseases of the muscles, which indeed show the sole constant pathological lesions. They are so closely related clinically to the less frequent spinal atrophies that they are here considered. Their status is provisional, since changes in the cord, nerve trunks and nerve filaments have been found in some cases of each type. In all types (*v. i.*), with few exceptions, there are certain common characteristics; (*a*) *heredity* is an obvious cause in 56 per cent. of cases; though this factor may be absent, a familial incidence is often observed; the muscular system seems deficient in nutrition, development or vitality. (*b*) It usually occurs in *childhood* or pubescence, rarely in adult life. (*c*) The *pathology* is practically the same in all types; the *muscles waste*, with or without hypertrophy, or with increase of fat or interstitial growth. (*d*) Being strictly a muscular affection, fibrillation and the reaction of degeneration are very rare. (*e*) In localization, certain muscles are involved, as those of the face (orbicularis oculi et oris), shoulder (pectoralis, latiss. dorsi, serrat. magnus, rhomboidei, trapezius), arm (biceps, brachialis, supin. long., triceps), trunk (erector spinæ), glutei and thigh. Others are usually exempt, as the deltoid, supra- and infraspinatus, small muscles of the hands and feet, leg muscles (gastrocnemius), and those of the tongue and pharynx. There are three forms.

Pseudohypertrophic Muscular Paralysis.—Lipomatous muscular atrophy is the least frequent, but is the longest and best known type. (Bell 1830, Partridge 1837, Meryon 1852, Duchenne 1861.) It occurs in males (80 per cent.), and is less marked, less early in life, and less fatal in women. It is often transmitted by healthy mothers, the males being impotent, and may be seen in five generations. It begins early (75 per cent. under ten years), and but rarely after twenty years of age.

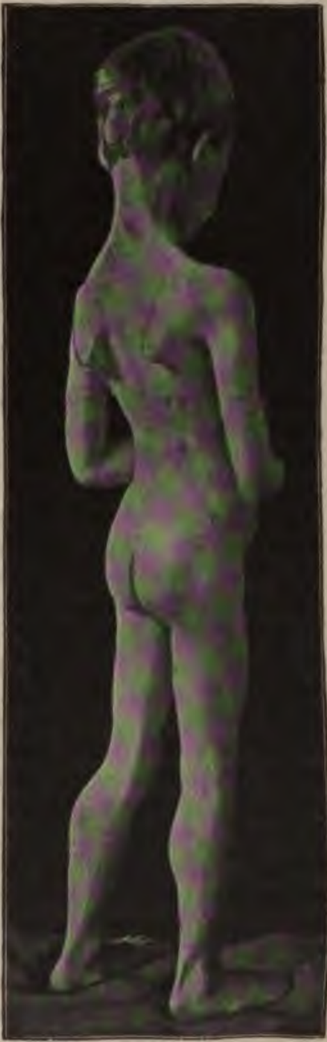
1. *Impaired motility* is the earliest symptom. The weakest muscles are the invisible hip flexors. Weakness of the hip and knee extensors makes climbing of stairs difficult, causes the waddling gait (paresis of glutei) and interferes with rising from the floor, so that the child must

resting on all fours, and then by "climbing his own legs with his
" (Gowers). Paresis of the back muscles and hip extensors causes
r lordosis and abdominal protrusion. The ankle extensors also
n. The child stands with legs far
The depressors of the shoulders
akened early in the disease, later
ulder muscles, then the biceps and
, but the hand remains normal
long time, or is permanently un-
d. The weak muscles show nuclear
se, atrophy and division of their
which are usually undersized, ir-
r in width, pale and narrow; fatty
ration and loss of striation are
Increase of connective tissue is a
ut most characteristic change.

ertain muscles show apparent in-
in size (*pseudohypertrophy*), which
largely to early adipose deposit or
to actual muscular hypertrophy.
uscles are lax. The calf muscles
pecially enlarged, then the glutei,
xtensors, or infraspinatus which
sts strongly with the wasted latis-
and deltoid; the muscles of the
eck and forearm seldom show en-
ent.

s to *other symptoms*, the electrical
ility is lowered, but there is no
n of degeneration. The deep re-
decrease and disappear, from the
lar lesion. Mentality, sensation
e sphincters are normal. Con-
es, such as the *pes equinus*, may
ate in the course. Cardiac hyper-
was observed by Glotz in 60 per
f his cases. The course is pro-
e, the patient becomes bed-ridden,
is rare, and death occurs after
from intercurrent disease or from
tory inflammation.

FIG. 76.



Typical pseudohypertrophic muscular paralysis. (Dercum.)

ntile Atrophic Form, with or with-
ial Involvement.—There is usually
ertrophy, but wasting occurs from the beginning. It may be
ed with the first form, and the legs then show pseudo-
rophy and the arms simple atrophy. The pathology is that of
hypertrophic muscular paralysis, save that there is no hyper-
, true or false; increased connective tissue characterizes this

type of atrophy and also that of the juvenile form (see below). The *face* may be involved, as was first observed by Duchenne and later emphasized by Landouzy and Déjérine. The lids droop and cannot be wholly closed, the forehead is smooth, the cheeks are flaccid and droop, the lips part and functionate poorly, as in whistling, the lower lip protrudes, the nasolabial fold disappears, the face becomes expressionless, (the "*facies myopathica*"). Sometimes the buccinator muscles are implicated but rarely the masseters or those of the tongue, palate, larynx, pharynx, or eyeball. Simultaneously or more often later, the shoulder girdle wastes as in the first type; this is the *facio-scapulo-humeral* form of Landouzy and Déjérine; the latissimus and lower part of the pectoralis are usually first affected, but in the biceps, triceps and often in the supinator longus, wasting and paresis are more conspicuous; the wasting is usually symmetrical. The forearm save for the supinator longus and the hand usually escapes. The neck is seemingly lengthened (Brissaud), because of the drooping of the shoulders and clavicles. The leg muscles then waste, especially the hip flexors, knee extensors and less often the glutei; lordosis, the waddling gait and symptoms observed in the next type (*v. i.*) may develop. The order given may be altered, for facial wasting may follow that of the arms. The course is usually progressive, very chronic (ten to fifty years) and the disease in itself rarely causes death, which results from intercurrent infections or respiratory catarrhs, tuberculosis, etc. In the second and third types, the prognosis is more favorable than in the first form and the outlook is better when the face is unaffected.

The Juvenile Form of Erb.—This type begins after puberty, possibly later (twentieth to fortieth year), and is more frequent in women than the above forms. It begins more often in the shoulders and arms, than in the legs. The pectorals, latissimus, trapezius, serratus mag., rhomboidei, sacrolumbalis, longiss. dorsi, supinator longus and later the triceps are wasted, while the sternomastoid, supra- and infraspinatus, lev. anguli scapulæ, coracobrachialis, teretes, deltoid, and the hand and forearm (except the supin. long.) are almost always exempt. If we lift the patient with our hands beneath his shoulders, he tends to "slip through"; this is due to "loose shoulders" (Erb). The scapulæ stand out prominently from weakness of the serrati. In the lower limbs, the glutei, quadriceps and less often tibialis ant. and peronei become atrophied. The lordosis and gait are as above described.

Diagnosis of Myopathic Atrophies.—(*a*) *Atrophy* from *cerebral lesions* is less marked than myopathic atrophy and more often follows the marked loss of power. (*b*) *Spinal muscular atrophy* (accordin Leube) is differentiated as on page 1135.

(*c*) *Congenital spastic paraplegia* occurs in children with weak and contractures in the calves but the history, increased reflexes, an regressive rather than a progressive tendency easily distinguish (*d*) As to *peripheral neuritis*, the hereditary neurotic (neuritic) muscu atrophy (*q. v.*) is easily recognized. Multiple neuritis (*q. v.*) is usual toxic, more acute and shows sensory symptoms and local tenderne

SPINAL PROGRESSIVE MUSCULAR ATROPHY. vs.	MYOPATHIC PROGRESSIVE MUSCULAR DYSTROPHY.
Begins in small hand muscles.	In trunk, shoulder, or pelvic girdle, leg and upper arm.
Extends upward (face free).	Progresses downward; face sometimes affected.
Muscular atrophy.	Frequently with hypertrophy.
Fibrillation the rule.	Very exceptional.
Tendon reflexes may be increased in the legs, if we include the amyotrophic lat. sclerosis.	Never increased.
Reaction of degeneration, partial, perhaps complete.	Practically never present.
Age: usually begins after thirtieth year.	Usually in childhood; rarely after twentieth year.
Heredity—rare.	Frequent.
Bulbar symptoms not uncommon.	Never present.

over the nerves and muscles. (*e*) In *congenital defects* in the pectoral muscles and trapezius, any progression is naturally absent.

Treatment.—Electricity is recommended, but a current sufficiently strong to excite the wasted muscles causes too much pain. Massage, but especially reasonable exercise, is advisable, because the patients begin to fail when they cannot walk. In advanced cases the respiratory tract must be watched, since catarrh predisposes to tuberculosis.

Under diseases of the motor neurones, ophthalmoplegia, Landry's paralysis, bulbar palsy, myasthenia, acute poliomyelitis, etc., might also be described; these affections, however, are treated elsewhere for anatomical or differential reasons.

III. Combined System Diseases.

In 1867 Westphal described these affections in paretic dementia, but Kahler and Peck in 1877 brought to more general attention this group of cases with simultaneous disease in several systems of neurones. Von Leyden and Goldscheider consider them diffuse or even myelitic affections rather than true system diseases, of which, in their opinion, there are but two examples, tabes and progressive muscular atrophy.

1. Hereditary Ataxia, Friedreich's ataxia (Friedreich, 1861). —**Etiology.**—This affection is hereditary in 33 per cent. and in the other instances it is usually familial, although isolated cases are seen. Most cases occur in boys. It is held by some to be a congenital hypoplasia of the pyramidal and other tracts, tending to degeneration

(Friedreich, Schultze, Strümpell), though others regard it as a combined system disease (Kähler, Pick.) Cerebellar atrophy has been held as causal (Senator, Mentzel) but this is more striking in the hereditary cerebellar ataxy, described below.

Pathology.—The cord or medulla, and even the pons are small. The earliest and dominant change is atrophy with secondary *sclerosis of the post. roots and post. columns,* and this occurs in Goll's more than in Burdach's columns. Lissauer's "root zone" is less affected than in tabes, but more than in ataxic paraplegia. The *direct cerebellar tract* and Clarke's gray columns share clearly in the atrophy, as does the ant. lat. ascending tract of Gowers. The *lat. pyramidal tract* suffers in most cases, its inner portion being usually normal; the ant. pyramid. tract is less altered and principally in the cervical region. Chronic leptomeningitis, sometimes with annular sclerosis of the cord, is occasional.

Symptoms.—(*a*) The *cardinal sign and the one first observed* is *ataxia,* which is less rapid, excessive and stamping (as to gait) than tabetic ataxia, and more closely resembles cerebellar ataxia in its tumbling character and involvement of the trunk. Static ataxia appears later but to a marked degree, and the head oscillates as it does in multiple sclerosis, or the arms show jerky movements. *Romberg's sign* is usually present, and the subject leans over to watch his feet; this leads to some kyphosis. Testing the sense of location elicits ataxia, as in the knee-ankle test. The arms become atactic after a few years. *Nystagmus* develops in most cases, though it is a late symptom; it is elicited chiefly on lateral (or upward) movements and is atactic, as is also the *disturbance* of speech, which develops in three to ten years; the speech is monotonous, irregular, unclear, slightly scanning, slow and strained; syllables are often elided, the tongue may tremble and a nasal twang may be noticed.

(*b*) Regarding the *reflexes,* the *knee jerks* are decreased early or are *abolished,* being present only in rare atypical cases. The skin, pupillary and sphincter reflexes are usually normal. In some (possibly syphilitic) cases the pupil reflex may disappear. (*c*) *Other signs* embrace: *Club foot* (pes equino-varus) is common, and is best seen in the dorsal decubitus; the foot is shortened and widened, the arch is exaggerated (pes cavus), the great toe is strongly flexed dorsally (extended). Scoliosis and kyphosis are common. Muscular paralysis or wasting is rare, although late paraparesis may develop. Sensory signs are few; tactile and muscle sensation are sometimes blunted; the pain sense is but rarely impaired; paræsthesiæ, crises, lancinating pains, and trophic, vasomotor and sensory disturbances are lacking. General or sexual development may be retarded at puberty. The pulse is often increased and hæmic murmurs may be heard. Cerebral symptoms, as mental dulness, develop late, if at all.

Course and Prognosis.—About 33 per cent. develops before the sixth year, 33 per cent. between the sixth and tenth, and 33 per cent. after the tenth year. The atactic gait is the first sign, then speech is involved, possibly then the kyphosis and club foot develop, and ter the arms are affected; the course is steadily progressive, possibly ith remissions, and the patient becomes bed-ridden in twenty to irty years. Death follows from acute infections, cystitis and sepsis.

Diagnosis.—The diagnosis is based (*a*) on the hereditary or familial tiology; (*b*) the age; (*c*) the ataxia; (*d*) absent knee jerks; and (*e*) isturbance of speech.

DIFFERENTIATION.—1. In *tabes*, there are also ataxia and absent knee rks but in hereditary ataxia, the syphilitic stigmata or history, the nmobile pupil, eye paralysis, optic atrophy, pains, crises, and vesical, ophic and marked sensory disturbances are absent.

2. *Multiple sclerosis* (in which there is also speech alteration, ystagmus, ataxia and tremor), is differentiated by the history of tecedent acute infections, the presence of spasticity, increased reflexes, tic atrophy, compulsory laughing and other psychical signs, apoplecti- rm insults, a distinctly intentional tremor and slow, scanning speech.

3. In *cerebellar disease* the ataxia is similar, but in tumors there is tic neuritis, headache, and vomiting. Marie, Londe and Nonne scribed a clinically similar affection, *hereditary cerebellar ataxia*, hich is probably due to simple congenital atrophy of the cerebellum iddle lobe), degeneration in Clarke's columns, cerebellar tracts and sterior columns, is also hereditary or familial, but develops later, tween the twentieth and thirtieth years. The ataxia is cerebellar. he *reflexes are increased* (which Patrick thinks is the sole differentiat- g point), often with spasticity; quite frequently there is *optic trophy*, limitation of the visual field for objects and color, eye paralysis nd sometimes an immobile pupil, nystagmus, choreiform movements, nd moderate sensory and speech disturbance, which may be nasal and arked by unequal inspiration and expiration. The club foot, scolio- s and Romberg's sign are absent. Sanger Brown has reported 25 nd J. H. Neff, 13 cases.

4. *Cerebral diplegia* of children, and ataxic paraplegia are dis- nguished by reflex increase and spasticity.

5. "*Amaurotic family idiocy*" (Sachs, Peterson, Hirsch) is char- cterized by paresis or paralysis, idiocy, optic atrophy, marasmus, nd a rapidly fatal course before the third year of life. At autopsy ypoplasia, microgyria, decreased pyramidal cells, tangential and other bers are noted. Sachs observed 27 cases in Jews, and Haveroch (1904) ollected 36 cases.

Treatment is wholly symptomatic.

2. Ataxic Paraplegia (Gowers).—Most cases occur in males between irty and forty; cold and concussion are possible causes; syphilis is rare cause. *Pathologically*, there is a lateroposterior sclerosis; in e lat. column the degeneration is not wholly systematic, symmetrical r pyramidal; it may invade the mixed zone; in the post. column the rocess is not more intense in the lumbar cord, and is less intense in the ot zone than is the case in tabes. Sometimes the process is so diffuse at it suggests myelitis or arterial obstruction (Marie). The early *ymptoms* are those of a gradual *spastic paraplegia* (muscular weakness, exaggerated reflexes, moderate rigidity), followed by *ataxia* (mostly in the legs, with Romberg's sign). The arms are involved later, though to a less degree. In the last stages the ataxia is obscured by the spastic paraplegia; vesical or rectal tenesmus and muscle cramps are fairly common. Cranial involvement is infrequent, though sometimes suggested by slightly disturbed articulation, nystagmus, tremulous facial movements, and increased jaw-jerk. The mind is usually clear, though in some cases late dementia develops. Sexual activity is often lessened, and the sphincters are sometimes moderately disturbed. Dull sacral pain is sometimes observed.

Differentiation is necessary (*a*) from *tabes*. Certain tabetic symptoms are absent, as the high-stepping and sudden-stamping gait, the immobile pupils, optic atrophy, eye paralysis, lancinating pains, crises, other sensory disorders and absent knee jerks. (*b*) *Friedreich's ataxia* is distinguished by the age, heredity, speech, and absence of patellar reflexes. (*c*) *Myelitis* is more acute, diffuse and later is regressive. (*d*) From simple *spastic paraplegia* it is recognized by the ataxia.

The *prognosis and treatment* are those of spastic spinal paraplegia.

3. Other Combined System Diseases.—Combined sclerosis may be (*a*) *exogenous*, as in pellagra (*q. v.*) with degeneration, and then sclerosis of the posterior lateral columns; as in ergotism (*q. v.*), the longest known variety, and in lathyrism (*q. v.*), or it may be (*b*) *endogenous*, as in multiple neuritis, diabetes (Williamson), pernicious anæmia (Lichtheim), cachexia, icterus, leukæmia (Minnich), brain tumor, plumbism, or diphtheria. Some toxin causes degeneration which is followed by sclerosis. It was found by Putnam in enfeebled middle aged persons, especially in women. Dana, Bastianelli, Batten, Russel, and Collier have described similar cases.

Pathologically, there is posterolateral sclerosis with diffuse or at times discrete degenerative foci. All the columns may be affected. The most diffuse changes are in the cervical and the least in the lumbar cord, and sometimes changes exist in the ant. horns and Clarke's columns. There are sensory *symptoms* (lancinating pains, paræsthesia, anæsthesia, ataxia), motor symptoms (paresis, paraplegia, choreiform movements) or reflex symptoms (the patellars, for instance, being exaggerated or even lost, the sphincters incompetent); optic neuritis and œdema are occasional findings.

V. DISEASES OF THE PERIPHERAL NERVES.

Mononeuritis.—Inflammation of a single nerve trunk.

ology.—(*a*) *Injuries, wounds,* fractures, dislocations, delivery, vio- auscular contractions as in blacksmiths; exhaustion or disturbed olism; anæsthesia (ether) paralyses, the arms being held over the (in the Trendelenberg position), the legs bound in holders, or Esmarch's bandage is applied; ether hypodermatically, malpo- s of the arm during sleep, and the use of crutches, are causes. *ontiguous disease* (pleurisy, pelvic inflammation, compression mors, or aneurysm) may produce neuritis. (*c*) *Cold* (rheumatic ysis) causes inflammation in the sheath, whence extension to the . (*d*) *Toxic causes,* as plumbism and alcoholism, and acute or ic infections (*v. i.* MULTIPLE NEURITIS) are factors.

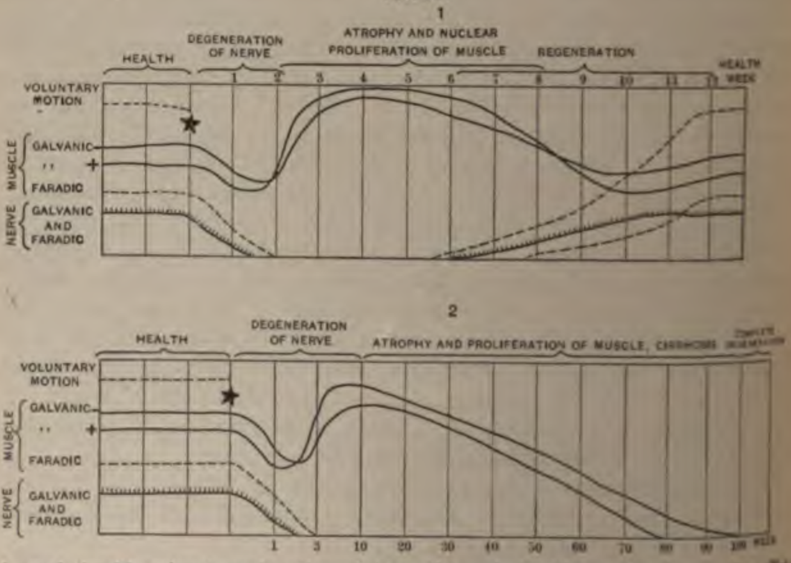
hology.—In *acute cases,* the nerve is red, swollen, soft, and imes the seat of punctate hemorrhages or extravasation of cells in the sheath or between it and the nerve fibrils. In some inflammation involves the sheath chiefly (*perineuritis*), and in the interstitial tissue (*interstitial neuritis*), in which the included fibrils are readily compressed. The process is either disseminated al (where the nerve divides or enters the fascia or bone). In many the nerve fibrils are most affected (*parenchymatous neuritis*). It n difficult to distinguish between inflammation and degeneration; h processes there is nuclear multiplication and protoplasmic swell- n the neurilemma; this compresses the myelin sheath, which nes varicose, granular, fragmented, shows nuclear increase and becomes emulsified. The axis cylinder, composed of thirty to primitive fibrils, becomes granular, divided and may largely or y disappear. *Regeneration* is probably accomplished from the or cerebral centres but also from the periphery.

nptoms.—Symptoms vary with the intensity, extent and seat of euritis (see INDIVIDUAL NERVES). Constitutional disturbance attend acute cases.

SENSORY SYMPTOMS.—The chief local symptom is *pain,* ex- nced largely in the sensory nerves, either locally or eccentrically heir area of distribution; it is increased by tension or movement, ally intense and is sometimes increased at night. *Tenderness* is ble to involvement of the nervi nervorum (Wier Mitchell). ræsthesia, hypæsthesia and paræsthesia may be noted, but com- anæsthesia is rare except for relatively small areas. Sometimes vollen nerve or perhaps nodules (*n. nodosa.*) may be felt.

MOTOR SYMPTOMS.—The *muscles* supplied are *tender* to touch, ul on movement, *paretic* or paralyzed, and atrophied (separation their trophic centres or loss of reflex connections). They are nar- , less clearly striated, granular and finally indurated. Muscular actures may result reflexly, especially in traumatic and professional upation neuritis. The *reaction of degeneration* is present. The normal nerve is stimulated by the faradic (induced) and by a stronger degree of the voltaic (constant) current; the faradic produces a continuous muscular contraction and the voltaic produces a contraction when the circuit is made or broken,—the cathode closing contraction, which is first to appear and stronger than the anode closing contraction. In disease, the following changes from normal occur (Church). (*a*) "The *muscle* responds weakly, sluggishly and deliberately to faradism, and tends to maintain contraction after the current is withdrawn. This is the *modal change*, the most essential element in the reaction. (*b*) "The *nerve trunk* loses progressively and equally its responsiveness to both galvanism and faradism—a *quantitative change*. (*c*) "The *muscle* becomes much more excitable by galvanism and much less excitable by faradism; the latter reaction and the responses in the nerve trunk are completely lost after two or three weeks. This is the *qualitative change*. (*d*) "A *polar change* appears in the muscle about the second week when directly stimulated by galvanism; the muscle is not only more readily caused to contract by the constant current, but the normal mastering strength of the negative closing contraction over the positive has disappeared and the positive closing contraction is equal to or greater than the negative" (A. Church).

FIG. 77.



1, paralysis with early return of motion; 2, incurable paralysis with complete degeneration (Erb, modified by Church).

In the middle or *mixed form of Erb* there is a normal reaction in the nerves and increased voltaic response in the muscles.

3. TROPHIC SYMPTOMS.—The skin is sometimes red, "glossy," œdematous or more rarely thickened. Increased secretion of sweat, eruptions as herpes, erythema or pemphigus, inflammation or adhesions in the joints, and perforating ulcers are less common.

Neuritis may sometimes *ascend to the cord* or its membranes as in traumatic cases or in bladder disease. In rare instances the corresponding opposite nerves may become involved without disease of the cord; this is the so-called *sympathetic neuritis.* The *progressive hypertrophic interstitial neuritis of childhood* (Déjérine and Sottas, 1893) is a rare affection, in which there is anatomically sclerosis and hypertrophy of the nerves, ant. and post. nerve roots, and sclerosis of the post. columns as in tabes, and clinically, muscular atrophy, pains, anæsthesia, nystagmus, pupils immobile to light, and kyphoscoliosis.

Diagnosis.—Diagnosis depends on (*a*) localization of symptoms to a nerve trunk and its distribution; (*b*) pain, tenderness and objective sensory alteration; (*c*) muscular atrophy or paralysis; and (*d*) trophic changes. The early diffuse pain may simulate disease of the *bones or joints.* Chronic (sensory) forms may resemble *neuralgia,* in which the pain is paroxysmal, the tender points more localized, the initial tenderness less, and objective sensory alteration absent. Disease of the cord or *nerve roots* usually paralyzes several muscles and affects sensation segmentally.

The **prognosis** depends on the cause and intensity of the process. Acute cases last for weeks; chronic cases, for months. The outlook is usually good in traumatic cases, and bad when they are due to contiguous suppuration.

Treatment.—The treatment is (*a*) that of the cause. (*b*) *Absolute rest* is indicated. Exertion increases just as it may initiate, neuritis. (*c*) *Pain* should be relieved. The salicylates and iodides are of uncertain value. Local heat may relieve, but sometimes causes burns and ulcerations, for the anæsthetic skin may not perceive its intensity. Coal-tar products should be given in preference to morphine, cocaine or Schleich's infiltration anæsthesia, because habits may be contracted; opiates are necessary in severe cases. The positive pole of the voltaic current, just strong enough to be felt, may relieve pain. (*d*) *Paralysis* should be treated only after the acute stage. The weak interrupted voltaic current (not the faradic) is indicated. Massage and later passive motion are beneficial largely because the veins from the nerves empty into the muscle veins and surround the muscle arteries.

2. Multiple Neuritis; Polyneuritis, Peripheral Neuritis. This affection was described by James Jackson (1822) and Ladd (1854), but the first accurate and pathological description was given by Dumeil in 1864, while Joffroy, 1879, Leyden, 1880, and Stewart, 1881, made important contributions.

Definition.—Neuritis (or nerve degeneration) due to the selective action of toxic substances on the less resistant peripheral nerves, causing (*a*) multiple, symmetrical, bilateral, flaccid, or atrophic paralysis with (*b*) the reaction of degeneration, (*c*) sensory and (*d*) often trophic disturbances.

Etiology.—The causes are (*a*) *poisons*; *alcohol* is the most important; lead, arsenic (in beer), mercury, phosphorus, carbon monoxide, silver, aniline and ergot may also produce the disease. (*b*) The *infections*, (i) acute, as *diphtheria*, typhoid, sepsis, and also, occasionally, almost every active infection, and (ii) chronic, as *beri-beri*, *leprosy*, tuberculosis or syphilis, are etiological factors. (*c*) *Intoxications*, as auto-intoxications, *diabetes*, *nephritis*, *digestive disorders*, *cachexias* and pregnancy, may be causal. (*d*) *Other causes* include the seemingly primary cases. Cold and *rheumatism* may produce it; our present knowledge cannot explain their mode of action; probably these cases should be classified as infectious, for spontaneous multiple neuritis has occurred epidemically. Arteriosclerosis (of the nerve vessels) is a rarer cause. It may occur with tabes. It is a disease of adults (twentieth to fiftieth year); in children it may occur with poliomyelitis.

Pathology.—In *acute cases* the nerve may be red, swollen and the seat of punctate hemorrhage, exudation of leukocytes or albuminous exudation in the connective tissue, but usually *parenchymatous degeneration* predominates. It appears first and most markedly in the smaller nerves. As in mononeuritis, the axis cylinders waste and the medullary sheaths disintegrate. The symmetry and multiplicity of degeneration in the nerve endings is caused by the altered condition of the blood by toxins or poisons. Acute polymyositis of similar origin may accompany neuritis multiplex. In cases which are *chronic* from the beginning, there is slow degeneration of the nerves. The connective tissue is increased in certain forms, as gouty or leprous neuritis. The toxic cause may also produce degeneration of the cord or brain and changes in the liver (alcoholic cirrhosis), kidneys (alcohol and lead), and lungs.

Symptoms.—The symptomatology varies considerably with the cause. In *acute cases* it may begin with chills, fever, delirium, headache, diarrhœa, icterus, bronchitis, acute splenic tumor, albuminuria or other toxæmic symptoms; these are followed by prodromal numbness, tingling, pains, cramps or vasomotor symptoms, while in *chronic cases* the symptoms develop slowly.

1. *Sensory symptoms* are usually *first to appear*, the last to disappear, and are throughout the most vexing manifestations. Paræsthesia precedes pain and pain precedes paralysis. The muscles, nerve trunks and skin are the seat of *spontaneous pain*, *tenderness to pressure* and *pain on movement*. The palms of the hands and soles of the feet are especially hyperæsthetic in the tabetic, diabetic and diphtheritic forms. *Sensation* is often *blunted*, perverted or less often completely suspended; the most marked findings exist *below the elbows and knees*, although higher extension, even to the face, is possible. Sensory dulness may concern pain, temperature, tactile or muscular sensation. Some sensory disturbance is rarely absent. Delayed conduction may occur. The joints may be painful and swollen, especially in the hand, causing thickening and limitation of movement.

2. *Motor symptoms* occur chiefly in the *peripheral* distribution of

the *spinal nerves*; they develop first and are most marked in the *extensors* of the *leg and foot, forearm and hand,* and produce the *ankle and wrist drop* (ext. popliteal and musculospiral nerves). The foot is also inverted. The paralysis is *flaccid,* being rarely attended by muscular spasm. If the paralysis is only moderately severe in the legs, the "*steppage*" gait (Charcot) is noted, and the patient lifts the legs high to clear the pendant toes ("turkey-gobbler walk"). The paralysis later involves the flexors and the muscles of the arm and thigh. The muscles may show an early increased irritability to both the galvanic and faradic currents; this is followed by some or all of the elements of the *reaction* of *degeneration* (sometimes before paralysis appears, Popoff). The muscles rarely fail to respond to the galvanic current, even in the extreme cases. Then the *muscles atrophy,* chiefly in *distal parts, i. e.,* where the sensory and motor changes predominate. Other motor manifestations are cramps, tremor, fibrillation, ataxia and contractures. The patient either lies on the back with the members extended or on the side with flexor contractures, which draw the heels to the buttocks and the thighs to the abdomen. The trunk muscles may be affected as well as those of the neck and eyes, face, tongue, or indeed those supplied by any cranial nerve; rapid pulse, palpitation, dyspnœa, and vocal disturbance indicate involvement of the vagus; optic neuritis and amblyopia may occur. Involvement of the cranial and spinal nerves is observed in the neuritic form of Landry's paralysis. The tendon and skin *reflexes decrease* and *disappear* although initial increase or exceptional persistence has been noted. The organic reflexes are almost always preserved. Involvement of the bladder and rectum, which is very rare, is referable to the mental state or to neuritis of the pudendal plexus.

3. *Trophic symptoms* may be present. The skin is cold, turgid, harsh, sometimes glossy; eruptions (herpes, erythema), ulceration, bed-sores, and trophic changes in the nails or hair, are uncommon; œdema over the wrists and ankles may occur, especially in alcoholics, and is trophic or vascular in origin.

4. There may be *general symptoms.* Mental symptoms and malnutrition may result from the antecedent toxæmia or cachexia, pain or disturbed circulation. Korsakow and others have described the "neuritic psychosis," marked by amnesia, delirium, and hallucinations, which are probably due to the causes just mentioned. Recovery is the rule. Some visceral symptoms have been referred to involvement of the cardiac, sacral or pudendal plexuses, although mostly without positive evidence.

Course and Prognosis.—Whether the disease begins acutely or slowly, the common order of symptoms is paræsthesia, pain, paralysis, amyotrophy, trophic and mental symptoms. The distal parts are involved more early and intensely than the proximal. In most cases recovery requires about a year. Paralysis of the vagus may cause death. The *prognosis* depends in part on the cause. It is good in sensory types, and in forms of slow evolution, when the sensorium, arms and trunk, cranial nerves and sphincters escape involvement, and when visceral disease as liver cirrhosis, cord or brain complications and œdema are absent. The prognosis must regard the possibility of recurrence (recurrent type) and cover the progress of the disease, which is inevitably slow, despite treatment.

Diagnosis.—Early diagnosis is important, in order that etiological therapy may be instituted. The *cardinal signs* are (*a*) the motor and sensory symptoms, which correspond in function, peripheral distribution and symmetry; (*b*) the reaction of degeneration; (*c*) tenderness of the nerves and muscles; (*d*) loss of reflexes and (*e*) the etiology.

DIAGNOSIS OF TYPE.—(*a*) The *alcoholic* form is the most frequent; 70 per cent. of cases occur in women; the legs are often involved; there is the "steppage" gait; sensory and mental symptoms are most marked; the atactic form is not infrequent (*v. i.*); recurrence is probable. (*b*) When due to *lead poisoning*, the occupation, lead line, colic and constipation are characteristic; the arms alone are involved as a rule; there is little or no sensory participation; the involvement rarely extends; the wasting is conspicuous. (*c*) *Diphtheritic* form (*q. v.*). (*d*) In the *toxæmic* form there is fever, and constitutional reaction; it begins in the legs or in all members simultaneously, involves equally the flexors and is attended by slight sensory symptoms (Gowers). (*e*) When due to *beri-beri* (*q. v.*), vagus symptoms, dropsy and cardiac failure are frequent. (*f*) *Leprous neuritis*, erythromelalgia and Dercum's disease are considered elsewhere.

DIFFERENTIATION.—Atactic polyneuritis (pseudotabes peripherica, Déjérine) with little or no motor disturbance, may resemble locomotor ataxia:

LOCOMOTOR ATAXIA.—*vs.*	—MULTIPLE NEURITIS.
History or stigmata of syphilis.	Rare: history of infection or intoxication.
Argyll-Robertson pupil.	Absent (loss of accommodation in diphtheria).
Optic atrophy, 10 per cent.	Rare, but toxic amaurosis is frequent.
Eye paralysis frequent.	Much more rare.
Early girdle sensation and lightning pains.	Very rare.
Muscular sense early and frequently disturbed; ataxia.	Rare.
Gait ataxic, stamping, striking heel first.	Gait due to motor more than sensory disturbance. Striking toe first.
Sensory symptoms predominate.	Motor predominate.
Paralysis, reaction of degeneration and amyotrophy rare.	In nearly all cases; rapid in development and symmetrical.
Nerve trunks often insensitive.	Tender, as are muscles.
Arthropathies, osteopathies, *mal perforant*.	Unknown; glossy skin more common.
Crises.	Almost unknown; toxæmic vomiting etc.
Sphincters early and frequently involved.	Very rarely, if ever, and from delirium etc.
Clinical evolution very slow.	Rapid.
Disease incurable.	Recovery usual, if not early death.

Poliomyelitis.———*vs.*———Multiple Neuritis.

Usually in children.	In adults.
More sudden onset.	Less acute.
Embraces whole limb; random distribution.	Peripheral and symmetrical.
Muscles functionating together. Reaction of degeneration in both, but strong galvanic current not required (during first months) to produce muscular contraction.	Dissimilar muscles involved. Muscles respond only to strong galvanic.
Pain and tenderness most rare.	Common in nerves and muscles.
Sensory disturbance rare.	Usually (but not invariably) present.
Immediate tendency to regress, but always some deformity.	Tendency to progress. Total recovery the rule.

In *acute myelitis*, the onset is acute, there are girdle pains, paraplegia, with corresponding anæsthesia, bed-sores, disturbance of the bladder and rectum; there is no tenderness of the nerves or muscles, little or no ataxia, and recovery is unusual. *Landry's paralysis* often cannot be distinguished (which is natural, as one form of Landry's disease is neuritic); sensory, trophic and sphincter changes are absent.

Treatment.—In the etiological therapy, alcohol must always be considered and deception guarded against; it should be withdrawn at once. In sepsis, Gowers gives full doses of iron. For treatment of pain see Neuritis. Absolute rest should be enforced. A full diet, rich in fats, should be given if the stomach can tolerate it; tonics should be given to maintain nutrition, but arsenic should be avoided. When the lower cranial nerves are affected, feeding by the nasal tube or by rectum, measures to avoid hypostasis as in typhoid, and cardiac stimulants are indicated. To overcome contractures from ankle- or wrist-drop, splints or sand-bags should be employed when they do not cause excessive pain. The weight of the bedclothes on the feet may promote contractures, whence frames to lift the bedding should be used. Gentle massage and voltaism may be tolerated after the acute stage, may prevent contractures, and may aid the return venous flow. Bromides and chloral are the most certain somnifacients but chloral must be used with care in chronic alcoholism. Orthopedic measures are indicated in neglected contractures.

3. Neuroma.—Two varieties exist, the true and the false neuroma. The true neuroma may be medullated, or non-medullated, though ganglion cells are exceptional (only 3 cases are recorded); they are usually multiple, sometimes very numerous (3,020 in one case), and have been found in instances on every cranial, spinal, and sympathetic nerve. They may undergo sarcomatous degeneration. The *tubercula dolorosa* (Virchow) are small, painful, subcutaneous nodes on the trunk or extremities, the anatomy of which is not well understood. The *neuroma plexiforme* consists of twisted cords with nodes, is congenital and occurs largely in the distribution of the fifth nerve. The *false neuroma* is usually fibrous; glioma is rare; sarcoma or carcinoma may by contiguity invade adjacent trunks; neuromata have been found in the cerebral nerves in syphilis, and in the peripheral nerves in leprosy. The nerve trunks suffer more in the false than in the true form. *Neuromata in amputation stumps* are of frequent occurrence.

There may be no *symptoms;* local tenderness and local or projected pain are frequent. Reflex muscular spasm is common from nerve irritation. Mentality, sensation or motility may be affected. Pigmentation has occurred. Diagnosis is certain only when the growths are felt. *Treatment* is symptomatic for the exhausting pain, or radical in localized growths. Recurrence is not infrequent.

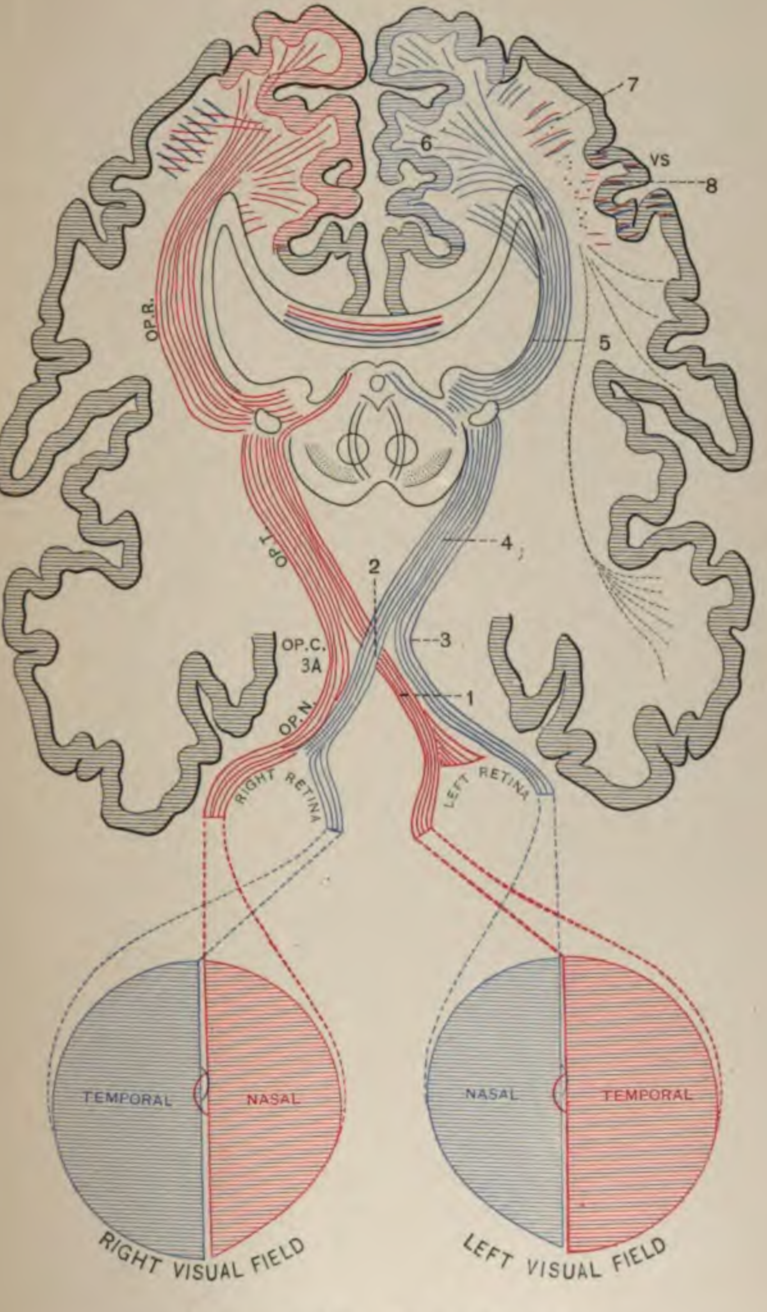
4. Diseases of Cranial Nerves.

Olfactory Nerve.—The end nerves of smell are located on the two upper turbinate bodies and the upper septum; thence they run to the olfactory bulbs, which are really part of the brain. The central connections are not well understood. The centre is thought to be in the uncinate gyrus and the hippocampus, with relations to the optic thalamus, opposite internal capsule and cerebral cortex. The anterior nares perceive odors, the posterior, flavors.

Anosmia is loss of smell. Tests are made not with pungent substances, as ammonia, which irritates the fifth nerve, but with musk, asafœtida, cologne water and the essential oils. (*a*) Disease in the nasal mucosa is the most frequent cause; it may result from paralysis of the fifth nerve, arrest of secretion, or atrophy in old age. (*b*) Disease of the bulb and tract may result from congenital absence of the bulb, basal fracture, or meningitis in the ant. fossa, disease of the middle cerebral artery, tabes (possibly centric atrophy), hydrocephalus, and foci in the opposite internal capsule. (*c*) Central disease may be located in the hippocampus and uncinate convolution, as softening. The prognosis is usually unfavorable and treatment is ineffectual. *Hyperosmia*, or oversensitiveness of smell, occurs in insane and neurotic subjects; it may be cultivated, so that individuals are recognized by their odor. *Parosmia*, or perverted sensation, is rare, and arises from irritation of the nerves or centres. Hallucinations of smell may occur. Imaginary odors, as of burning flesh, may constitute the epileptic aura.

Optic Nerve.—(A). THE RETINA.—*Retinitis* occurs in general diseases, as nephritis (in 15 to 25 per cent.), especially in interstitial forms, and late in the disease rather than early. The degenerative form is most common, the next is the hemorrhagic form, with "flame like" hemorrhages along the vessels, and finally the inflammatory type, with swelling of the retina, serum exudation, cloudiness and disk blurring, which may be as marked as that of brain disease; the white foci which are seen are due to exudation, fatty change or sclerosis.

PLATE XIX.



Visual Paths. (After Vialet.)

OP.N., optic nerve. OP.C., optic chiasm. OP.T., optic tract. OP.R., optic radiation. V.S., visual peech centre. A lesion at (1) causes total blindness in that eye; lesion at (2), bitemporal hemi-nopsia; at (3), unilateral nasal, and lesions at (3) and (3A), bilateral nasal hemianopsia; at (4), emianopsia of both eyes and the hemianopsic pupillary reaction; at (5) or (6), hemianopsia of both yes (pupillary reflexes being normal); at (7), amblyopia, especially of the opposite eye; at (8), word-lindness.

ilar findings may occur in syphilis, anæmia (Quincke), malaria,
æmia (*q.v.*) purpura, plumbism, and diabetes (see PLATES V and
. There are apparently primary cases.

B). OPTIC NERVE.—Optic neuritis (papillitis, choked disk) has
n fully described and differentiated (see BRAIN TUMOR and PLATES
nd VI). Tumors or inflammation in or back of the orbit may
causal. Errors in refraction must be excluded. In the *primary*
a there is no initial inflammation. The edges of the disk are dis-
t, its arteries nearly normal and its color grayish; it may develop
editarily in males at puberty; it occurs in tabes, paretic dementia,
tiple sclerosis, syphilis (in connection with all of which it has been
ussed), and less often from exposure, excesses, diabetes, alcoholism,
nbism, the specific fevers, amaurotic family idiocy and familial
bellar ataxia. The *form secondary to inflammation* commonly follows
ked disk and has the same significance; the disk has an irregular
tour, small arteries, and is translucent. The symptoms are
inution in acuity of vision, field of vision, color perception, and
pupillary reaction.

C). CHIASM.—Each half of the retina contains fibers from the
esponding occipital lobe (see PLATE XIX), that is, each right
receives fibers from the right, and each left half from the left,
pital lobe. The point of sharpest vision, the macula, receives over-
ping fibers from both sides of the brain. The outer (temporal)
tion of the field of vision is more limited than the inner (nasal)
tion, because the nose cuts off light, and consequently it receives
er nerve fibers. The larger number of fibers from each eye, *i. e.*,
se from the nasal retinal half, decussate at the chiasm and lie in its
re. Those from the temporal half are direct and lie at the side of
chiasm. While division or disease of one optic nerve causes blind-
in the corresponding eye, lesions affecting the centre of the chiasm
ussating fibers to the nasal half of each retina) cause half blindness
he opposite (temporal) visual fields, *bitemporal hemianopsia*, since
que rays passing the pupil fall on the opposite half of the retina.
macula receives fibers from both sides of the brain and therefore
pes involvement. Temporal hemianopsia may result from tumors
he pituitary gland, pressure from the infundibulum of the third
tricle, gummata and hemorrhage. The "oscillating" or varying
mporal hemianopsia is often indicative of syphilis. The diagram
strates effects of lesions of the lateral part of the chiasm (3), lesions
cting both sides of the chiasm (3 and 3A) as calcarious or aneurys-
ic carotids; tabes or tumors are most rare; *binasal hemianopsia*
herefore most exceptional; a large lesion might produce temporal
ianopsia in one eye and total blindness in the other; still more
nsive disease might produce total blindness; these changes may
uccessive.

D). OPTIC TRACT.—Just back of the chiasm the temporal fibers
he right and the nasal fibers of the left side form the right optic
t, *i. e.*, the right tract contains all fibers from the right half of each

retina. The tract runs over the crus cerebri, where a lesion very often causes hemiplegia on the same side as the blind field. The fibers enter the geniculate bodies (in which 80 per cent. of the optic fibers end), opt. thalamus and ant. quadrigeminate body, from which gray ganglia the fibers pass by way of the posterior limb of the internal capsule and, by radiating fibers (optic radiation), to the visual centre in the posterior internal part of the occipital lobe, the cuneus. A lesion (at 5, in diagram) in the optic tract anywhere between the chiasm and cuneus produces *lateral or homonymous hemianopsia*; for instance, if it occurs in the right optic tract it produces loss of function in the temporal half of the right and nasal half of the left retina, so that the left half of each field of vision is lacking. Hemianopsia may be partial and may involve only a quadrant, or only a quadrant may remain after partial recovery. The unaffected fields may suffer some reduction. *Heteronymous hemianopsia* designates blindness of the right half of one field and the left half of the other, or the converse. Disease in the gray ganglia or internal capsule may produce contralateral hemiplegia; indeed in 50 per cent. of cases of hemianopsia this association is observed, and less frequently hemianæsthesia. Transient hemianopsia is sometimes seen in migraine. The optic tract is injured in tumors, multiple sclerosis trauma, hemorrhage or softening.

(E). OPTIC CENTRE (see CEREBRAL LOCALIZATION).—A lesion in the cuneus produces (*a*) *homonymous hemianopsia*. Sometimes only an upper quadrant is blind (disease in the upper part of the cuneus) or the lower quadrant is blind (disease in its lower part). Purely cortical hemianopsia is impossible, and Monakow held that the optic radiation must be involved; (*b*) *hemichromatopsia*, or homonymous color blindness or confusion, in which all colors seem gray, is propably due to lesion of the anterior superficial cuneal cortex. Other results are (*c*) *mind blindness*, or loss of visual memory; (*d*) *alexia* or word blindness; (*e*) *optic aphasia;* and (*f*) crossed *amblyopia*, concentric blurring or limitation of the visual fields for form and color; this is usually referred, however, to disease of the gyrus angularis, in which there is thought to be a higher centre in which the half fields are combined and the whole opposite field and part of the field of the same side are represented.

DIAGNOSIS.—The eyes should be tested separately. Disease of *an optic nerve* causes loss of sight and pupillary reaction in the corresponding eye. *Central scotoma* (blindness in the centre of the visual field) indicates inflammation or hemorrhage in the central (axial) fibers of the nerve, usually back of the orbit; it is common in poisoning from tobacco and alcohol. *Peripheral limitation* of the visual fields indicates damage to fibers coursing in the periphery of the optic nerve and some damage to all the nerve fibers. *Sectorial blindness* indicates a pronounced but circumscribed affection of the nerve. *Bitemporal hemianopsia* always results from chiasm disease; unilateral temporal hemianopsia in one eye with complete blindness in the other has the same significance, but also implies that the process has extended laterally, possibly even to the optic tract. *Lateral homonymous hemian-*

opsia indicates disease of the optic tract between the chiasm and cuneus. If the geniculate (or quadrigeminate) bodies are the seat of the lesion, *Wernicke's hemianopsic pupillary reaction* is obtained, because the pupil reflex, while not at present perfectly understood, implies that the retina, the optic nerve, the centre and fibers of the third nerve and the iris are intact. A small beam of light in a dark room should be thrown on the hemianopsic half of the retina, and the patient should look at a distance to eliminate accommodation. If the pupil does not react to light on the blind half, Wernicke's reaction indicates that the disease focus lies at or somewhere to one side of the geniculate (or quadrigeminate) bodies. If the lesion lies between these gray ganglia and the cortex, the reaction of Wernicke is absent. Transient hemianopsia is common in vascular lesions; if it lasts for weeks it will probably remain. In Sequin's case it was present for twenty-three years. It may occur in migraine and very rarely in hysteria. Its association with the crus, internal capsule or cortex symptoms which have been already considered is of localizing importance. *Amblyopia and concentric limitation* in the fields, marked in one eye and less in the other, may result from (*a*) optic atrophy, with the absent pupillary reaction, and seen with the ophthalmoscope; (*b*) disease of one hemisphere, the ophthalmoscopic finding and pupillary reflex being normal, (or (*c*) possibly functional disease).

Functional and toxic blindness may result. (*a*) Hysterical blindness is transitory, and is rarely complete (it is clear that the eye functionates normally though the visual centre neglects the impressions); it is often associated with loss or inversion of the color perception and often with hemianæsthesia. (*b*) *In amblyopia* from tobacco, vision is gradually impaired in both eyes, largely as central scotoma with invariable loss of perception for red and green colors; the fundus is usually normal but sometimes congested; recovery is usual, but atrophy may result. (*c*) *Toxic amaurosis*, usually transitory, may occur from uræmia, poisoning by lead, alcohol, or quinine, syphilis, acute anæmia, and irritation of the fifth nerve.

Ocular Paralysis; Third, Fourth and Sixth Nerves.—General Etiology.—(*a*) *Trauma* to the eye, orbit or base may cause ocular paralysis; it is characterized by a slow course and often imperfect recovery. (*b*) *Compression* by tumors in the orbit which often produce exophthalmos; tumors at the base, aneurysm, and rarely hemorrhage into the nerve sheath may produce it. (*c*) *Neuritis* produced by the above factors, orbital cellulitis, meningitis, syphilis, cavernous thrombosis, alcoholism, and rheumatism, which is made to cover many obscure cases, may be etiological factors. Neuritis may also result from acute infections, as diphtheria; exophthalmic goitre, plumbism and diabetes are rarer causes. (*d*) *Nuclear disease* is usually due to chronic wasting disease, as in tabes, multiple sclerosis or bulbar palsy, but it is sometimes acute from polio-encephalitis or vascular lesions. (*e*) *Supranuclear disease* (*v. i.* CONJUGATE DEVIATION) may be a cause. (*f*) In rare cases there is a myopathic form, as in muscular dystrophies.

Anatomy and Physiology of the Motor Nerves of the Eye.—The nerve supply is by the sympathetic (which through the ciliary ganglion dilates the pupil) and by the third, fourth and sixth cranial pairs, which arise from the middle ventricle, Sylvian aqueduct and fourth ventricle, and are arranged as follows from before backward: (*a*) the third nerve nucleus with first the ciliary and behind it the pupillary centre, then its muscles in this order: the internal rectus, levator palpeb. superior, inferior rectus and inferior oblique; (*b*) fourth nerve nucleus for the superior oblique, lying close to the third nucleus, and (*c*) nucleus of the sixth nerve for the ext. rectus. Their connection with the motor cortex (and possibly with the occipital and parietal lobes) is through the internal capsule and corona radiata. The third nucleus is connected with the seventh; winking and movements of the eyelid are thus associated with the pupil reaction and accommodation. The action of the various muscles is indicated by their names but convergence is supplemented by the sup. and inf. recti and the two oblique muscles. The inf. oblique turns the eye upward, the sup. oblique downward. The upper lid is raised by the levator palp. sup. (acting usually with the sup. rectus) and by Müller's muscle.

PARALYSIS OF THE THIRD NERVE.—This nerve emerges from the crus and follows the cavernous sinus, and therefore may be involved in disease of these structures. A complete paralysis involves all muscles save the external rectus and sup. oblique, and results in drooping of the lid (ptosis), which the patient attempts to overcome by overaction of the frontalis. The eye can be moved only outward and a little down and inward. Accommodation is lost, the pupil is wide from the unopposed action of the sympathetic nerve and does not react to light. There is *double vision from divergent strabismus*, due to action of the unrestrained ext. rectus muscle. Paralysis is often partial, for the levator palp. sup. may escape involvement or be affected alone or with its associate (the sup. rectus), especially in bilateral nuclear disease. *Ptosis* may be (*a*) part of an ordinary oculomotor palsy; (*b*) part of a chronic ophthalmoplegia (*v. i.*); (*c*) reflex in rare cases of trigeminal irritation as from disease of the teeth; (*d*) myopathic as in muscular dystrophy; (*e*) a result of sympathetic paralysis, false or pseudoptosis (with a red, hot, œdematous skin, slight recession of the eyeball, and contracted pupil); (*f*) an incurable congenital form, usually partial and bilateral; (*g*) a transient form, observed in the morning in women, in which there is usually a good prognosis; (*h*) an hysterical form, usually bilateral, in various associations with the sup. rectus, inf. or int. rectus, the eyebrow being frequently lower on the side of ptosis.

Internal oculomotor palsy includes cycloplegia and iridoplegia. (*a*) *Cycloplegia* (paralysis of the ciliary muscle) results in loss of accommodation. Near vision is poor; distant vision is good; extreme myopia or presbyopia make its detection difficult. Isolated cycloplegia in one eye may indicate disease of the lenticular ganglion, in both eyes it is nuclear, as in diphtheria or tabes. (*b*) *Iridoplegia* may assume three forms (Gowers). The first is loss of associated action, *accommodation iridoplegia,* in which there is no pupillary narrowing in the act of accommodation. It is due to the same causes as cycloplegia and is usually but not always associated with it. The second is *loss of the light reflex,* reflex iridoplegia, the Argyll-Robertson pupil, which is seen especially in tabes and paretic dementia (very seldom in softening, tumors, senile atrophy, hydrocephalus or congenitally). The reflex occurs through the optic nerve, optic tracts, corp. quadrigemina or corp. geniculata, anterior part of the oculomotor nucleus, probably the second branch of origin of the third nerve, its trunk, the ciliary ganglion, and its nerves. The pupils are often equally affected and small (spinal myosis) but may be unequal (aniscoria, which is also observed in some normal persons). If the pupils are not small, cutaneous iridoplegia is not present. The third form is *loss of the skin reflex,* the cutaneous iridoplegia of Erb, in which the normal dilatation of the pupil upon pinching, sticking or faradizing the skin of the neck is lost. It indicates, in general terms, disease in the cervical sympathetic nerve, in the fibers between it and the cervical cord, diseases of the cord, especially those affecting sensation, or of the centre, probably situated in the corp. quadrigemina just outside the light reflex centre in the trunks of the first and third nerves. The seat of lesion is probably nuclear, unless special localizing symptoms indicate some other localization. Specifically it is characteristic of tabes, paretic dementia or syphilis.

Recurrent or periodic palsy, to which Gubler (1860) and Möbius particularly drew attention, is a rare form, involving chiefly most (or possibly only one branch) of the third nerve, and rarely the sixth nerve. It occurs chiefly in women. At intervals of months to years, possibly throughout life, a recurring paralysis develops, lasts a few days (two to fourteen) or weeks and then disappears. Forms which begin with headache, pain and vomiting were called *ophthalmoplegic migraine* by Charcot. Its nature is unknown. It is probably peripheral in form (Mauthner), although Möbius thinks it is nuclear; von Monakow considers that its usual cause is organic, as from tumor, meningitis or tubercle; the lesion affects the eye nerves (or centres) and also the descending branch of the fifth nerve; some few cases are seemingly hysterical.

PARALYSIS OF THE FOURTH (TROCHLEAR) NERVE.—The superior oblique muscle moves the eye downward and somewhat inward. Its paralysis is hard to recognize, but it is of little clinical importance. In attempts to converge downward the normal inward rotation of the eyeball is lacking.

PARALYSIS OF THE SIXTH NERVE.—The eye cannot be moved outward, and is rotated inward by the unopposed internal rectus, marked *diplopia* resulting. This nerve is frequently involved, because of its long and exposed course. The nerves of the eye are involved in the following order of frequency: 1, ext. rectus; 2, sup. oblique; 3, inf. rectus; 4, sup. rectus; 5, int. rectus; 6, inf. oblique (von Graefe).

COMBINED EYE PARALYSIS.—Convergence and accommodation are associated functionally and their centres lie adjacent, whence they are often lost together. The internal recti may fail to contract together but may move separately in lateral eye movements. *Conjugate deviation* may occur (see BRAIN HEMORRHAGE AND PONS LOCALIZATION); if a lesion occur above the sixth nucleus, the eyes cannot be directed toward the side of lesion, and are, therefore, directed toward the paralyzed side; if the lesion is at the sixth nucleus, the sixth nerve on that side is paralyzed, often also the seventh, and the opposite third nerve is paralyzed, not from lesion in the third nucleus but from the fact that each third nucleus receives afferent impulses from the contralateral sixth nucleus for associated eye movements.

SYMPTOMS.—The *general symptoms of paralysis of the eye muscles* are five (Gowers):

1. *Limitation of movement* in the paralyzed muscle, followed by secondary contraction of the unresisted opponent. The habitual position of the head corresponds in every way to the physiological action of the paralyzed muscle (Landholt).

2. *Strabismus* (squint), causing lack of parallelism in the visual axes, which are divergent when the int. rectus and convergent when the ext. rectus is affected; this constitutes the *primary deviation.*

3. *Secondary deviation* is the extra movement of the sound eye in its efforts to see, due to its increased innervation, detected by testing with the diseased eye closed, or by testing with ground glass over the sound one.

4. *Erroneous projection* in the paralyzed eye. Distance is normally judged by the position of the eyeballs and muscles, and therefore when secondary deviation from excessive innervation of the sound muscles occurs, it gives the mind an impression of distance greater than is actually the case.

5. *Diplopia* (double vision) results from failure of the visual axes to correspond. The sound eye sees the true image, while the paralyzed eye sees the false one, which is less clear, since the image falls on a less sensitive retinal area. When the images are either near together or else widely separated, the false one may be "neglected." Erroneous projection and diplopia always produce vertigo at first. If the false image is on the same side of the true image as the eye by which it is seen, the diplopia is simple or homonymous; if on the other side, it is crossed or heteronymous. The eyes are tested by holding red glass before one eye and testing vision with a strip of white paper. If the red image and covered eye are on the same side the diplopia is simple; if on opposite sides, it is crossed. When the eyes converge it is simple, when they diverge it is crossed; *i.e.*, "when the prolonged visual axes cross, the diplopia is not crossed." Holding the test object stationary and the head being turned in one way or another, in some directions the images converge or perhaps blend, while in the opposite direction they separate. "The affected eye is that one in the direction of whose image the diplopia increases; the paralyzed muscle is the one which would give to the eye the direction of the false image" (Landholt). Monocular

diplopia is almost invariably hysterical (unless there is disease of the eye media or error in curvature).

Treatment.—Treatment is often unsatisfactory from the etiological standpoint. Surgical interference is sometimes indicated. Syphilitic forms (*q. v.*) may respond to specific treatment, but often recur or become permanent. In subacute or acute forms, counterirritation back of the ear by blisters, leeches or hot fomentations is indicated. Some find mercury useful in peripheral forms. Galvanism is the best form of electrotherapy, but may be painful and is seldom beneficial. Prisms and tenotomy are sometimes helpful.

Progressive Nuclear Ophthalmoplegia (Nuclear Palsy).—This disease was first described clinically by von Graefe (1856) and later pathologically by Hutchinson and Gowers. The etiology is not clear. Wernicke and Siemerling found syphilis causal in 17 per cent., which estimate is too low according to Hutchinson. Lead, alcohol, and toxæmia as from diabetes, grippe, or diphtheria, are possible causes. A few cases are hereditary or congenital. Most cases develop in males under thirty years of age. The *pathology* usually consists of degenerative nuclear atrophy resembling that of bulbar paralysis; sometimes slow compression or meningitis is found or much more rarely no anatomical alteration is detected; this is the so-called functional form.

Symptoms.—The symptoms are those of a slowly progressive paralysis of the eye muscles; ophthalmoplegia is called external when it involves the extrinsic muscles, internal when it involves the pupil, or total when both are concerned. The *eye muscles* are *successively affected,* but in no given or invariable order, until many or all of them are implicated. The expression, the *facies of Hutchinson,* is peculiar, being staring, from immobility of the eyeballs, or sleepy from the partial bilateral ptosis. Diplopia is quite uncommon, or at the most only an early symptom, because of the chronicity of the process. Associated muscles are often affected by groups, because of their successive nuclear involvement (*v. s.* ANATOMY); as the pupil and convergence, one internal and the opposite external rectus, the two internal recti, and the sup. rectus and the levator. Chronic ophthalmoplegia may be associated with the quite analogous *bulbar paralysis* or *progressive spinal muscular atrophy;* with *optic atrophy* or with *double facial paralysis* (the rare and obscure infantile or congenital oculofacial paralysis, without pupillary involvement, of which Möbius has collected 44 cases); it may be a part or a precursor of tabes, paralytic dementia, or multiple sclerosis.

Diagnosis.—Nuclear involvement is probable when there is (*a*) partial and bilateral ptosis; (*b*) no involvement of the pupil; or (*c*) involvement of one or more conjugate groups. The lesion may be supranuclear, when the head and limbs are involved, exceptionally when there is isolated convergence paralysis or paralysis of up-and-down movements with ptosis. Sudden apoplectiform nuclear palsy from basilar thrombosis is usually asymmetrical, acute and often associated with hemiplegia. Wernicke's acute superior polienceephalitis is likewise distinguished by its acuity, and toxæmic symptoms as delirium. Total or partial ophthalmoplegia may result from syphilitic meningitis, polyneuritis, tumors (corp. quadrigem.) or orbital disease, but they are generally distinguished by their relatively acute course and concomitant symptoms.

Prognosis.—The disease is very chronic, possibly lasting twenty to forty years and is usually progressive. Arrest is possible, but can never be predicted. The disease itself causes little risk to life, but its associates are dangerous, as tabes or psychoses, which develop in 18 per cent.

Treatment.—Treatment is generally ineffectual. Iodides and mercury are useless in nuclear disease, but may be given if the localization is not definitely known. Strychnia and galvanism usually produce little result.

Sympathetic Paralysis.—This disease was first described by Horner; it produces *myosis*, which is due to paralysis of the dilator iridis, and slight *ptosis*, due to paresis of Müller's muscle. The ocular tension is decreased, the cheek is wasted, and the skin is reddened and of higher temperature than normal. *Irritation of the sympathetic* is characterized by wide pupils, widening of the interpalpebral space and by exophthalmos.

Ocular muscular spasms occur in conjugate deviation, irritation of their nerve trunks when compressed by tumor, and rarely in chorea, facial tic, and spasmodic torticollis. In hysteria the eyes may be rolled upward and to one side, concealing the iris, or they are often fixed in convergence; they never diverge; there may appear to be ptosis, which is really spasm of the orbicularis. Convergent spasm may also occur in hypermetropia, and divergent spasm in myopia. Isolated spasm of the levator palpebræ may occur in irritation of the cervical sympathetic nerve, in advanced years, in Graves' disease, and in irritation of the fifth nerve. Secondary deviation (*v. s.*) is spasmodic, as is also *nystagmus*, which latter consists of rapid, rhythmic, clonic, involuntary oscillations, due to muscular spasm with alternating contraction of the opposing muscles. It is continuous, as distinguished from the "tremor of weakness" which occurs in paretic muscles on movement. It is usually horizontal, less often rotatory and but rarely vertical. Its physiology is not understood, but it is probably centric. The causes are (*a*) disease of the eye, as opacities and choroiditis; (*b*) it occurs very frequently in albinos, and (*c*) in miners, from working with the head down and eyes strained to one side. (*d*) It also occurs in numerous, usually degenerative nervous diseases; as a cardinal sign in multiple sclerosis and Friedreich's ataxia, as a common sign in tumor of the cerebellum, corp. quadrigem. and opt. thalamus, occasionally in meningeal inflammation or hemorrhage, sinus thrombosis, brain hemorrhage or softening, and very rarely in hysteria.

Fifth Nerve (Trigeminus; Trifacial Nerve).—The nerve originates from its motor nucleus in the pons, from fibers (which are probably motor) descending from beneath the corpora quadrigemina, and from sensory fibers ascending from the medulla. It is connected with the motor cortex and with the cerebellum. The motor and sensory fibers leave the pons separately and join only after the sensory trunk has passed through the Gasserian ganglion. The Gasserian ganglion has three branches; (1) which passes to the orbit by the sphenoidal fissure, supplying the skin of the forehead, anterior scalp, upper lid, bridge, and top of the nose; it is connected with the cervical sympathetic nerve; (2) which passes by the foramen ovale to the sphenomaxillary fossa to supply the upper and lower lid, cheek, fore part of the temples, side of the nose, upper teeth, gums, pharynx, tonsils, soft palate, uvula and roof of the mouth; it has connections with the facial nerve by the Vidian, and with the sympathetic nerve; (3) which passes by the foramen rotundum and sphenomaxillary fossa to the rest of the temples, anterior and upper part of the ear, auditory meatus, lower cheek near the mouth, lower lip, lower teeth, gums, chin, tongue, mucosa of the mouth, salivary glands, and as a motor nerve to the muscles of the lower jaw, temporals, masseters, pterygoids, mylohyoids, and post. belly of the digastric muscle. The lingual branch gives off the chorda tympani which joins the facial nerve. From the otic ganglion arises the small superficial petrosal nerve which connects with the facial nerve and ends in the tympanic branches of the glossopharyngeal nerve.

Causes of Paralysis.—(*a*) Lesions of the cortex may produce it in very rare cases. (*b*) Focal disease in the pons, especially of the nerve-root fibers, as hemorrhage, and tumors may cause it and much less often degenerative lesions as tabes or bulbar paralysis. (*c*) It may result from diseases of the base, as syphilis, meningitis, tumors, caries or aneurysms. (*d*) Affections involving any of the three branches, as of the first branch by cavernous thrombosis, pituitary tumor, aneurysm or orbital disease, or of the second and third branches by lesions in the sphenomaxillary fossa may cause paralysis. (*e*) Trauma, puncture or bullet wounds, but rarely fractures, and (*f*) infrequently neuritis, unless it is secondary, are causal.

Symptoms.—1. SENSORY PORTION.—Anæsthesia, which is generally unilateral, is found in one or all branches, and tactile sensation is more involved than the sense of pain. It is often preceded by tender-
along the nerves, irritation or neuralgia. In complete cases, the
and mucosa of the mouth, nose and conjunctiva are anæsthetic.
is furred on its anæsthetic half, because the food is not
conjunctival reflex is abolished. The
on or decreased by paralysis. Smell
ometimes there is redness, cyanosis
may be loosened. Two trophic
it. The first is neuroparalytic
le inflammation of the eye into
; it is due not simply to con-

junctival anæsthesia, which may lead to inflammation because of foreign particles remaining unnoticed in the eye, but is rather due to irritation of the fifth nerve, especially of the Gasserian ganglion or nerve trunks anterior to it. (It has not occurred, however, in many cases in which the ganglion has been surgically removed.) It is rare in lesions of the pons. The second is herpes zoster, which is most common in disease of the first branch, results from irritation of the Gasserian ganglion or the nerves just anterior to it, is often preceded by pain and hyperæsthesia, and sometimes leads to iritis or ophthalmitis. The relation of trigeminal disease to hemifacial atrophy will be considered below, under the latter head.

2. Motor Portion.—Placing the fingers on the temporal and masseter muscles, when the patient attempts to close the teeth, reveals their failure to contract. Paralysis of the ext. pterygoid is shown (*a*) by failure to move the jaw toward the sound side and (*b*) by deviation toward the paralyzed side, when the jaw is depressed. The muscles waste, but they are rather too deep to distinctly elicit the reaction of degeneration. The mylohyoid and digastric muscles cannot be separately tested and the role of the tensor tympani and tensor palati in trigeminal paralysis is still in dispute.

3. Gustatory Portion.—The sense of taste covers only bitter, sweet, sour, salty and metallic substances, while flavors are recognized by the olfactory nerve. The tip and edges of the tongue detect saltiness or sourness; the base of the tongue, bitterness and sweetness. It is often stated that the fifth nerve supplies the anterior two-thirds of the tongue and the glossopharyngeal nerve the posterior third and the palate; but disease of the glossopharyngeal nerve alone does not destroy the sense of taste. In a study of 26 cases of extirpation of the Gasserian ganglion, Cushing decides that taste is only temporarily lost and that it does not travel by the fifth nerve. Disease of the lingual nerve (from the fifth), after the chorda tympani joins it, produces loss of taste (*ageusia*); disease of the lingual above the juncture or disease of the central part of the fifth in the pons does not result in loss of taste. It is thought that the gustatory fibers of the fifth run with the chorda to the seventh nerve which they leave by the petrosal nerve to again reach the fifth through Meckel's ganglion. It is interesting to note that anæsthesia of the tongue may result from facial neuritis and disease of the middle ear.

Diagnosis.—The diagnosis is usually easy when there are symptoms in the motor or sensory nerves. Organic irritation characterized by pain alone may be difficult or impossible to differentiate from trifacial neuralgia, although Gowers holds that there is less radiation and more hyperæsthesia in organic irritation. The *localization* must be determined. When all branches are anæsthetic, the disease probably lies at the base of the brain or in the Gasserian ganglion. In disease of the middle part of the pons, the first division alone may be affected; in disease at its side all divisions are involved, often with implication of the third and sixth, or less often of the seventh and eighth nerves. Cortical lesions are extremely uncommon.

Treatment.—Counterirritation over the occiput or mastoid is indicated by an inflammatory causation, but no irritatant should be applied over the anæsthetic areas themselves, lest ulceration occur. Galvanization is said to be beneficial. Gelsemium, cocaine and morphine must at times be used because of severe pain, but the acquisition of drug habits must always be borne in mind. The eye should always be watched, and boric acid and castor oil be dropped in several times daily. Antisyphilitic remedies should be tried in every doubtful case.

Masticatory Spasm.—The masticatory spasm of Romberg is not common. In the *tonic type* (trismus) the spasm is usually bilateral, and the tense muscles are hard and may be tender. The cause may be tetanus, hysteria, epilepsy, tetany, tumor in the pons, cortical hemorrhage, or it may be reflex from trigeminal irritation as from dental caries. The *clonic type* is rare and may be serial or intermittent; chattering of the teeth is an example. It may occur in chorea, epilepsy or hysteria. Grinding of the teeth occurs in paretic dementia, meningitis or typhoidal states. In *diagnosis*, tumor, inflammatory irritation and disease of the temporomaxillary joint must be considered. *Treatment* consists of tonics in nervous subjects, galvanization, blisters in hysteria, and the local use of morphine and cocaine.

Progressive Hemifacial Atrophy.—This affection, first described by Parry (1825), is rare, only 135 cases having been reported up to 1900. Its pathogenesis is not clearly understood, but it is classified under disease of the fifth nerve because the only autopsy on record, by Mendel, showed neuritis of the ascending root of the fifth nerve. Möbius holds that acute infections, as measles or scarlatina, are causal, because some virus enters through the skin or tonsils and results in atrophy. The essential change is a *hemifacial atrophy*, which develops most often in persons between ten and fifteen years of age, in girls (66 per cent.), and in about 60 per cent. of cases affects the left half of the face. In perhaps a half-dozen cases the atrophy was bilateral. There may be premonitory pains, hyper- or paræsthesias, but the cardinal sign is *atrophy of the skin*, which may show initial infiltration. The skin is decolorized and shrunken, its *fat disappears*, and later in the disease the *muscles waste* and the rough skin lies directly on the bone. The hair on the affected side loses its pigment and may fall out, the eyebrows and beard especially, sweat secretion is lessened or absent, and the mouth, if involved, is drawn toward the side of lesion. The *bones* on the affected side *waste*, especially in young subjects, most notably the maxillæ, but even the smaller bones and nasal cartilages are involved. On the wasted side there may be no vasomotor reaction; sensation is usually normal, although pain or paræsthesia may be noted in the distribution of the trifacial nerve. The tongue, tonsils and palate are rarely involved. The course is *very chronic* and practically always progressive, although remission is possible. The outlook is hopeless and there is no treatment other than administration of thyroid extract and the injection of paraffin by the Gersuny method, to lessen the facial deformity.

Hemifacial hypertrophy, of which 13 cases only are on record, affects largely the cheek; Werner collected 9 congenital cases. Irritative neuritis is its suspected cause.

Seventh or Facial Nerve.—The face is represented in the lower Rolandic cortex, whence the fibers run through the corona, internal capsule, crus and pons (*q. v.*), where they decussate and enter the nucleus of the opposite side. The nucleus lies in the floor of the fourth ventricle inside of the ascending branch of the fifth nerve and receives fibers from the third nucleus above for the orbicularis of the eye (whereby ocular movements, winking and accommodation are coördinated) and fibers from the hypoglossus below for the orbicularis oris (whereby correlation of lingual and labial movements is effected). It curves around the sixth nucleus, emerging near the juncture of the pons and medulla, and enters the auditory meatus with the eighth nerve, bends sharply, and presents a ganglionic swelling which receives the large superficial petrosal from the Vidian nerve, containing fibers of taste from the fifth nerve. These latter again leave the facial nerve by the chorda tympani. In the Fallopian canal the facial nerve gives off branches as follows: (*a*) a motor branch to the tympanic plexus; (*b*) a motor twig to the stapedius muscle; (*c*) the chorda tympani nerve with secretory branches to the salivary glands. Passing through the stylomastoid foramen, it supplies (i) a few sensory branches to the external ear; (ii) motor fibers to the external auricular muscles; (iii) motor fibers to the posterior part of the occipitofrontalis, and in the parotid gland, (iv) breaks up into branches which supply all the muscles of the face, the platysma, stylohyoid and post. belly of the digastric.

Peripheral facial paralysis (mimetic facial paralysis, prosoplegia, Bell's paralysis, Bell, 1830) is the most frequent peripheral palsy.

Etiology.—1. *Neuritis* is the most common cause. Exposure to cold ("refrigeration," "rheumatism") produces 73 per cent. of cases especially in men between the twentieth and fortieth years, and produces a perineuritis which compresses the nerve trunk. Angina, gout, diabetes, diphtheria, typhoid, erysipelas, mumps, sepsis and other infections are occasional causes.

2. Disease of the petrous portion of the *temporal bone* (6 to 9 per cent.), especially caries and otitis media in childhood, may cause paralysis. The danger is usually proportionate to the extent of the bone disease, but infective matter may reach the nerve by way of the vessels. Eighty-three per cent. of cases of disease of the labyrinth cause facial paralysis.

3. *Trauma* causes 6 per cent. of cases, as paralysis which results from basal fracture by tearing, hemorrhage, inflammation or undue formation of callus; much more rarely from meningeal or cerebellar hemorrhage at birth; from the use of obstetrical forceps, and from

rauma to the jaw or surgical operations on the parotid gland. Facial paralysis may occur in "head tetanus" (E. Rose).

4. If it results from lesions *at the base*, it may be due to syphilis (3 per cent.), which should always be considered when the cause is intracranial; it is however, less common than the usual descriptions would ndicate; syphilis rarely causes a peripheral facial paralysis. Aneuysm, tumor, meningitis and hemorrhage are next in frequency and nearly always involve simultaneously the closely contiguous eighth nerve.

5. *In the pons*, the root fibers or nucleus may be affected, usually with the sixth nerve of the same, or the arm and leg of the opposite side. Degenerative affections, as tabes, bulbar paralysis, multiple sclerosis, and progressive muscular atrophy or acute poliencephalitis rarely invade the seventh nucleus.

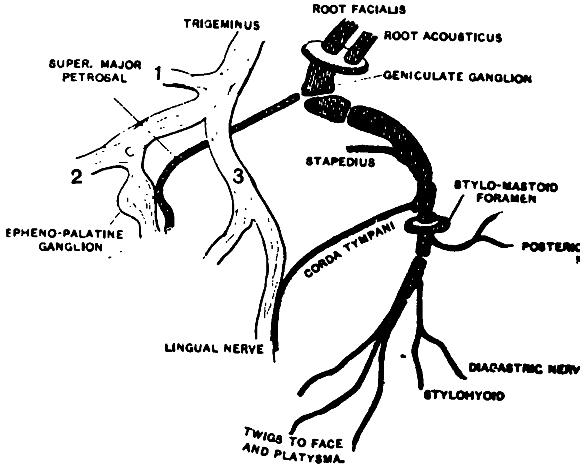
6. Other rarer factors are primary hemorrhage into the Fallopian canal, disease of the parotid or lymphatic glands, and leukæmic infiltration. It is suggested by Neumann that disease of the lymph vessels at the point of exit of the facial nerve may produce paralysis.

Pathology.—Few autopsies have been made on simple cases. The nerve has been found severed, its sheath and cylinders destroyed, the nuclei in Schwann's sheath increased, the connective tissue increased and the muscles the seat of fatty change. It is thought that the essential and usual change is a *perineuritis* and that the exudation in the sheath compresses the nerve fibers, which then undergo parenchymatous degeneration. In some cases degeneration only has been seen, and it is probable that slight swelling interferes with conduction. Facial neuritis differs from multiple neuritis in that alcoholism is a subordinate actor and the chief change is not in its peripheral twigs but in the trunk, usually within the Fallopian canal.

Symptoms.— The symptoms sometimes begin with prodromes as vertigo, headache, tinnitus, pain or more often twitchings in the face, ven fever or albuminuria, or sometimes without prodromes, in which ases one *half of the face* is rather suddenly paralyzed and becomes "*completely immobile and toneless*" within two days. The forehead is mooth, the lids cannot close (lagophthalmos), the tears run over the heek, and when a strong effort is made to close the eyes, the eye on he affected side is turned upward by the inf. oblique muscle (Bell's ign). The orbicularis at times escapes involvement, since it receives ome fibers from the third nerve. Smell is impaired because of absence of tears, the nasal aperture is smaller and cannot be dilated, sniffing on he affected side is impossible, and the nasolabial furrow is lost. The mouth is lower from paralysis of the levator anguli oris, it cannot be closed, and the saliva runs out, although its total secretion is often lessened (*v. i.*). Labial enunciation is imperfect, puckering the lips, as though for whistling, is impossible, and expectoration is impaired. The loss of wrinkles and expression on the affected side is more marked in adult and aged than in young subjects, especially about the eye, since years lessen the elasticity of the skin and the subcutaneous fat. Chew-

Taste is lost in disease of the nerve between the gen
and the offset of the chorda; it is lost in half the case
neuritis; it is not impaired in disease of the pons or in

Fig. 78.



Relations of the fifth and seventh nerves and branches of the se

the skull (see Diagram). The chorda supplies the ant
of the tongue and the glossopharyngeal the posterior th
taste (parageusia) results from irritation of Jacobson
tympanic plexus. *Hearing* may be impaired. Deafn
result mostly from coincident disease of the ear. Whe
is paralyzed, the unopposed tensor tympani (which
fifth nerve) overacts and results in aural oversensitiv

trunk, are sometimes observed; they are caused either by involvement of the fifth nerve, or possibly because the facial nerve carries some sensory filaments. Accidental sympathetic symptoms, as œdema or sweating, have been observed. Herpes is rare and indicates trifacial involvement. The saliva is decreased (the fibers for its secretion run with the chorda) and secretion of tears is lessened (the fibers from the facial nerve run to the fifth by the large petrosal nerve).

Diagnosis.—The diagnosis is made at a glance in fresh cases, while in older cases secondary spasm (*v. i.*) on the paralyzed side may at first cause confusion. The etiological diagnosis is important, as is also the topographical.

1. *Supranuclear paralysis*, paralysis in the upper neurone, from the cortex to the nucleus, is differentiated as follows:

CENTRAL OR SUPRANUCLEAR PALSY. ——— *vs.*	——— PERIPHERAL (NUCLEAR AND INFRANUCLEAR) PALSY.
Etiology; vascular disease, tumor, abscess.	Neuritis, ear disease, etc.
Distribution of paralysis: partial, lower facial involved or only slight or transient weakness of orbicularis palpebrarum.	*Complete:* orbicularis and occipito-frontalis involved. When eyes cannot be closed, probably peripheral or nuclear.
Reflexes persist and increased.	Lost. Paralysis flaccid.
Taste, auditory, salivary and sensory symptoms lacking.	Often present.
Pilocarpin increases salivary flow.	Does not increase it.
Voluntary motion lost, while emotional movement very often persists.	Both lost.
Degenerative reaction and wasting absent.	Present.
Cerebral symptoms present, general as headache, vertigo, delirium, increased reflexes or focal as cortical epilepsy, hemiplegia, usually weak tongue, alternating paralysis (see crus and pons localization).	Absent.

In lesions of the pons, all parts of the facial nerve are affected, wherein it differs from facial nerve disease in the internal capsule. In nuclear disease, the orbicularis oris is less involved, or escapes affection, its centre being in the hypoglossus nucleus.

2. *Localization in the peripheral type* (see Fig. 78) may be (*a*) in the *pons* (*q. v.*). General disease of the posterior pons also affects the sixth nerve about whose nucleus the seventh nerve curves. (*b*) In localization *at the base*, taste is not involved and deafness results from coincident disease of the eighth nerve. (*c*) It may be in the *Fallopian canal*, when taste is impaired, the salivary flow decreased,

and hearing often abnormally acute. (*d*) It may be *external to the skull* and then there is no involvement of taste, salivary flow, or the stapedius.

Double facial paralysis is rare and usually indicates disease within the skull. It may occur in bulbar paralysis, in disease of both sides of the pons, or one focus in its centre, in double otitis, especially syphilis, multiple neuritis, tetanus (of which J. H. Lloyd in 1905 collected 7 cases), or in a form observed in childhood with ocular paralysis (*q.v.*). Hysterical facial paralysis is extremely infrequent; a case is reported in which organic facial paralysis existed on one side and an hysterical paralysis on the other.

Course and Prognosis.—The onset is rapid and the disease is usually manifest during the first day. Its duration varies from a week to over a year, two or three months for moderate, and six to eight (eighteen) months for severe cases. Even in cases which have seemingly recovered and in those lasting over a month, some slight vestige of paralysis usually remains. The *prognosis* depends (1) on the cause; it is favorable in syphilis, birth trauma, and polyneuritis; in ear disease it is better if the drum has not ruptured; it is unfavorable in tumor or nuclear disease; (2) it depends on the electrical reaction. If after ten days, the irritability is not reduced, the outlook is good and recovery may be expected in about three weeks; if after fourteen days it is lowered but not lost, recovery occurs within two months; if after fourteen days the irritability is lost, the prognosis is doubtful and the course is likely to cover months. Recurrence is infrequent, although five attacks are recorded. *Contracture* (secondary overaction) usually occurs in four to six months and progresses for eight to twelve months, when it lessens, and disappears in mild but remains to some extent in severe cases. In old subjects it balances up the facial contour; in young subjects it produces deformity. It is thought to depend on an altered functional state of the nucleus.

Treatment.—(*a*) The *cause* should be reached, if possible; for example, antisyphilitic remedies and ear-drainage have cured cases even of twenty-three years' duration. (*b*) *Antirheumatic* drugs are inefficacious, and the salicylates do not usually shorten the course. A mercurial purge, hot fomentations, which must be carefully maintained for two or three days, keeping the patient indoors, and the application (invariably) of a blister over the mastoid and one over the side of the neck are beneficial. (*c*) *Correction of deformity* is always necessary. The eye should be douched with boric acid, bandaged closed during sleep and the lids massaged, the cheek and mouth should be massaged upward, and the placing of a hook in the angle of the mouth (sustained by tape over the ear or by adhesive plaster) prevents deformity to some extent. At night the eye should be closed by a light compress. (*d*) *Electricity* should not be used for two or three weeks, and then the galvanic current should be given, for fifteen minutes each day, in strength just sufficient to produce muscular contraction; the application of the anode over the muscles is the least painful method. The faradic current causes pain. (*e*) For *contracture* little can be done, excepting massage and steaming the face. (*f*) In irreparable cases, surgical transplantation of the facial trunk to the spinal accessory nerve has been successfully performed. (Spiller thinks anastomosis with the hypoglossus produces better results.)

Facial and Mimetic Spasm (Tic Convulsif).

Etiology.—The causes are (*a*) organic disease in the cortex, pons or nerve trunk, which irritates (perhaps later paralyzes) the centre, nucleus or paths. Irritation of the fifth nerve is considered causal. (*b*) There is an idiopathic form, which occurs in persons between the fortieth and sixtieth years, most often in women with neuropathic or emotional tendencies (see HABIT SPASM under CHOREA). There is also a reflex form which results from worms or uterine disease.

Symptoms.—The spasm begins paroxysmally on one side and is more often clonic than tonic. The *clonic form* affects the orbicularis palpebrarum (blepharospasm) and zygomatici most frequently, although the frontalis, chin depressors or all the facial muscles may be concerned in severe cases. Pain and paresis are absent save in progressive organic disease. The spasm is lessened by rest, and is increased by cold or emotion. It is usualy unilateral, but may spread to the opposite side or exceptionally to the muscles of mastication, or those of the tongue, pharynx, larynx, neck or arm. The *tonic form* is seen in photophobic disease of the eye, in paralysis agitans, tetanus, tetany or hysteria. The *prognosis* is unfavorable after the first few months, and the disease is then likely to last for years or for the rest of the patient's life.

Treatment.—Treatment is often useless, and varies with the cause of the affection. Trigeminal irritation should be removed if possible. Arsenic has proved beneficial in some cases, but nervines and antispasmodics are generally useless. Galvanization, especially if tender points are found, has cured a few cases (Berger). Wier Mitchell has recommended freezing of the cheek with ethyl chloride. Stretching of the facial nerve usually affords relief, which is but temporary, although a few cures are on record.

The Eighth or Auditory Nerve.—The eighth nerve in the old nomenclature was called the portio mollis of the seventh pair, the facial nerve being the portio dura; as the names imply, the auditory is softer than the facial nerve and therefore less resistant to pressure. It is purely sensory and consists of two distinct roots, the cochlear and vestibular. At the point where their roots separate is found a group of cells, analogous to the spinal ganglia, from which arises Wrisberg's nerve, an accessory structure with vasomotor and salivary secreting fibers.

The *cochlear nerve* is the true auditory nerve and its nuclei are found in the floor of the fourth ventricle. It is connected through the tegnot absolute, indicate disease of the *cochlear nerve.* Nystagmus, double vision and ataxia are occasional symptoms, and result from the relations between the labyrinth, cerebellum and cerebrum. The *prognosis* is always uncertain. Acute or progressive disease is unfavorable. The vertigo may cease when complete destruction of the nerve results in deafness. Some cases recover.

Diagnosis.—Aural vertigo constitutes 90 per cent. of vertigo. Differentiation is as follows: (*a*) *Gastric disease* may cause vomiting or vertigo (*a stomacho læso, Trousseau*) but does not produce tinnitus nor deafness. Gastric vertigo was once thought to cause 80 per cent. of cases of vertigo; now, but 5 per cent. (Gowers) is attributed to it. Many of these cases are neurotic. (*b*) *Epilepsy*, especially *petit mal*, may produce vertigo, very rarely tinnitus or deafness, and much oftener loss of consciousness. (*c*) *Cardiac disease*, as aortic regurgitation or arteriosclerosis, may cause vertigo but not the other symptoms. (*d*) *Organic brain affections*, as tumor, have other distinctive signs, as headache, or focal lesions. Confusion is possible with arteriosclerotic vertigo, and especially with coincident accidental deafness. (*e*) *Ocular vertigo* ceases on closing the eyes. Gerlier (1887) described a form which is endemic in France and Switzerland, and is characterized by vertigo, great depression, paretic weakness of the neck and limbs, ptosis, strabismus, diplopia and retinal hyperæmia, hemorrhage or œdema. A similar form occurs in Japan. Recovery occurs in one to four months.

Treatment.—Labyrinthine disease is little influenced by drugs, except when syphilis and gout are its causes. Quinine, salicylates and nitroglycerine should be given (see TINNITUS). *Bromides* are most useful in daily doses of one dram. Galvanization with the anode over the ear and cathode to the neck has seemingly helped some cases, as has blistering of the mastoid. Disease of the pharynx, Eustachian tube, and accumulation of wax must be recognized and treated. Removal of the malleus and incus with mobilization of the stapes relieves a few cases.

Ninth or Glossopharyngeal Nerve.—This nerve is almost inseparably connected in its course and functions with the vagus and the internal part of the spinal accessory nerve; their nuclei blend, their sensory and motor filaments supply the digestive tract from the spinal accessory nerve, and send motor branches to the lungs, larynx and heart. Compared with disease of the first eight cranial nerves, affections of these visceral nerves are infrequent. Of all the cranial nerves, the ninth is least understood and is almost never the seat of isolated disease. The causes of its disease are essentially the same as those of the vagus (*v. i.*); its nuclei suffer in bulbar palsy and its roots in basal disease.

It is distributed as follows: (*a*) its sensory fibers supply the back of the tongue, pharynx, soft palate, tonsil, upper pharynx, Eustachian tube and tympanic cavity. It is probably the nerve of nausea. (*b*)

While nothing is usually heard on examination, in children or less often in adults we may hear a systolic brain murmur (see BRAIN TUMOR). *Treatment* depends on the cause. Bromides are most useful when combined with a few drops of the tr. belladonna or cannabis indica. Quinine was recommended by Charcot and Politzer and salicylates by Gowers, but they seldom give relief. Full doses of nitroglycerine relieve certain cases and counterirritation over the mastoid sometimes mitigates tinnitus.

The vestibular nerve arises from the medulla, supplies the vestibule and semicircular canals, subserves sensation of space and equilibration, and has cerebral and cerebellar connections.

Ménière's Disease, Auditory Vertigo, *Vertigo ab aure læsa.*—Paul Ménière in 1861 described a condition consisting of vertigo, tinnitus, vomiting and sometimes deafness, developing with apoplectiform suddenness, and due to acute labyrinthine disease. The lesion is usually a chronic, slightly irritative disease of the vestibular nerve, which supplies the organs of equilibration, the semicircular canals. According to Gowers 80 per cent. occurs in persons between the thirtieth and sixtieth year, 66 per cent. in males, and 90 per cent. of all cases of vertigo not due to epilepsy or organic brain disease are caused by lesions of the labyrinth. Fränkl-Hochwart thus classifies Ménière's complex, which may be elaborated as follows: (*a*) *Apoplectiform cases*, which occur in healthy ears, of which few more than 30 cases are recorded, due to labyrinthine hemorrhage, or to trauma; leukæmia and arteriosclerosis are promoting factors and it is thought that acute angioneurotic transudation of lymph may occur in the canals. (*b*) Cases developing in *ears already acutely or chronically diseased*; the internal ear is the usual seat of disease, but the external or middle ear may be affected, or the branches of the eighth nerve, perhaps by tumors, syphilis, tabes, paretic dementia, gout, cold, facial paralysis or drugs, such as quinine and the salicylates. (*c*) Cases caused by *external influences* or procedures, as pressure on the drum by violent douching of the ear; (*d*) *pseudo-Ménière's disease*, which occurs in the neuroses, as epilepsy, hysteria, neurasthenia migraine, or sea-sickness.

Symptoms.—The symptoms begin gradually or with a sudden stroke, and are paroxysmal; the attacks may be separated by weeks or months or perhaps occur daily. (*a*) The *vertigo* is subjective or objective, perhaps combined; the patient cannot stand; he is likely to fall backward, or, if standing is possible, is ataxic. These symptoms indicate lesion in the *vestibular nerve*; there is occasionally momentary loss of consciousness; some slight vertigo may persist between paroxysms. (*b*) *Nausea and vomiting* are usual, save in the lighter forms, and are often attended by headache, sometimes collapse, pallor and clammy skin, and rarely by death. (*c*) The *tinnitus* of a roaring, throbbing character and (*d*) *nervous deafness*, which is usually not absolute, indicate disease of the *cochlear nerve*. Nystagmus, double vision and ataxia are occasional symptoms, and result from the relations between the labyrinth, cerebellum and cerebrum. The *prognosis* is always uncertain. Acute or progressive disease is unfavorable. The vertigo may cease when complete destruction of the nerve results in deafness. Some cases recover.

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It is distributed as follows: (*a*) its sensory fibers supply the back of the tongue, pharynx, soft palate, tonsil, upper pharynx, Eustachian tube and tympanic cavity. It is probably the nerve of nausea. (*b*)

Its motor fibers supply the upper pharynx and possibly the palate. (*c*) Taste fibers are usually thought to be distributed to the posterior third of the tongue and palate, but they are not found in the nerve root and are considered to pass through the fifth nerve to the brain. Increased flow of saliva and loss of taste on the root of the tongue may result from disease of the middle ear due to relations between the nerve of Jacobson (ninth) and the branches of the fifth nerve. The nerve is thought to regulate and inhibit the contractions of the pharynx, which become frequent (spasm) in disease or on section of the nerve.

Tenth Nerve or Vagus.—This nerve arises in the medulla above the ninth, below the eleventh, and outside of the twelfth nuclei. With the internal part of the accessory nerve it has an enormously diffuse and important visceral distribution. It is a mixed nerve, the motor fibers originating from the nuclei, and the sensory fibers from the jugular and plexiform ganglia. It is generally maintained that the vagus carries the sensory and the accessory, motor fibers.

Etiology.—Affections of the vagus result from: (*a*) Nuclear disease, as degenerative bulbar palsy or acute vascular lesions, which usually also involve other nuclei; (*b*) involvement of the root and trunk by meningitis, syphilis, caries, trauma, tumor, aneurysm of the arch or its branches, carcinoma, œsophageal growths, tuberculous or other adenopathies, pleural (right-sided especially) or pericardial effusions or adhesions, mediastinitis, goitres, operations on the neck (laryngeal paralysis occurs in 14 per cent. of thyroid extirpations) or rarely neuroma and neuritis from diphtheria or other infections, tabes, or plumbism. Cortical, especially functional, causes may occasion symptoms. Irritative and paralytic manifestations occur in numerous combinations and will be treated topographically.

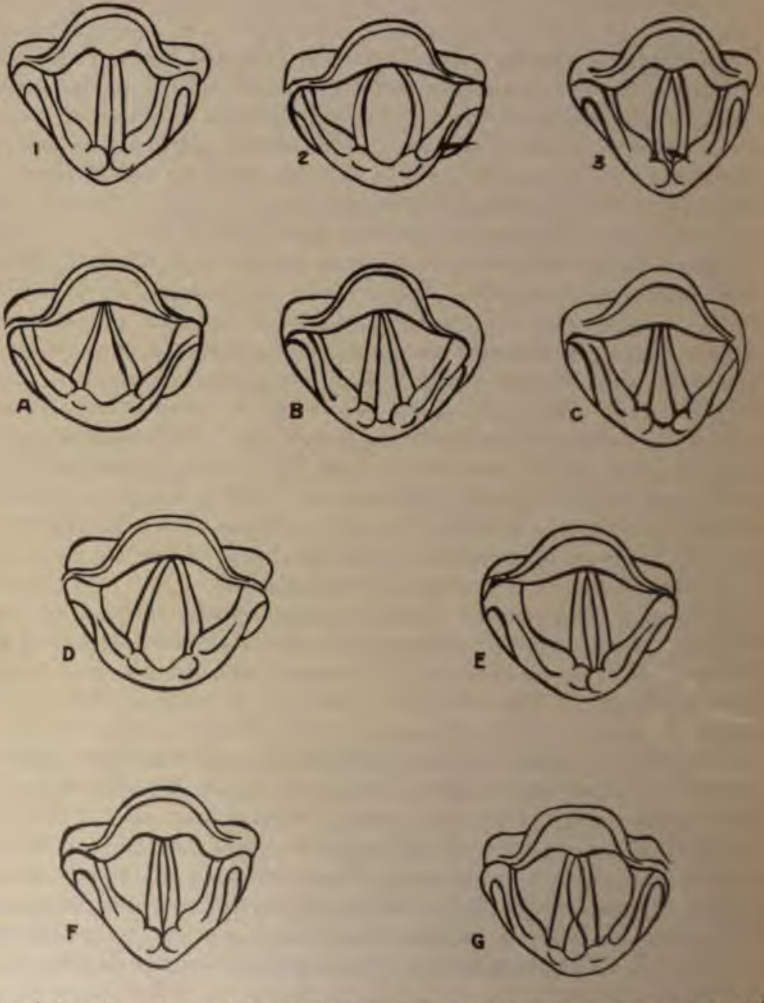
PHARYNGEAL BRANCHES.—The pharyngeal plexus consists of branches from the ninth and tenth nerves. In *paralysis*, sensation and the pharyngeal reflex are lost, while bilateral (rarely unilateral) disease causes dysphagia. The stomach tube can be introduced without sensation or spasm. Pulpy foods are better swallowed than dry foods or fluids. Food is likely to enter the larynx, and when the palate is involved there is regurgitation into the nose, and nasal speech results. The innervation of the palate is not definitely known, but it is probably supplied by the accessory nerve. *Spasm* is not frequent, is always functional, is transient or recurrent, does not allow of introduction of the stomach tube, and includes the globus hystericus, œsophagismus, pharyngismus, false and genuine hydrophobia and the pharyngeal crises of tabes. The œsophagus is rarely affected except in diseases of the centres or trunk.

LARYNGEAL BRANCHES.—There are two laryngeal nerves, the superior laryngeal and the recurrent laryngeal.

The *superior laryngeal* is (*a*) the sensory nerve for all parts above the vocal cords, (*b*) the motor nerve for but one larynx muscle, the cricothyroid, which modulates the voice by regulating tension in the cords, and (*c*) it is the motor nerve for the depressors of the epiglottis, the thyro-epiglottic and ary-epiglottic muscles, paralysis of which causes the epiglottis to stand upright, thereby risking entrance of food into the larynx and aspiration pneumonia. (Some maintain that the recurrent nerve supplies these depressors.)

Figures illustrating the laryngeal paralyses, from Church's *Nervous Diseases.*

Fig. 79.



1, normal phonation; 2, normal deep inspiration; 3, normal cadaveric position; A, bilateral adductor palsy; attempted phonation; B; Left adduction palsy; attempted phonation; C, bilateral abductor paralysis; deep inspiration; D, left abductor paralysis; deep inspiration; affected cord in cadaveric position; E, left abductor paralysis; phonation; affected cord in cadaveric position; right cord crossing medium line; F, bilateral thyro-arytenoid paralysis; G, bilateral thyro- arytenoid paralysis and paralysis of arytenoideus, giving an hour-glass opening.

The *recurrent laryngeal* curves around the aortic arch on the left and the subclavian artery on the right side; (*a*) it is the sensory nerve to the mucosa of the larynx below the cords and to the trachea and (*b*) is the motor nerve to every intrinsic muscle of the larynx except the cricothyroid. The larynx muscles are physiologically complicated, but they have three main movements: (*a*) to render the cords tense (this is accomplished by the cricothyroids and to a less extent by the thyro-arytenoids); (*b*) to adduct the cords, which is done chiefly by the lateral crico-arytenoids (also by the thyro-arytenoids and arytenoids); (*c*) to abduct the cords, which is done by the post. crico-arytenoid; it is especially important because it is the only special abductor; it is very frequently and almost invariably first involved in incomplete recurrent paralysis (Rosenbach and Semon), even though the adductors are affected later in complete paralysis.

Treatment depends wholly on the cause, which is usually peripheral. In nuclear types there is no therapy. Suggestion, or its equivalent, electrotherapy, is valuable in hysterical forms. The faradic current may be used, with the positive pole to the forehead and the negative to the larynx externally or internally; should this fail, the galvanic current may be given. Other measures include laryngeal gymnastics, in which the patient repeatedly attempts to speak, intralaryngeal insufflations of alum, other topical applications, the administration of strychnia hypodermatically, and Ollivier's procedure, in which the thyroid cartilage is compressed between the index-finger and thumb during inspiration and expiration, while the subject attempts to phonate. Tracheotomy and feeding by the nasal catheter are sometimes indicated.

Anæsthesia of the larynx is rare but may occur uni- or bilaterally in disease of the sup. laryngeal nerve, the vagus roots, its nucleus or in the hemianæsthesia of hysteria or organic brain disease. In nuclear disease it is partial; in affections of the nucleus, root and trunk the reflexes are lost, but they are present in supranuclear and functional affections. *Hyperæsthesia* is even rarer, being observed in neurotics as a tickling, tendency to cough or spasm; neuralgia and crises also occur.

The most common form of *laryngeal spasm* is spasm of the adductors (which are especially concerned in reflexes inimical to entrance of foreign bodies into the larynx); it may result from centric, local (laryngitis with croup-like cough at night) or reflex irritation from distant or neighboring foci as elongated uvula, goitre or hypertrophy of the pharyngeal tonsil. It includes *laryngismus stridulus,* which occurs in children under three years of age, especially in boys, and was previously referred to the thymus gland, but is due to rickets in 66 per cent. of cases and also to tetany, and reflexly to alimentary disorders; it comes on at night or on awakening in the morning, with croup-like cough, and presents all the symptoms of acute suffocation, as inspiratory and expiratory stridor, pallor, cyanosis, wide pupils, protruding eyes, low diaphragm, involuntary evacuations, centric vasomotor and

TABLE FOR DIFFERENTIATION OF LARYNGEAL PARALYSIS, (elaborated from Gowers and Archibald Church.)

Form of Paralysis.	Nerve and Muscles Affected: Function.	Causes.	Symptoms.	Signs by Laryngoscopy.
Total bilateral palsy.	Recurrent nerves; all muscles involved or all save cricothyroids.	Nuclear disease, disease of trunk or of recurrent nerve.	Voice lost; cough impossible; stridor only on deep inspiration.	Both cords moderately abducted, motionless in cadaveric position, neither receding on deep inspiration nor approximating in phonation. FIG. 3.
Total unilateral palsy.	Recurrent nerve: ditto.	Ditto. (In general, paralysis of one cord always suggests pressure on the recurrent).	Voice low-pitched and hoarse: cough impossible: stridor absent or only on deep inspiration.	Paralyzed cord as above: sound cord moving freely, even beyond median line.
Bilateral abductor palsy.	Recurrent nerves: both posterior crico-arytenoids, abductors.	Central disease, toxæmia, pressure or disease of trunk or recurrent nvs. (In general, marked bilateral cord paralysis always suggests centric disease.)	Voice little changed: cough possible: extreme inspiratory stridor, long inspiration, marked laryngeal excursion, cyanosis, even death; expiration free whereby differentiate from bronchial stenosis even without laryngoscopy.	Glottis is chink-like, cords near together, not separated on inspiration but even drawn closer by unopposed adductors. FIG. C.
Unilateral abductor palsy.	Recurrent: one post. crico-arytenoid.	Same; especially aneurysm, injury, etc., to one recurrent laryngeal nerve.	Symptoms inconclusive, little affection of voice or power to cough.	The same, but unilateral. FIG. D and E.
Bilateral adductor palsy.	Recurrent: both lateral crico-arytenoids and arytenoids; adductor muscles.	Hysteria (women under thirty years), overuse of voice, catarrh of larynx, anæmia.	Voice lost, but coughing, laughing even singing are possible, hence called phonetic paralysis by Türck; no stridor or dyspnœa; often with anæsthesia of larynx and pharynx (hysteria), etc., hysterical aphonia.	Cords normal in position but not brought together on phonation: normal movement on breathing. FIG. A.
Unilateral adductor palsy.	Recurrent: one lat. crico-arytenoid and arytenoid.	Toxæmia, lead, diphtheria, cerebral disease, syphilis, tuberculosis.	Voice hoarse: coughing or laughing imperfect; sometime dysphagia.	Ditto: but unilateral. FIG. B.
Paralysis of cords proper.	Recurrent: thyro-arytenoids; tensors and adductors of cords.	Very frequently involved from overuse; hysteria; catarrh.	Loss falsetto notes; uncertainty of phonation; often with adductor or arytenoid paresis.	Cords atonic and concave. FIG. F. When thyro-arytenoids and arytenoids are involved, the space between cords is hour-glass in form.
Bilateral paralysis of tensors.	Superior laryngeal nerve: crico-thyroids, the tensors and modulators of cords.	Cold, straining of voice, diphtheria.	Hoarseness: high notes imperfectly taken; voice weak.	Cord higher on paralyzed side, but hard to see with mirror.

cardiac disturbance and convulsions either generalized or of the infantile carpopedal type. The attacks last from a few seconds to one-half or one minute and are sometimes attributed to loss of temper when they follow a reproof. In the adult, laryngeal spasm occurs in tabes, hysteria, tetany, hydrophobia, epilepsy (the initial cry of which arises from spasm), asthma or migraine. The attacks are recognized by the negative laryngoscopic findings, paroxysmal occurrence, and the stridor being manifest in both inspiration and expiration. Partial spasm occurs in some types of stuttering, in clergymen's sore throat, and in explosive, unmodulated speech.

Nervous cough is present in the explosive hysterical type, in the metallic ovarian form in young girls, and in the barking variety in young masturbators.

Treatment of spasm includes removal of the cause, relief of local rritation, as affections of the ear (which has branches from the vagus) or pharynx, correction of hygiene, administration of tonics, as cod-iver oil in children, and such immediate measures as thrusting a finger nto the throat, dashing water in the face, hydrotherapy, and for the attack chloral and bromides by enema, local application of cocaine, nhalation of chloroform and in adults nitrite of amyl and morphine.

Pulmonary Branches.—The vagus and the sympathetic nerve orm the pulmonary plexus. Afferent fibers convey sensory impres-ion to the respiratory centre; these include accelerating fibers which redominate, and inhibitory fibers, stimulation of which (sup. laryn-eal nerve) arrests breathing. Afferent impulses are also carried to he vasomotor centre. Efferent motor impulses are conveyed to the ronchi. Division of the vagus causes rapid congestion of the lungs ith hemorrhage (also observed in acute lesion of the pons) and ower but deeper breathing; stimulation of the divided end of the erve accelerates breathing even to its tetanic arrest. Bronchoneumonic foci are as yet thought to be trophic but should be condered aspiration pneumonia from paralysis of the palate and larynx. he pulmonary twigs are probably concerned in Cheyne-Stokes reathing, bronchial asthma and hiccough.

CARDIAC BRANCHES.—The vagus and sympathetic nerves constitute e cardiac plexus. (*a*) Motor fibers from the spinal accessory nerve hibit and coördinate cardiac action, which is sometimes under the ntrol of the will; the heart may at times be slowed by pressure on e neck; marked irritation of the vagus slows the heart (bradycardia) r even arrests its action. Division may cause sudden death, though is is unusual in unilateral lesions. Paralysis of these fibers allows e sympathetic accelerator fibers to dominate, producing arrhythmia nd tachycardia. Digitalis is naturally ineffective in paralysis of the agus. (*b*) The sensory fibers are concerned in pain and palpitation see CARDIAC NEUROSES). (*c*) These are probably trophic fibers, as Eichhorst's experiments show fatty myocardial degeneration on division of the vagi.

GASTRIC BRANCHES.—(*a*) Motor branches supply part of the stomach and are concerned in reflex vomiting; they cause nausea and are concerned in direct or centric vomiting, as in brain lesions, wherein the vomiting usually occurs without nausea. (*b*) There are sensory branches; the vagus is seemingly the nerve of hunger and thirst, and its disease often but not always causes anorexia and loss of thirst. Centric disease causes the gastric crises of tabes, and cortical functional disease produces the epigastric aura of epilepsy and perhaps gastralgia in the neuroses. Vertigo probably causes vomiting through the vagus. Intestinal and splenic symptoms are lacking in affections of the vagus, although the nerve somewhat accelerates peristalsis.

Eleventh or Spinal Accessory Nerve (external portion).—The internal and minor part runs with the vagus, while the external and major segment is really a spinal motor nerve, arising from the lateral portion and ant. horns of the cord between the sixth cervical segment and the medulla. It courses upward through the foramen magnum into the cranium and leaves it with some of the true cranial nerves. It is the chief or sole supply of the sternomastoid muscles and the upper part of the trapezius, the lower two parts of which are supplied by the cervical and upper dorsal nerves. Rarely the eleventh supplies the whole trapezius muscle.

Paralysis is rarely caused by cortical disease, because the muscles have bilateral representation. The usual causes are bulbar paralysis, lesions near the foramen magnum, meningitis (then usually with involvement of the inner accessory branches and the twelfth nerve), syphilis, trauma, disease of the cord (tabes, syringomyelia, progressive muscular atrophy), of the spine (caries, tumor), or disease of the nerve trunks, as cold, trauma or neuritis. The symptoms are paralysis of (*a*) the sternomastoid, in which there is impaired rotation of the face to the side opposite the lesion; in old cases contracture turns the face toward the paralyzed side. (*b*) That part of the trapezius from the occiput to the acromion is paralyzed, showing a concave instead of a straight line, which is best seen in deep inspiration or on shrugging the shoulders. (Paralysis of the second portion causes imperfect elevation of the arm, some drooping and winging out of the scapula, the lower part of which is drawn toward the spine by the rhomboids and lev. anguli scapulæ; paralysis of the third portion results in weakness in the adduction of the scapula to the spine.) If it is bilateral as in chronic meningitis of childhood or progressive muscular atrophy, the head falls forward or backward as the sternomastoids or trapezii, respectively, are affected. In peripheral disease there is wasting and the reaction of degeneration. In centric disease the electrical reactions vary, as in progressive muscular atrophy.

Accessory Spasm, Spasmodic Torticollis or Wryneck.—**Etiology.**—It occurs in women in 66 per cent. of cases (Gowers), although Osler finds it largely in males, between 30 and 50 years of age. A neurotic tendency, bad health, trauma, overexertion, acute infections, alcoholism, plumbism, cervical caries, brain tumor, and diseases of the eye and ear (torticollis ab oculo læso, ab aure læsa) are among its uncertain causes.

Symptoms.—The symptoms vary as the affection is clonic or tonic, unilateral or bilateral, and also with the number of muscles involved. One sternomastoid is generally involved, especially in tonic spasm, in which the occiput is turned to the diseased side, the face to the opposite side and somewhat upward, and the ear brought nearer the clavicle. The upper part of the trapezius comes next in frequency of involvement, and is affected equally by clonic and tonic spasm. The affected shoulder is raised and slightly rotated, the scapula drawn toward the spine and the head backward. The spine is often convex toward the diseased side. The splenius is half as frequently affected (Gowers), the head being drawn backward, the face a little to the affected side and upward. Less often the scalenus, platysma, rectus and obliquus capitis are affected. The muscles in tonic spasm are hard, prominent and sometimes hypertrophied. Clonic spasm disappears during sleep, but is increased by fatigue and emotions; it may also invade the face, neck, eyeballs, vocal cords or the arm. There may be pain, fatigue and rarely tender spots or formication from compression of the brachial plexus by the scaleni. Bilateral clonic or tonic spasm of the trapezius draws the head backward (retrocollic spasm), and always involves the frontalis muscle.

Course and Prognosis.—The onset is gradual, the disease is usually stationary or progressive in its course, rarely recurrent; recovery is infrequent. The outlook is generally grave in well developed types.

Diagnosis.—(*a*) In *congenital or fixed wryneck* there is no active spasm. One sternomastoid (but rarely the trapezius) shows atrophy, induration and cicatrization, which Stromeyer considers due to traction on the neck with injury during delivery. Seventy per cent. of cases occur in left occipito-anterior presentations. The disease escapes notice for years, because of the shortness of the child's neck. Wilks always found facial asymmetry. Golding-Bird considers the affection a contracture from a central lesion. Tenotomy cures it. (*b*) *Hysterical torticollis* occurs in subjects under thirty years of age; other stigmata usually develop. Bompaire and Brissaud describe a *mental torticollis*, which is clearly a cortical or psychical phenomenon. (*c*) Torticollis may occur *in children* from dentition to puberty; clonic spasms, especially in the deep muscles, lead to nodding, salaam movements, spasmus nutans, nictitatio spastica. The eyes may show nystagmus. The affection may develop from reflex dental or digestive disorders, or from organic cerebral disease. It is sometimes habit spasm, hysteria, or even part of an epilepsy or idiocy.

Treatment.—Treatment is first etiological. Nervines, sedatives and electricity seldom produce permanent results. Morphine is a dangerous agent, although its local hypodermatic use has led to recovery. Massage and deep compression are occasionally helpful.

In chronic cases resection of the accessory nerve is indicated. If many muscles are involved, the third and fourth cervical nerves must also be resected. Successful results are reported by W. W. Keen and Noble Smith.

Twelfth or Hypoglossal Nerve.—This is purely a motor nerve. *Paralysis of the tongue is (a) rarely cortical;* supranuclear glossoplegia from lesions between its centre in the lower part of the ant. ascending and the third frontal convolutions, almost always occurs with hemiplegia and paralysis of the lower facial fibers; there is no wasting or reaction of degeneration. The root of the tongue is higher (hyoglossus paralysis) and on protrusion the tongue deviates to the paralyzed side, being pushed over by the sound genioglossus. (*b*) *Nuclear paralysis* occurs often in degenerative disease as bulbar paralysis, sometimes in tabes or paretic dementia, and much less frequently in acute inflammation or softening in the medulla. Nuclear lesions are almost invariably bilateral (since the nuclei are closely contiguous), and show the reaction of degeneration and wasting, the mucosa being wrinkled over the atrophic half. Wasting may be more marked than paralysis. Fibrillation is frequent, as in paretic dementia, from cortical and nuclear changes. In bilateral glossoplegia the tongue cannot be protruded, there is impairment of the first act of swallowing, in chewing and articulation, all of which are but slightly marked in unilateral paralysis. (*c*) *Infranuclear paralysis* results from lesions near the surface of the medulla, which may produce unilateral paralysis of the tongue, palate and vocal cord. Contralateral hemiplegia may occur. The lesion may affect one or both nerves. Syphilis, trauma or tumors are causal; neuritis is infrequent (there being recorded only 40 cases of peripheral, isolated hypoglossal paralysis, Panski, 1903). Wasting, the degenerative reaction and fibrillation are present. *Treatment* is that of other paralyses.

Spasm is rare either in the tonic or clonic form. It occurs in chorea, facial spasm, certain types of stuttering, epilepsy (in which the tongue, protruded by the genioglossus, is bitten), hysteria (in which the tongue is rarely bitten), certain psychoses, meningitis, neurotic spasm in speakers (aphthongia) or musicians. The prognosis varies with the cause and in general is favorable.

5. Diseases of the Spinal Nerves.—The spinal nerves differ from the cerebral in that they contain sensory, motor and vasomotor fibers Disease or injury therefore suspends motility and sensation, causes vasomotor paralysis, trophic changes or wasting, glossy skin, the reaction of degeneration, atrophy and contracture.

Phrenic Nerve.—PARALYSIS.—(*a*) Disease, or trauma of the vertebræ, cord, membranes or of the third, fourth and fifth cervical nerves of

in may cause it. In disease of the cord other paralysis is always
ent; phrenic paralysis is usually bilateral. (*b*) Its trunk is well
ected but may be injured in its course or damaged by aneurysm,
ors, or pleurisy. Neuritis is a rare cause, although it is sometimes
lved in multiple neuritis or degenerative lesions as tabes or pro-
sive muscular atrophy.

ymptoms.—The diaphragm is paralyzed on one or both sides.
abdomen retracts in inspiration and bulges in expiration, which is
converse of its normal movements. Litten's diaphragmatic sign
turally absent, and there is no inspiratory descent of the liver or
en. Blowing, coughing and pressing at stool are impossible. Costal
thing is exaggerated, the accessory muscles are strongly in play,
nœa and cyanosis are present especially on exertion, and coincident
chitis may cause most alarming symptoms, allowing stagnation
ucus and basal congestion. The nerve may be tender in the neck.

iagnosis and Treatment.—Costal hysterical breathing may at first
e confusion, as may inflammation and degeneration of the dia-
gm from diaphragmatic pleurisy. The *outlook* is always grave.
more favorable in syphilis, lead or diphtheritic neuritis than in
holic forms. *Treatment* is that of the cause, but is usually futile.
dic phrenic stimulation is sometimes beneficial, as in apparently
yxiated new-born infants.

hrenic Spasm.—1. *Clonic spasm,* singultus, or hiccup is a form
hich the diaphragm spasmodically contracts with a sound due to
len closure of the glottis, sometimes 50 to 100 times a minute. It
robable that the vagus and respiratory centres are involved, for
nœa, dysphagia or dysarthria may occur. Clonic spasm occurs
in inflammation, either thoracic (pleurisy, pericarditis) or abdom-
(peritonitis, appendicitis, obstruction); (*b*) in irritation; œsopha-
, mediastinal, hepatic or gastric, directly or reflexly; (*c*) in
ous affections, as centric disease or the neuroses; and (*d*) in
emia, fevers or uræmia. *Treatment* is that of the cause. Gastric
ge; apomorphine; circular constriction of the lower chest with
head bent forward; attempts to sneeze, cough, breathe deeply or
s down with the glottis closed; faradization, freezing, blistering
neck and epigastrium; antispasmodics and depressomotors, as
œtida, chloral, or bromides; and narcotics, and inhalations of
roform or amyl nitrite may be beneficial, although the affection is
n intractable.

Tonic phrenic spasm is rarer. The causes are tetany, epilepsy,
nus, rheumatism, and hysteria. The diagnosis is easy. The lower
t and upper abdomen are distended and often sensitive, the upper
t moves rapidly and forcibly, with the accessory muscles in full
, there is no respiratory excursion, the lungs are acutely distended,
abdominal viscera are luxated downward, there is dyspnœa, and
osis and death may result. *Treatment* consists of the application
eat, blisters and the faradic current to the lower chest, and the
of chloroform inhalations and morphia internally. Analogous

spasm may occur in the respiratory muscles, as yawning, sneezing, coughing, laughing and weeping spasms, possibly from vagus disease, or from the neuroses, psychoses, multiple sclerosis, bulbar paralysis or disease of the ear, nose or stomach.

The Posterior Thoracic Nerve.—This nerve is often affected in its long course, from trauma, muscular strains in overhead work (the nerve originates in the scalenus medius) and from carrying loads on the shoulder—whence its occurrence in 90 per cent. of cases in men; it is also involved in muscular atrophies with other muscles and in neuritis. The results are impaired fixation of the scapula, movement of the arm, and inspiration. The scapula wings out when the arm is moved forward, and stands less obliquely than normal, its outer part moving forward, while the lower angle is drawn in by the rhomboids and trapezius. The sole sensory change is pain in the neck and shoulder. The course is at best tedious, and rest, especially of the scaleni, is the prime indication.

Suprascapular Nerve.—It originates in the fourth, fifth and sixth cervical nerves, and is affected in disease of the plexus or alone in trauma, shoulder dislocations, or falls on the hands. The supraspinatus and infraspinatus are paralyzed, giving some weakness in fixation of the humerus, as uncertainty in writing, turning of the lower scapular angle up and in, some anæsthesia over the scapula, frequently shoulder girdle pain, and often involving the **Circumflex Nerve,** which arises from the fifth, sixth and seventh cervical nerves of the posterior cord of the brachial plexus. Deltoid paralysis (dislocation, crutch paralysis, plexus disease) produces inability to deviate laterally the arm, changes in the shape of the shoulder, which becomes angular, sometimes adhesions in the joint, which are probably trophic from its articular branch, and anæsthesia over the lower part of the deltoid. It may be confused with primary joint disease, in which, however, the scapula follows the arm movements. Paralysis of the teres minor is unimportant.

Musculospiral Nerve.—Arising from all the roots in the plexus except the first dorsal, and curving around the humerus, it is the most exposed and most frequently injured nerve in the arm or body; injuries result from fracture, callus, blows, muscular exertion, carrying heavy weights, tying the arms to the side or behind the back, sleeping with the arm over the back of a chair, especially in alcoholics, lifting the arms during surgical anæsthesia, and Esmarch's constrictor; less often injury results from injections of ether, from cold or from lead palsy. *Musculospiral (radial) paralysis* involves (*a*) the extensors of the upper extremity, *i. e.*, the *triceps*, so that the elbow cannot be extended, and the supinators; the triceps sometimes escapes affection; the supinator longus is not involved in lead palsy or traumatism below the lower third of the humerus and in centric disease it escapes because its centre is with the flexors. The *extensors of the wrist* are involved, producing marked atrophy of the back of the forearm and the characteristic wrist drop; the *extensors of the thumb and fingers,* are affected, and become semiflexed and can only be extended by the interossei (ulnar nerve) after the first phalanges are passively extended. Changes in the synovial sac or bones of the dorsal wrist produce the painless carpal tumor of Gubler, from involvement of branches to the wrist, or more probably from the mere mechanical action of flexion (see LEAD INTOXICATION). (*b*) *Sensory disturbance,* which is slight compared with the palsy, may rarely cause anæsthesia over the deltoid and over the back of the forearm or more often over the radial side of the dorsum of the hand, thumb, index and half of the middle finger. Most cases recover with rest and immobilization. Flexor contracture can be avoided by careful splinting.

Median Nerve.—The median nerve originates from the outer and inner cords of the plexus and contains fibers from all its cervical roots. Isolated paralysis is not common. It results from trauma, fractures, stabs, carrying weights in the bend of the elbow, and sleeping on the arm. (*a*) *Motor symptoms* are conspicuous. Both pronators are involved, besides all the flexors of the forearm, wrist and fingers except the flexor carpi ulnaris and the ulnar part of the deep flexor of the third and fourth fingers. In the hand, the palmaris longus, the abductor, opponens and short flexor of the thumb (resulting in flattening of the thenar prominence and the "ape hand" from the index finger and thumb lying parallel) and the two radial lumbricales are affected. The second phalanges cannot be flexed on the first (save in the two ulnar fingers); flexion of the first phalanges by the interossei is possible; metacarpophalangeal subluxation may result from the unopposed extensor action of the interossei on the second and third phalanges. The paralyzed muscles may waste and trophic manifestations are quite common. (*b*) *Sensory symptoms* are often slight. Anæsthesia, if present, is greatest in the palm, front of the thumb, the first two fingers and half of the third.

Ulnar Nerve.—Its origin is the last cervical and first dorsal nerves; its superficial course at the inner condyle, forearm and wrist, expose it to injury, as by dislocation of the elbow, cuts and pressure on the elbow during sleep and work. Neuritis occasionally causes paralysis. It must be distinguished from spinal disease, which ascends from the dorsal to the cervical region; this form is usually bilateral, and is preceded by ulnar anæsthesia (Biernacki) in tabes and paretic dementia. The *flexor carpi ulnaris* is paralyzed (the hand deviates to the radial side in attempts at flexion), and also the ulnar half of the *deep flexors* of the *fingers,* the muscles of the little finger, palmaris brevis, the interossei, the third and fourth lumbricals, the adductor and inner head of the short flexor of the thumb (the thumb being rotated toward the palm). The first phalanges cannot be flexed nor the others extended, although this is less in the first and second fingers, in which the lumbricals are supplied by the median nerve. The "*claw hand*" results, although it is less marked than in spinal muscular atrophy; the hypothenar eminence, palm and interosseous spaces become thin. *Sensory loss* is limited to the back and front of the ulnar side of the hand, the dorsum of two and a half fingers, and the front of one and a half fingers on the ulnar side.

Combined Paralysis of the Arm Nerves.—This affection, described by Erb (1884), is very common and is due to affections of the vertebræ, cord or the nerve roots, to tumors, trauma or cicatrices of the neck, and wrenches or forcible delivery in obstetrics. Several nerves are involved, as the musculospiral and ulnar in fracture of the humerus, or in subcoracoid luxation; in Erb's case, the circumflex, suprascapular and musculocutaneous nerves, fifth and sixth cervical roots, with some branches from the fourth were imbedded in a cicatrix and paralyses of the deltoid, teres minor, spinati, biceps, brachialis and supinators, with few sensory signs resulted. Excision of the scar, resection and suture of the nerve resulted in recovery. Erb found one spot between the scaleni where all these nerves could be stimulated and where, therefore, they could be simultaneously affected. Neuritis ascending from the median nerve may cause ulnar and musculocutaneous or other paralysis. Obstetrical paralysis usually results in recovery, while adult forms depending on their etiology are often protracted or permanent. Flaubert (1827) described a combined paralysis, which usually bears Klumpke's (1885) name, in which paralysis of the first dorsal and eighth cervical roots (ulnar and median paralysis) also causes sympathetic symptoms, as narrowing of the pupil and the interpalpebral fissure.

Brachial Neuritis.—This affection was first clearly described by Gowers and held to be a perineuritis of the plexus or nerve roots (radicular neuritis), occurring largely in gouty or rheumatic subjects over fifty years of age, but at times due to compression or trauma. The author saw one case which occurred in the right arm of a busy surgeon. A cervical rib may cause compression symptoms; Reisman (1904) collected 46 cases of which 24 were operated on with good results. It is marked by great pain, the first, most severe and most enduring symptom. It is constant, with paroxysmal exacerbations, is increased by the least movement, and is experienced over the plexus, nerves, or referred to the wrist or scapula. Associated with it are tenderness over the trunks or plexus, hyperæsthesia of the skin, some muscular wasting, mostly in radicular forms, and somewhat less often anæsthesia, anæsthesia dolorosa, œdema, glossy skin and trophic alterations in the joints. Neuralgia, angina pectoris and aneurysm may cause diagnostic difficulties. Remak holds that reported recoveries of acute poliomyelitis in children (Kennedy's temporary paralysis) are only plexus neuritis. The *outlook* is usually favorable, but a long course of from a month to more than a year, relapses and even inte recovery (some wasting, cramps, rheumatic pains and joint ons) must be considered in framing the prognosis, despite s early and careful treatment as in reduction of shoulder dis- ns. Treatment as in neuritis, consists of rest by immobilization, on of cocaine in severe cases, and treatment of the underlying , gout or rheumatism.

res of the Trunk.—These are rarely involved except in lesions spine or cord. The *neuritic pain in Pott's disease* or cord tumor, e *girdle pain of tabes* are of great localizing value. It has been that *herpes zoster* is due to inflammation in the spinal root (Campbell and Head).

res of the Lower Extremities.—These nerves, except in sciatica, s frequently affected than those of the arm.

Lumbar Plexus.—The lumbar plexus (the first three and a half r roots) may suffer in abdominal tumors, glandular enlargements, ition, psoas abscess, pelvic inflammations, spinal meningitis, ral disease or in neuritis ascendens. The symptoms are *paralysis* cremasters, *flexors and adductors* of the *hip* and *extensors of the* all the hip flexors are involved in plexus disease only; great neuritic forms is noted in the ilio-inguinal, iliohypogastric and crural areas. *Anæsthesia* exists over the lower abdomen, front les of thigh and inner side of leg and foot and adjacent sides of d second toes.

rior Crural Nerve.—Lesions in the abdomen cause paralysis of cus (not the psoas), the hip flexors and knee extensors, with g, loss of knee jerk and anæsthesia over the front and sides of gh and the inner side of the leg and foot. It has occurred in r cent. of labor cases and may result from stab wounds of the in which case the iliacus escapes involvement. Neuritis of its *l cutaneous branch* (multiple neuritis, alcoholism, nicotinism t) may cause isolated anæsthesia over the lower outer half of gh, paræsthesia or marked hyperæsthesia; the last condition, as *meralgia*, was described by Bernhardt and Roth and is neuritis but may be due to pressure from varicose veins. It d, chiefly, on standing or walking. In 1900 Musser and Sailer ed 99 cases (74 per cent. in men).

sure on the cord at the fourth lumbar roots produces the same as a partial lesion of the ant. crural nerve, as paralysis of knee rs, loss of knee-jerk and anæsthesia of anterior part of thigh, t clonus is present. Disease of the anterior horns on one side ceptionally occur in adults, but its wider prevalence and sensory tion are usually sufficiently distinctive. Caudal lesions are bilateral.

rator Nerve.—This nerve is rarely alone involved save during parturition. Its paralysis affects the thigh adductors, so that the knees cannot be crossed, and the pectineus and ext. obturator, so that impairment of outward rotation of the thigh results. Sensory symptoms concern the hip and knee joints, which sometimes show trophic disturbance. The pain of hip disease is referred to the knee by the obturator nerve.

Superior Gluteal Nerve.—This arises from the lumbosacral cord, being intermediate between the lumbar and sacral plexuses. Its rare isolated involvement results in loss of thigh abduction and circumduction (gluteus medius and minimus).

The sacral plexus (half of the fourth, the fifth lumbar and the first four sacral nerves) supplies motion to the extensors and rotators of the hip, flexors of the knee and all the foot muscles, and sensation to the gluteal region, the back of the thigh, outside and back of the leg and most of the foot by the gluteal nerves and the two sciatic nerves. The *lesser sciatic* is affected only in disease of the plexus; it is often involved in pelvic inflammation and tumors, parturition, traction in leg deliveries, plexus neuritis and diseases of the hip.

Great Sciatic Nerve.—This is the largest, the most exposed throughout its course and hence the most frequently diseased nerve of the lower extremity. Paralysis of the gluteus maximus interferes with rising more than with walking. A lesion near the sciatic notch paralyzes the extensors of the hip, the flexors of the leg and all muscles below the knee. If the lesion lies below the upper third of the thigh, the flexors of the leg may escape involvement, and walking is still possible, even though all muscles below the knee are paralyzed, if the ant. crural nerve for the hip flexors and knee extensors is intact. Sensation is lessened over the outer half of the leg, most of the dorsum of the foot and all of the sole. Wasting is marked over the back of the thigh and perforating ulcer of the foot may develop. It has two *branches:* (*a*) the *external popliteal* or *peroneal nerve,* involvement of which causes paralysis of the tibialis anticus, peronei, long toe extensors, extensor brev. digitorum; the ankle cannot be flexed dorsally, the first phalanges of the toes cannot be extended, adduction and lifting the inner edge of the foot is difficult and abduction and lifting its outer edge is impossible. Foot-drop results and contracture assumes the equinovarus type. This nerve is the analogue of the musculospiral in the arm. Wasting is often observed and the anæsthesia covers the anterior outer half of the leg and the dorsum of the foot. (*b*) *Internal popliteal nerve,* involvement of which affects the popliteus, tibialis posticus, long flexors of toes and all the foot muscles except the extensors; plantar flexion, abduction of the foot, inability to stand on tiptoe, talipes calcaneus and the claw foot from overextension of the proximal and overflexion of the second and third phalanges result. Sensory alterations are variable; anæsthesia may be observed on the outer lower posterior part of the leg and on the sole of the foot. Its plantar branches are rarely affected alone; there may be (i) paralysis of the internal plantar nerve, which is analogous to the median; anæsthesia on the inner part of the sole and the plantar surface of the three inner toes and one-half of the fourth; paralysis of the short toe flexors, plantar muscles of great toe (except the abductor) and the two inner umbricals; the big toe is overextended, "the hammer toe"; (ii) paralysis of the external plantar nerve, which is the analogue of the ulnar, produces anæsthesia of the outer half of the sole, little toe and one-half of the fourth, paralysis of the accessory flexor, small toe muscles, all he interossei, two outer lumbricals and great toe adductor; the results are serious, walking being impaired by flexion of the second and third and extension of the first phalanges.

Caudal Lesions.—(See SPINAL LOCALIZATION.)

Sciatic Neuritis.—Sciatica, Ischialgia, Malum Cotunnii (Cotugno, 1764).—Sciatica is, after facial neuritis, the most frequent isolated form and is rarely simply neuralgic.

ETIOLOGY.—*General Causes.*—(*a*) Sex and age; 88 per cent. of cases occur in men; 16 per cent. in persons between twenty and thirty, 31 per cent. between thirty and forty, 25 per cent. between forty and fifty, and 18 per cent. between fifty and sixty years of age. It is very rare in children. (*b*) Acute infections, rheumatism, and grippe, are causative. (*c*) Intoxications; gout, fascial rheumatism (sometimes by extension from lumbago), and diabetes, are factors. (*d*) Anæmias and neuroses are much less significant than in true neuralgia.

Local Causes.—These include (*a*) spinal affections, tumor, and neuritis of the cauda; (*b*) abdominal and pelvic disease, as aneurysm, abdominal and pelvic tumors or inflammation; (*c*) cold; exposure and dampness, favored by certain occupations; (*d*) trauma to the spine, pelvis, hip or peripheral course of the sciatic trunk or branches, as from the use of forceps or pressure of the head in labor, fractures or dislocations, falls, protracted sitting or riding, use of the sewing machine, marching, vigorous contractions of the leg flexors, peripheral pressure by popliteal aneurysm, tumors, callus or varicosities.

PATHOLOGY.—In the infrequent autopsies or in surgical stretching of the nerve, its sheath shows perineuritis with œdema and in some cases increase of connective tissue or varicosities of the sheath vessels, especially in the middle thigh. Sometimes interstitial neuritis is found.

SYMPTOMS.—The symptoms are unilateral and chiefly sensory, as *pain and tenderness* over the sciatic roots, trunk and branches. (*a*) The onset of *pain* is usually gradual but is sudden and febrile in some rheumatic cases. It is usually constant, with exacerbations which in some cases are agonizing. The pain descends more often than it ascends; it sometimes radiates to the buttocks and crural region, even into the other leg. Gowers attributes the local pain to perineuritis, and the distant pain to interstitial neuritis. Any movement which increases sciatic tension increases or renews the pain (Lasègue's phenomenon); pain is increased or initiated by flexing the hip and extending the knee, walking, stooping, or by defecation. The patient seeks positions which spare stretching of the nerve and its compression by the muscles; he may lie on the opposite side with the thigh extended, knee flexed and foot extended, or on his back with hip and knee flexed and foot extended; in standing, he rests on the sound leg, and in protracted cases scoliosis develops (ischias scoliotica, Albert, 1886), in which the lumbar convexity is toward the diseased side, heterologous scoliosis, which usually disappears with the disease; a homologous scoliosis (with the lumbar concavity toward the affected side) may result in case the muscles of that side become spasmodic. (*b*) *Tenderness* may be detected over the entire course of the nerve or in certain locations only; (i) over the lumbar region; (ii) near the sacrum and post. sup. iliac spine; (iii) at the lower gluteal fold where it leaves the sciatic notch; (iv) between the tuber ischii and trochanter; (v) especially over the middle of the *posterior surface of the thigh*; and (vi) in other lower areas of distribution over the popliteal space, head of fibula, calf, internal and external malleoli and sole. The enlarged nerve may occasionally be felt. Pain is often experienced in the hip and sciatic notch when pressure is made in the popliteal space. (*c*) Other symptoms are less frequent, as vasomotor changes, herpes, muscular cramps, twitchings, fibrillation or wavy movements (myokymia); there is usually some diminution in the electrical excitability or perhaps an increase, but there are rarely marked evidences of the degenerative reaction; muscular atrophy may occur from disuse or neuritic atrophy; sciatic paralysis may occur; hyperæsthesia, paræsthesia, or in severe cases anæsthesia, may develop in the posterior part of the thigh, leg or foot; very rarely there is polyuria or glycosuria. The patellar reflex is usually normal, the Achilles reflex is often decreased or absent and Gibson found the cremasteric usually increased.

Diagnosis.—(*a*) The etiological factor should be sought. (*b*) The pelvis should always be examined in women and the rectum in both sexes, because compression produces more distant than local pain; the urine should always be examined for sugar and albumin; and free catharsis and bowel lavage should always be given. (*c*) Bilateral sciatic pain suggests disease of the cord, especially tabes, a lesion of the cauda, in both of which there is no sciatic tenderness, or it prompts examination for sugar. (*d*) Coxitis, sacro-iliac or psoas disease present appropriate local signs, without however Lasègue's sign or sciatic tenderness, both of which are also absent in (*e*) hysteria. (*f*) Muscular rheumatism is irregular and is chiefly confused with pain over the sciatic branches.

Prognosis.—The prognosis is generally good, in regard to the ultimate outcome. It depends on (*a*) whether the affection is primary, as from cold, or secondary, as from disease of the cord, or pelvic neoplasm; (*b*) the amount of rest which the patient accepts; (*c*) the intensity of the neuritis, which may extend to the lumbosacral plexus;

and (*d*) the chance (in 33 per cent.) of relapse. The average course is from two to eight weeks, but cases may last over a year, indeed one to three or exceptionally six to thirty years.

TREATMENT.—(*a*) Treatment is that of the *cause*; the salicylates, in some, but unfortunately few, cases afford relief; gouty or syphilitic taints may be benefited by colchicum or antisyphilitic remedies, which are often given as a last resort in other cases. (*b*) *Rest in bed* is the most important indication. In this, the use of Wier Mitchell's long splint is often beneficial. (*c*) *Counterirritation* by blisters over the nerve's course or by touching the tender or painful points with the hot iron, may give temporary though rarely lasting relief. (*d*) *Analgesics and narcotics* may be used. Acetanilide has little effect. Gelsemium (fl. extr. ♏ ii) at intervals of two to four hours, not infrequently gives relief, though it is necessary to watch for pulse weakness and cutaneous tingling. Codeine and atropine should be given before resort is had to morphine, though morphine is often inevitable, but always dangerous. Chronic morphinism may be promoted by severe pain, the long course, and the tendency to relapse. Hypodermics of cocaine are very beneficial, though this method is attended by the same danger. Simple hypodermics of water are sometimes valuable, and the author has seen good results from deep hypodermatic use of Schleich's solution given in quite large amounts over sensitive points. Lange recently advises the use of 160 c. c. of 0.1 per cent. eucain into the muscle and nerve at the sacrosciatic foramen. Injections of chloroform and ether are recommended, but should be avoided because of the possibility not only of sloughing but also of accentuating the neuritis. (*e*) *Acupuncture*, the old Chinese treatment, is recommended by Gibson; it resulted in complete relief in 56 per cent. of his cases, much improvement in 32 per cent., slight benefit in 10 per cent., and failure in but 2 per cent. (*f*) *Massage* may be given. Negro has obtained results from frequent and severe local rubbing and rolling, over the nerve trunk. (*g*) *Electrotherapy* may aggravate the pain and is at least of uncertain value. Galvanism is reported to have helped some cases. (*h*) *Nerve stretching* may be effected by extension by weights, although it is rarely tolerated by the patient in personal experience. The direct exposure of the nerve has justly fallen into disuse, for the same results attend simple stretching, which should be performed under anæsthesia. The hip should be strongly flexed, the knee extended, the foot dorsally flexed, and the pelvis firmly immobilized. Ordinary care prevents luxation of the hip. The results were excellent in half of twenty personal cases, and were seemingly curative in seven.

V. NEUROSES.

HYSTERIA.

Definition.—The neuroses are nervous affections, provisionally isolated and often designated "functional," since no anatomical basis has yet been discovered. Hysteria is difficult to define. Möbius considers it "a state in which ideas control the body and produce morbid changes in its functions." The school of Charcot has established the psychogenic nature of hysteria. It is a cerebral, cortical condition, in which the sensory and motor centres are affected, the perception, association and recollection of ideas are disturbed and trophic and vasomotor disorders occur. As its name implies, hysteria was long thought to be due to the uterus; Sydenham first called it a nervous affection.

Etiology.—(*a*) *Age:* It is most frequent at puberty and adolescence (fifteen to twenty-five years). It is occasionally seen in children (six to ten years or even earlier), and rarely in middle or advanced life. (*b*) *Sex:* Women are six to ten times as frequently affected as men, although in the lower French classes, it is more frequent in males. (*c*) *Race:* The Jewish, Latin and Slavic races are most frequently affected. (*d*) *Heredity:* Charcot, and most of the modern French school, hold that this is the sole cause, other factors being only *agents provocateurs*. Parental intoxications, toxæmias, constitutional diseases, as gout, diabetes, arthritis deformans and a neurotic taint are important in the transmission of a congenitally weak nervous system. (*e*) *Acquired hysteria* may be due (i) to various intoxications, notably lead, alcohol and drugs; (ii) to infections, as typhoid, malaria, diphtheria, and syphilis; (iii) to constitutional maladies, as gout, diabetes, chlorosis, secondary anæmias or cancer; (iv) to exhaustion from mental or physical overwork or excesses; (v) to emotional disturbance of any kind; (vi) to trauma, especially factors involving psychical shock. (vii) It may occur as an associate with chronic nervous disease, epilepsy, syringomyelia, tabes, brain tumor, and multiple sclerosis. (viii) Imitation is an important factor in schools, hospitals, and at religious revivals; endemics occasionally occur now as they did in the middle ages. (Refer to Zola's *Lourdes*.) Reference to the sexual organs as causes and to reflex hysteria is purposely omitted, because they have no etiological bearing. The factors enumerated under acquired hysteria are, according to the French School, merely incidental causes, making manifest the latent hereditary disposition.

Symptoms.—There is no typical clinical picture, and the disease is much more commonly polysymptomatic than monosymptomatic. French writers distinguish two main groups of symptoms, the *stigmata* and *accidents*.

The **stigmata** are usually but not necessarily persistent symptoms.

1. SENSORY STIGMATA.—Sensory symptoms are present in 85 per cent., are more frequent than the motor and are often combined with them. (*a*) Hysterical *anæsthesia* in some form is very seldom absent. It must often be looked for, but the fact should not be too obviously suggested to the patient. It varies in *degree and form*. It may be absolute, affecting appreciation of touch, pain, heat and cold. *Analgesia* is its most common form and, in decreasing order of frequency, there may be complete anæsthesia, hypæsthesia, loss of tactile sense, loss of all sensation save to the faradic current, or loss of faradic response alone. In the mucosæ the same deficit frequently occurs, and sometimes the bones, ligaments, joints and muscles are likewise anæsthetic or analgesic. The *distribution* varies. In rare cases there is universal cutaneous and muscular anæsthesia; the following are more frequent: Hysterical *hemianæsthesia*, which involves precisely one-half of the skin, often the accessible mucosæ and the special senses; *segmentary* or geometrical anæsthesia, which affects one limb or part of it, is variously described as "mitten, or stocking" anæsthesia, and is frequently associated with motor or special sense disturbance in the part affected, as paralysis, aphonia, amblyopia, and deafness; and *insular* anæsthesia, which affects small areas, and is therefore frequently overlooked.

Characteristics.—The anæsthesia is psychical, is associated with the functions of the part affected and does not follow the area of distribution of the peripheral nerves or the spinal segments; it is not noted by the patient, at least until suggested; fine movements may be conducted with the aid of the eyes, as the using of a pen; it may be localized because of injury or disease in the part. Most of the reflexes which are usually altered by organic disease, remain normal, as the pupillary, lachrymal, epigastric, cremasteric, patellar and organic reflexes, while the pharyngeal in 90 per cent. of cases is lost as well as the plantar (though the knee reflex remains); the anæsthetic areas may last during life, but they vary, move even under examination (Patrick), or disappear from emotion, strongly aroused attention, drugs or on application of various indifferent substances, as metal or wood (*transferring* it to the opposite side).

Hysterical hyperæsthesia and *hyperalgesia* are very common in localized areas, but never in universal or hemiplegic distribution. They occur spontaneously, or from attention directed to a part or organ, as by traumatism. A joint may be affected with or, without contracture (Brodie's joints), and this often leads to great and unfortunate errors. There is sometimes hypæsthesia in the sphere of any of the special senses. The feeling of a nail in the top of the head (clavus hystericus), sensitive spots in the mammæ, epigastrium, groins or spine are the most common forms and may occur in the midst of anæsthetic areas. The so-called ovaralgia is found more often in the left side and is cutaneous; it has no relation to the location or to disease of the ovary; it occurs also in men and in women normal as to the pelvis. Because hysterical attacks have been initiated and sometimes stopped by pressure over this and other sensitive areas, they have been called *hysterogenic* points or *zones*, but attacks are only so produced in patients already hysterical and the hysterical seizure on pressure results only from some associated memory on the part of the patient; they are often the result of suggestion. The *special senses*. *Taste* and *smell* may be dulled or hypersensitive, and *hearing* may be blunted, or more rarely complete central deafness results, which is usually associated with anæsthesia in and about the auditory meatus. Complete *loss of vision* is both rare and transitory. Hemiopia and scotomata are almost always organic and not hysterical. Partial stigmata are more common. In most cases there is *concentric contraction of the visual field*, which is usually bilateral, but unequal on the two sides, and generally greater on the side of the cutaneous defects. Concentric *contraction of the field for colors* (dyschromatopsia) is more characteristic, even pathognomonic. In the normal eye, blue has the largest field, then yellow, orange, red, green and violet; in hysteria the red field is often wider than the blue and is the last color lost. Achromatopsia designates the complete loss of color perception. Errors in accommodation (always excluding abnormal corneal curvatures) may cause double images in one eye—the other being closed—(monocular diplopia,) triple images (polyopia), or large or small images (macropsia, micropsia). In hysterical *amblyopia*, impairment of vision in the affected eye is decreased when the other eye is opened.

2. Motor Stigmata.—These are much less conspicuous but foreshadow the motor accidents described later. Voluntary movements are weaker, more clumsy, slower, less coördinate than normal, and there is a slight tendency toward contractures.

3. Mental Stigmata.—These are in certain cases so pronounced as to justify the term psychosis. Hysteria is always a psychical affection. The most conspicuous alienation is impairment of memory (*amnesia*), which concerns memory in the ordinarily accepted sense, as memory for persons, things, words, hysterical paroxysms experienced, etc., or concerns memory and associations for speech, writing, walking or standing. The amnesia, like the paralysis or anæsthesia, is functional, and like them, returns, save in rare cases. *Mental power* is distinctly decreased, *will power is lost* (aboulia) or lessened, and the patient becomes the ready victim of impulses, impressions, suggestions, fixed ideas and misconceptions both on his own part and on that of others who incorrectly consider him deceitful, vain or erotic.

Accidents.—The accidents of hysteria are the incidental attacks, also called *seizures or paroxysms*. They are usually transient or intermittent, but may become fixed and are then ranked as stigmata.

1. Motor Accidents.—(*a*) *Convulsions* occur in 54 per cent. of all and 84 per cent. of female hysteria. They are much less frequently the hysteria major (*grande attaque* of Charcot) than the partial, modified type (hysteria minor). The "*grand attack*" (Charcot, Richer) comprises (i) a prodromal stage, with mental depression or exaltation, palpitation, vasomotor symptoms, nausea, polyuria, an

ra beginning in the epigastrium and culminating in feelings of oking (globus hystericus), vertigo, and unconsciousness; (ii) a riod of epileptiform convulsions (so-called hystero-epilepsy) in which, exceptional cases, there may be biting of the tongue or even the cry epilepsy; the face is at first pale, then red; the teeth are ground; convulsion is most pronounced on the anæsthetic side, toward ich the face turns; the tonic spasm lasts less than two minutes, and often characterized by stupor, stertor and slow wide movements of ination, circumduction, and overextension, for the trunk and body tetanically rigid; then the clonic spasm develops, with sobbing piration, audible swallowing, abdominal rumbling and small nic movements. Resolution follows slowly. (iii) There is a period "clownism," characterized by contortions and grand movements, opisthotonos (*arc de cercle*), bowing movements, violent struggling, eams, strikes and bites. (iv) There is next a period of "passional itudes," in which the subject mimics terror, rage, joy, and love; is is followed by (v) a stage of delirium with hallucinations, perhaps animals (zoöpsia). The entire attack covers fifteen to thirty minutes d frequent repetitions constitute the "status hystericus."

The *partial attack* contains some of the elements of the major type, d infinite variations occur; in the vertiginous type, there is vertigo d sometimes the other features of Ménière's disease; the globus e is extremely common; the epileptiform variety closely resem-s epilepsy; there are also tetanic, emotional and syncopal types; nce-like sleep, with catalepsy in which the limbs retain any atti-le given them, with sleep, which is apparent only, for the subject er recalls everything; there may be attacks of somnambulism, in ich patients run miles or even undertake distant travel without collection of the transpired interval. (*b*) *Paralysis* is an accentua-on of the stigmatic muscular weakness. In over 50 per cent. it velops after convulsive paroxysms; again it follows emotional uses, traumatism or organic brain disease. It develops suddenly gradually; it assumes the paraplegic five times as often as the miplegic form; the monoplegic variety is less frequent; the tri-egic, diplegic (quadriplegic) or crossed forms are most common. hey are rarely complete; often automatic (gesticulatory) movements ay be seen; the antagonistic muscles are also involved, thus, in tempted flexion of the knee the knee extensors contract; remissions d relapses may occur; the paralyzed part may be limp or contrac-red; the paralyses may be ephemeral or life-long in duration; ralysis may be "ideal," *i. e.*, only for standing or walking. Vaso-tor changes and œdema are rare; Wier Mitchell described a hemi-lema; the tendon, skin and electrical reactions are usually normal, hough the tendon reflexes may be increased. *Hysterical hemiplegia* curs three times as frequently on the left as on the right side. Its fferentiation from organic hysteria is based on Babinski's table the following page.

ORGANIC HEMIPLEGIA.——*vs.*	——HYSTERICAL HEMIPLEGIA.
Coma: often complete.	Rarely complete (automatic movements often persist).
Reflexes: tendon and bone reflexes abolished at first; later increased. Ankle clonus.	Never abolished; no clonus (or only a spurious ankle clonus due to contraction of the calf muscles, Gowers).
Cutaneous reflexes (abdominal and cremasteric) early lessened or abolished; Babinski toe sign; lachrymal reflex abolished.	Skin reflexes normal; no toe sign. Never abolished (Spiller).
Paralysis: (1) Unilateral; (2) not systematized; face, arm and leg are weak during bilateral synergetic movements. (3) Involves subconscious as well as conscious voluntary movements, producing the platysma sign (lessening of its contraction on the hemiplegic side, *e. g.* on opening the mouth forcibly), and the combined flexion of the thigh and trunk (when the patient attempts to sit up). (4) Paralysis slowly improves some, but is not variable.	(1) Not always unilateral; if face involved, usually on both sides. (2) Paralysis sometimes systematised; and hemiplegic side functionates perfectly in bilateral synergetic movements. (3) Subconscious voluntary movements are not involved; absence of the platysma sign and that of combined flexion of the thigh and trunk. (4) Variable paralysis, improvement alternating with relapses. Absolute aphasia with ability to write (Charcot).
The *tongue* deviates slightly toward the paralyzed side.	Very slightly, very greatly or even toward the sound side.
Lower muscular tone, as lowering of the eyebrow or exaggerated flexion of the forearm, especially in early cases.	None. Asymmetry of the face is due to spasm, and exaggerated flexion of the forearm is absent.
Contracture cannot be voluntarily reproduced; at first flaccid paralysis, then contracture.	May be reproduced: variable paralysis, now persistently flaccid, now spastic from onset; flaccidity with increased reflexes highly characteristic.
Hemianæsthesia: rarely total; mostly of stereognostic and muscle sense; in general rare; coarser; more lasting when present; often with bilateral hemianopsia.	Often total, involving special sense; mostly involving pain and pressure sense; frequent; variable in duration and location; usually with concentric contraction of visual field, and sometimes with crossed amblyopia.

In hysterical *paraplegia* there are usually no trophic changes, no bedsores, no electrical alteration, no incontinence of urine or fæces, and rarely retention of urine; concomitant sensory changes are significant, but many errors in diagnosis are made. Hysterical *monoplegia* usually occurs with anæsthesia of even greater extent and segmental distribution; it is lawless and illogical, viewed from the standpoint of organic disease. In *astasia-abasia* (Charcot and Blocq), the patient is unable to stand or walk, although the muscles concerned may be efficient in other movements. The condition is essentially hysteria, although it was thought to result from neurasthenia and hypochondriasis. Lesser degrees are called *dystasia* or *dysbasia.* The term stasobasophobia is self-descriptive. Möbius' akinesia algera is similar. (*c*) *Hysterical contractures* precede, succeed or alternate with paralysis, or occur alone, but, as Church well says, they are "rigid palsies"; they usually persist during sleep and yield to ether anæsthesia, save in severe protracted cases with great fibrous transformation and irremediable deformity; they develop and terminate suddenly or gradually; their causes of onset and distribution are as in paralysis. In the arms, contracture of the flexors predominates; in the legs, that of the extensors (club foot). Various spinal deformities may develop, most frequently from pseudosciatica. Wier Mitchell has seen the muscles so contracted as to resemble actual muscle tumors. It is still stated that the *face* always escapes paralysis, but its lower portion may be weak; more often, however, it is contracted early, even when paralyzed; hemispasmus glossolabialis and trismus have been observed. Spastic or pseudo-ptosis may resemble the paralytic form, but spasticity, the lowered or more level eyebrow, and the concentric or parallel folds in the lid, are distinctive of contracture. The ocular muscles are seldom involved; voluntary movement may be impossible, but associated and reflex movements are normal; convergent squint may occur, but never the divergent type nor contracture of a single rectus or oblique muscle; pain suggests hysteria. The pupils are usually said to be normal but Karplus maintains that the light reflex is lost during convulsive seizures. Organic disease of the *hip* may be simulated; local hysterical œdema may occur. Brodie (1827) first drew attention to this group of cases, and later Duchenne. Many avoidable diagnostic errors are constantly made. (*d*) There may be ***rhythmical spasms***, tremor, or tic. Rhythmical spasms, chorea major, the saltatoric chorea (*v. i.*) and local manifestations, as barking cough or the various tics, may occur. Hysterical tremor is wholly a "rest tremor"; that is, it occurs without voluntary movement. It may resemble the senile, Basedow, or lead tremors, or that of paralysis agitans or may be combined with them. It rarely simulates the intention tremor of multiple sclerosis.

2. SENSORY ACCIDENTS.—These are frequent, and are often severe and painful. Headache may occur; it is often dull or may be severe; it may be superficial or deep; it may resemble trigeminal neuralgia, migraine or the headache of organic disease as tumor. Meningitis (hysterical pseudomeningitis) may be closely counterfeited. Spinal or vertebral pain is frequent and sometimes suggests tumor or caries. One and three-tenths percent.of cases of sciatica are said to be hysterical. Joint disease and various visceral lesions (*v. i.*) may be closely mimicked. Dieulafoy (1905) collated 59 cases of hysterical blindness, in 5 of which this accident was the only symptom.

3. VISCERAL ACCIDENTS.—(*a*) *Cardiac* accidents include tachycardia, palpitation and pseudo-angina pectoris. (*b*) *Respiratory* accidents: ***Aphonia***, usually attended by normal capacity to cough and often also to sing, has been described under paralysis of the vocal cords. It is usually due to an "idea," as in aphasia of the motor type, which allows the patient to write. Stammering, cough, and cries, may be provoked by local nasopharyngeal disease. Dyspnœa, resulting from paralysis of the diaphragm, and laryngeal spasm have already been considered.

Rapid breathing (tachypnœa) also occurs. The combination of dyspnœa, aphonia and paralysis of the diaphragm constitutes Briquet's syndrome. Hæmoptysis may occur, usually in the course of phthisis, but sometimes without it, possibly as a vasomotor derangement, since the blood is thin; one fatal case is on record. (*c*) *Digestive* accidents: Hysterical anorexia results from cortical conception, stomach pain, dysphagia from spasm, or simple sensory perversion; it may endure for weeks and cause extreme inanition or even death. Hysterical (pseudo-) hydrophobia is an analogous aversion to water. Vomiting is sometimes simulated by protracted globus or œsophageal spasm, in which the food is simply regurgitated. Vomiting itself may be occasional, or attacks may last weeks or months, often with remarkably little malnutrition; it is rarely fæcal but substances injected by rectum may be vomited in a few hours, or again may be blood-tinged, possibly from vasomotor disturbance or more often from actual disease, as round ulcer. Ulcer may also be simulated by pain and tenderness, which are surprisingly well confined not to one spot, but to the exact outlines of the stomach. The vomitus contains vicariously large amounts of urea when the urine is suppressed. Eructations are frequent and so much gas escapes that one must conclude that air has been swallowed; this also probably causes tympanites, in which swallowed air reaches the gut through the incompetent pylorus (Ebstein). Phantom tumors or spurious pregnancy (pseudocyesis) are explained by Gowers as protrusion of the distended bowel by tonic contracture of the diaphragm. Peristaltic unrest, diarrhœa on eating, enteritis membranacea, spastic constipation, simulation of peritonitis, rectal stricture or intestinal obstruction also occur. (*d*) *Genito-urinary* accidents: Anuria in some cases has existed for ten days without uræmia. Renal pain is not frequent, but hyperæsthesia of the bladder is common. The urine is often greatly increased, with low specific gravity and solids. During attacks of the major type, the solids are reduced, the urea one-third and phosphates one-half; the phosphates, which normally have a proportion of 3 of the alkaline to 1 of the earthy phosphates, show rearly an equal proportion of each. Depression of the genital functions is more common than exaltation; anæsthesia is frequent; vaginismus may be observed. Lower has found that the hysterical ovarian and other genital pains have no relation to local pelvic disease, a rather gratifying gynæcological observation.

Hysterical fever is a disputed topic. It is an established fact that severe forms may suffer a rise to 105 or 110° or higher. With visceral manifestations, this may lead to a diagnosis of meningitis or peritonitis; but on the other hand the possibility of simulation by the patient rubbing the thermometer, or more important still, the possibility of confusion with tuberculosis or typhoid, must always be kept in mind.

4. VASOMOTOR AND TROPHIC ACCIDENTS.—These are rare. Erythema is the most common. Gangrene, falling out of the hair or nails, Raynaud's local asphyxia, cutaneous hemorrhages (crucifixion stigmata, stigmata diaboli), bloody sweats, lachrymation, hæmoptysis,

hæmatemesis, sudden tender swellings in the mammæ, and angioneurotic œdema, which is sometimes blue in color, have been recorded in exceptional cases.

Course and Prognosis.—The course is always chronic. If we look on the disease as congenital, the prognosis is poor as to total recovery, especially with fixed psychical alteration and major manifestations. Jolly remarks that complete recovery is as rare as death, which rather overstates the facts. In mild forms practical recovery may follow, but relapses may occur on disproportionately slight provocation. The outlook is better in infantile and adolescent forms than in those of middle or advanced life. The stigmata may be ephemeral or life-long. Marriage may prove beneficial in light cases, but, with maternity, it is injurious in the severe types; marriage must never be regarded as a therapeutic agent.

Diagnosis.—The stigmata, mental, motor and sensory, are highly characteristic. The accidents are either unequivocally hysterical, or prove hysterical after consideration of their associations or history. The real danger lies in overlooking concomitant disease (*a*) nervous, as multiple sclerosis, brain tumor, paretic dementia; (*b*) postinfectious hysteria as hysteria plus neuritis; (*c*) lung, heart and other visceral diseases, all of which, as Sydenham remarked, may resemble hysteria. Simulation does not include the common borrowing or mimicry of symptoms by hysterics; simulation of hysterical stigmata and accidents is practically impossible.

Treatment.—1. PROPHYLAXIS.—This is possible when the earliest manifestations occur in childhood. Home treatment is frequently impracticable because of the family tendency. The etiological factors must be studied, especially the hygiene and the hours of sleep. Useful occupations, as out-of-door gardening, are preferable to less wholesome and more artificial plans of distraction. Cultivation of the cardinal virtues, courage and self-control, is sought. Servants who teach fear of the dark and tell fairy stories should be watched. Self-control should be insisted upon in adult cases.

2. GENERAL TREATMENT OF THE CAUSE.—As the disease depends on disturbed cortical relations between the psychical and material processes, the principal treatment must be psychical. The "fixed idea" must be grasped and eradicated, which is best accomplished in ordinary circumstances by isolation of the patient from home or from other psychical or emotional circumstances surrounding the inception of the disease. Then the undue susceptibility incident to the disease may be turned to therapeutic advantage by the self-confidence of the physician and by his covert suggestions. The Wier Mitchell rest-cure may result in great benefit, and later "vague hints regarding the curative power of nature," and fresh air and sunlight may captivate the patient. Symptoms must not be suggested to the subject, as anæsthesia or pelvic disease, and examinations, especially of the pelvis, should be omitted. Hysterics should not be allowed to associate with each other for obvious reasons. This general plan is psychotherapy,

which of course might include hypnotherapy, as advocated by the Nancy school, Bernheim and others. Hypnotism is generally dangerous in its ultimate and often in its immediate effects; though the definition of hypnotism may be a matter of difference or of dispute, its danger is shown by the fact that Strümpell considers it an "artificial hysteria," and Féré defines it as a "transformation of hysteria." "It is easier to make a sound person hysterical by hypnosis than to cure a hysterical one by it"(Liebermeister). Treatment is sometimes frustrated by the patient's vanity. Successful treatment requires unusual tact, profound knowledge of human nature, strong sympathies (but dominance of the patient) and enormous optimism. Of course certain single qualities, in an ignorant quack, may effect cures.

3. Special or Symptomatic Treatment.—(*a*) In relieving *pain*, the physician should first acknowledge its existence and intensity in order to soothe the patient, and should then apply blisters, liniments, or the galvanic current, with the anode on the painful area. Ovaralgia in most cases should be ignored; in exceptional cases pelvic surgery has given relief; Lower's position has already been mentioned (*v. s.*). (*b*) *Convulsions* are treated by dashing cold water into the face, by suggestive measures, or, if severe, by etherization. Bromides are of little value, valerian is sometimes beneficial and turpentine is recommended by Gowers as most efficient, 10 drops of the oil being given until slight vesical symptoms appear. Pouring water into the mouth or nose, which causes coughing, or Hare's method of holding a towel over the nose and mouth for 15 to 20 seconds, may also serve. (*c*) *Paralysis* and *contracture* should receive early treatment, for late neglected cases may necessitate surgical intervention, as tenotomy. Massage, faradism, blisters, and fixation in new postures are indicated, and especially should their significance be minimized and the patient be assured that they are not organic. In two cases the author has seen the paralysis disappear at once on administration of asafœtida in an effervescing mixture. (*d*) *Anæsthesia* should be ignored. (*e*) *Aphonia* is treated by intralaryngeal faradization. Laryngeal and other respiratory spasms usually answer to energetic traction on the tongue, and anæsthesia or tracheotomy is rarely necessary. (*f*) For *insomnia*, the patient should retire at an absolutely regular hour; nervines should be given rather than bromides, and hot milk should be very slowly sipped. (*g*) *Dysphagia*, vomiting and anorexia may be treated by rectal feeding, or by forced feeding through the stomach tube, which is the better method. Constipation is often obstinate, and its mixed spastic and paretic character is shown by the fæces which are like those of sheep. Free administration of olive oil by mouth and rectum, cold abdominal compresses and endorectal galvanization are usually effective.

NEURASTHENIA.

Definition.—Neurasthenia is a diffuse neurosis, which affects the entire nervous system, but chiefly the brain; it is characterized (*a*) by morbid irritability of the nervous system with a tendency to its rapid exhaustion ("irritable weakness"), and (*b*) particularly by psychical, motor, sensory, vasomotor, secretory and vegetative disturbances in function. The nervous tissue responds unduly to stimuli, its reaction time is short, and ready exhaustion results. It seems probable that the slight cellular and protoplasmic changes in nerve cells, noted by Hodge to follow fatigue, may have some bearing on its pathology. The affection was described and named by Beard in 1869, whose description was first received with some skepticism and ridicule abroad, but later gained universal recognition. It is possible that the disease is at present more frequent than formerly because of the more strenuous life and methods of education. The difference between "nervousness" and neurasthenia is only quantitative, and while nervousness is no disease in itself, it is also characterized by a short reaction time (irritability) and precipitate fatigue (weakness).

Etiology.—Neurasthenia is the most common neurosis, constituting 25 to 45 per cent. of all nervous diseases and fully 50 per cent. of all neuroses. (*a*) A *neuropathic tendency* is observed in 40 to 60 per cent. of cases, especially among Hebrews whose proneness to nervous diseases is referred by Erb to inbreeding and to desire of gain. Other neuroses, as hysteria, epilepsy or migraine, the psychoses, and parental infirmities or intoxications may be obtained in the family history. (*b*) *Age and sex:* It is a disease of adults (75 per cent. of cases occur in persons between the twentieth and fiftieth year). It is a question whether men are more often neurasthenic than women; we may say women are more disposed, men more exposed, to the disease, by their excesses in work, sexual activity and alcoholism. (*c*) *Psychical* causes include sorrow and care; hard mental or physical work rarely produces neurasthenia. Worry is a prolific factor; it is the irritable weakness of work. Hard work is injurious when combined with excesses in tobacco, alcohol, coffee, drugs or venery. Certain occupations entailing responsibility or emotional factors predispose to nervous weakness, as in railway or telegraph employes, board-of-trade men, teachers, actors, artists, and musicians, but even then it is questionable whether the work itself is most injurious. Trauma operates chiefly through psychical causes, as in the traumatic neuroses (*q. v.*). (*d*) *Intoxications*, as from the stimulants above mentioned, from drug habits, lead or arsenic poisoning, acute infections, especially typhoid or grippe, from chronic affections, notably malaria and syphilis, and altered condition of the blood, as gout, anæmia or diabetes, may be indirect factors. (*e*) *Sexual life:* Masturbation, excessive coitus, and coitus interruptus, sometimes promote neurasthenia, but their importance is enormously overestimated. Masturbation probably produces less direct physical than psychical injury, for it leads to brooding over its possible results. Continence is never injurious, as sexual indulgence is rather a habit than a necessity. In neurasthenic women gynecological complaints are more often subjective, secondary, and neurasthenic, than objective, primary and organic. The same statement usually holds true in male neurasthenics with chronic urethritis, or prostatitis. Pregnancy and lactation are sometimes apparent causes. (*f*) *Organic diseases* may possibly be factors, but it is questionable to what extent enteroptosis, chronic gastric or other somatic lesions promote neurasthenic symptoms. (*g*) *Climate:* Neurasthenia is more common in northern climates with relatively high altitude than in southern zones. It is possible that alcoholism with nervous tension is more potent than either climate or altitude.

Symptoms.—A clinical description holding for all cases is impossible. A patient presents himself for the treatment of some single visceral complaint, or describes symptoms in his head, heart, lung, stomach, sexual or other organs. The various types, which are often separately described, will be considered in the general picture, for pure forms are rare; they are the cerebral (*cerebrasthenia*), spinal (*myelasthenia*), cerebrospinal (*the general type*), the *sympathetic* or vasomotor and the *visceral* (cardiac, sexual, gastric) types. Some varieties concern the course, as (*a*) the *acute form* from simple nervous exhaustion in normal individuals, or in those with neurotic stigmata; (*b*) the *subacute* or chronic acquired form, and (*c*) the chronic *constitutional* neurasthenia, which is usually hereditary.

1. Cerebral Symptoms.—These are most important, because the disease is really a psychoneurosis. Headache is almost invariable, is more often dull than acute, and produces a sense of pressure ("lead cap" headache) or intracranial paræsthesia, and in localization is most often occipital. Vertigo is frequent. *Insomnia* is extremely common, but rarely absolute which always suggests organic brain disease; sleep is slow in onset, the patient being kept awake by his fear of sleeplessness, by a rapidly moving circle of uncontrollable memory pictures or by indigestion or palpitation; sleep, when once attained, is not resting or is too short, and the patient awakes in the night or early morning. It is broken by dreams, startings or sometimes emissions. In mild cases distinct *alterations in temperament* and character develop, as *loss of courage* and confidence, depression, *irritability*, undue response to emotional stimulation, as on seeing sad plays or hearing hard-luck stories, slight ethical blunting, tendency towards extremes, and development of quiet self-concentration or obtrusive egotism. The brain tires readily on mental or bodily exertion, especially as the irritable patient is often prodigal of time and energy. It is remarkable how in some cases the morning depression may disappear before an afternoon or evening excitement, but this is notably followed by reaction. Psychical processes are disturbed; perception is blunted and memory is weakened or often temporarily lost (dys-, para-, amnesia), as shown by the copious notes of his symptoms which the patient produces in the physician's office ("*l' homme aux petits papiers*," as Charcot called him); association and combination are difficult, as in thought, composition, judgment or conclusions, whence the speech is often slow and disjointed, names are forgotten and the patient re-reads addresses, runs repeatedly to see if the door was closed, etc. Compulsory conceptions may lead to homicide or suicide in highly psychopathic types. Fear, being an attribute of the weak, is common enough, and may in marked types assume the form of various phobias, as the fear of open places (agarophobia), of enclosures (claustrophobia), rivers (potomophobia), high places (acrophobia), stars (asterophobia), railways (siderdromophobia), dirt (mysophobia), of high things falling (batophobia), of standing (stasophobia), or walking (basophobia), of darkness (nyctophobia), of a single thing (monophobia), of all things (pantophobia), of places (topophobia), of men (anthrophobia) or of disease (nosophobia). The *special senses* are involved. Fibrillation of the lids, and wide, variable, sensitive and unequal (3 per cent.) pupils are frequent, although permanent irregularity almost always results from organic disease. Asthenopia is very common, especially in hypermetropia from weakness of the ciliary and int. recti muscles. The field of vision may be concentrically limited but this is rarely marked or permanent, and Föerster's shifting type may be found, in which objects brought from without into the field are better seen than those moved from within the field outward. Hyperæsthesia of the retina and ear, ringing in the ears, Ménière's complex, and disturbance of taste or smell are not common.

2. Spinal Symptoms.—These include the so-called "spinal irritation." Many symptoms ranked as spinal are essentially cerebral. In women they are more common and are localized chiefly to the upper dorsal and coccygeal regions; in men they are lumbodorsal and most frequent in the sexual type. *Sensory* symptoms are practically constant. Hyperæsthesia is more common in the bodies of the muscles than in their attachments or in the skin; paræsthesia is frequent, as tinglings, girdle sensation or even a sense of itching. Paralgesia in the form of backache (rhachialgia) is as frequent as headache and the pain may radiate into the various nerve plexuses; it is very often sacral; the spine may be exquisitely sensitive to pressure. Paralgesia is common in the skin about the hair roots, in the trunk and extremities. Neuralgia is rare, although it sometimes occurs as a form radiating from the spine into the limbs. Anæsthesia is no part of neurasthenia. Of the *motor* symptoms, muscular weakness, or myasthenia, is the rule. It is probably spinal and cortical in origin and is especially conspicuous in the morning. The muscles often show fibrillary contractions and a *tremor* (85 per cent.) which is as fine and frequent as that of alcoholism or Graves' disease. Muscular fidgets, starts, cramps and increased irritability of the nerve trunks, as in tetany, are also observed. Actual ataxia is most rare, although suggested by the writing. The akinesia algera (Möbius) or the abasia-astasia of Blocq, as in hysteria or the psychoses, is the result of a phobia or a fixed conception. One of Erb's patients remained in bed for fourteen years. The skin, tendon and periosteal reflexes are increased.

3. Vasomotor Symptoms.—These produce many central, visceral and peripheral manifestations. It has been proved by Mosso that during brain-work the cerebral vessels normally dilate while those of the arm contract. According to Anjel this does not occur in neurasthenia. The manometer in normal cases reveals no essential variation during work, while in neurasthenia marked manometric variations occur, as also shown by Weber by the sphygmograph. These findings furnish some diagnostic and prognostic aid, for their intensity parallels the prognosis, but they are especially valuable because they explain many otherwise vague findings. Neurasthenics often suffer from cold, and wear more clothes than is necessary; their skin is cold, even cyanotic or locally asphyxiated. On the other hand, the skin may be flushed and hot. Cold and clammy sweats over the forehead, hands and feet, develop without exertion. Salivation or a dry mouth, polyuria, pseudo-angina pectoris (vasomotor changes in the cardiac and brachial plexuses), the wide temporal vessels, the epigastric pulsation, circumscribed œdema, urticaria, and dermographism are but vasomotor manifestations.

4. Visceral Manifestations.—(*a*) Some of the *cardiovascular* signs and symptoms have already been treated. The arteries are relaxed and often throb, the pulse may be slightly of the "water-hammer" type and capillary pulsation is often apparent in the lips or under the nails. Palpitation and precordial anxiety or dyspnœa are frequent. The pulse is frequently faster and may increase 10 or 20 beats on pressure over some sensitive area (*v. i.* TRAUMATIC NEUROSES). Many neurasthenics seek consultation for cardiac disease. (*b*) *Gastric symptoms* (in 50 to 66 per cent.) are functional in character, and were first described by Leube. The chemism may be normal or show either achlorhydria, or hyperchlorhydria. The stomach is frequently hyperæsthetic. Anorexia is usually mental, as are polyphagia, polydipsia and adipsia (see NEUROSES OF STOMACH). (*c*) *Intestinal symptoms* may develop. Diarrhœa is less common than constipation, which, like previous sexual abuses, often occupies the patient's thoughts. They "would be perfectly well if they could only have one natural movement," and when they do, they complain of "exhaustion during the entire day afterward." Assimilation is often incomplete, as shown by the stools; a sense of pressure, Kussmaul's peristaltic unrest, hyperæsthesia, burning and flatulency torment the patient. Enteritis membranacea and Glénard's enteroptosis are not infrequent. (*d*) The amount of the urine is often increased, or seemingly so, yet equally often the 24-hour quantity is decreased. The specific gravity is lowered, but the urates, uric acid and urea are increased. Phosphaturia and oxaluria occur; the latter is often attended by flatulent dyspepsia, melancholia and nervous depression. Indicanuria is not constant, and glycosuria and albuminuria are accidental. Vesical tenesmus is an occasional complaint. (*e*) *Genital symptoms* may be marked. Fear of the results of previous excesses or of self-abuse may dominate the patient's mind, but the trouble is more a matter of conception than of reality, since the sexual appetite is actually decreased as a rule. In most cases there is premature ejaculation and in some patients there is actual or psychical impotence. Emissions are frequent, but their importance is greatly overestimated.

Course and Prognosis.—*Acute cases* are rare and fatal cases extremely exceptional. The *usual chronic course* is one of slow onset, it is attended by evidences of nervous fatigue which are at first relieved by rest, but which later persist in spite of rest and incline the patient to stimulants or drug habits. Later the signs of cerebral irritability, disturbed sleep, vasomotor disorders, reduced mental and psychical capacity, pains, spinal tenderness, paræsthesia or paralgesia, sympathetic dyspnœa or palpitation develop, sometimes with temporary remissions. The outlook is poor in severe hereditary forms, or when there are complicating psychoses or drug habits. In other types recovery is usual, although it is generally long deferred. Complication with hysteria, epilepsy, migraine, and other neuroses must naturally modify the prognosis.

Diagnosis.—The diagnosis is generally made with ease, but several rules must be borne in mind: (1) No single symptom is pathognomonic; (2) there must be no sign of organic disease; (3) neurasthenia may precede, follow or complicate organic nervous or visceral disease. The *subjective* signs, irritable weakness, emotivity, muscular weakness, headache, backache, insomnia, amnesia, impaired psychical coördination, and the phobias, together with *objective* findings, as asthenopia, insufficiency of the internal recti, tachycardia, epigastric pulsation, vasomotor alteration, as disturbed secretion of urine or sweat, increased reflexes, fibrillation and tender muscles cannot be mistaken. The neurasthenic prodromes of *progressive paralysis*, (*q. v.*) are usually distinguished on careful search for organic disease. *Hysteria* in its stigmata and accidents is so typical that elaborate differentiation is superfluous; anæsthesia, persistent contraction of the fields of vision, dyschromatopsia, achromatopsia, monocular diplopia, convulsions, paralyses, contractures and anuria are foreign to neurasthenia. *Hypochondriasis* differs from neurasthenia in these essential points: it is a pure psychosis, its concepts are primary, they are ill-based, illogical or absurd and cannot be eradicated even for a time.

Treatment.—**Prophylaxis.**—Prevention is difficult, for the neurasthenic cannot control his ancestry, but even with hereditary stigmata much may be done. Treatment starts with the education of both child and adult. Children are mimics and the clearest of all observers. The inculcation of self-control, fearlessness, self-sacrifice, moderation in all things, truthfulness, prompt decision and constancy of purpose must be based on actual parental example, and in this way the child should also educate the parent. The child must learn to give up, to sleep alone, to stop at the right time, to finish what he has begun, to obey, and to learn that he is not the centre of the family. He should be taught without being forced. Out-door living is more important than the school room. The English method of treating children with reserve has some advantages over American demonstrativeness. Simple varied food, without tea, coffee or alcohol, and long sleeps develop a strong nervous system. The sexual instinct requires attention. Parents or patients would do well to read Patrick's small classic, "How Not To Be Nervous"; *Jour. Amer. Med. Assn.*, Feb., 1903.

Therapy.—When once manifested, the disease should be treated as above outlined and causal factors should be investigated. Work alone is seldom injurious, and while it is said that the modern complexity in the struggle for existence is increasing the number of neurasthenics, work should not bear the stigma nor should disappointment, for both make character. Work must be systematic but not slavish. Worry, hurry, failure to learn individual limitations, uncontrolled planning beyond the immediate day's work, and worry (the irritable weakness of work) cause the damage, especially when work produces some indifference to nature, family life, exercise, rest, and the things higher and above one's own small life and ambition. The time for exercise should be taken from the work hours, it should not follow them, nor should it be enforced in every case, for some people are hurt by it. Of course, work under stimulation is injurious. It is held that the chief element in treatment is psychical and surely the physician accomplishes more than do drugs. He carefully examines and re-examines the patient, and his assurance that no organic disease exists, has its weight for the time. Conversely, careless diagnoses, as of fatty heart, with no objective justification, work damage to the patient's mental balance. The patient's hygiene should be regulated. Strict dietaries and stimulants should be avoided, moderate out-of-door life or exercise enjoined, and in very pronounced cases absolute rest from work, with sojourn near the sea or at a moderate altitude in a place not subject to sudden changes or high winds, is recommended. Sexual matters should be left alone unless gross violations are found. The patients need sympathy, which they should receive—in moderation for their sake, and lest the physician become "touched" with neurasthenia; but important as is the psychical element in therapy, the patient must be taught that he alone can cure his malady by self-control, and by avoiding extremes in each and every thing.

Pronounced cases in thin women, especially those with gastric symptoms, are often helped by the Wier Mitchell *rest-cure*, which consists of absolute rest in bed, isolation, forced feeding, massage and faradization. The patient is not allowed to lift a hand, and sees only the nurse, who is selected for her strength, optimism and lack of nerves. The patient is fed milk every two or three hours, and the muscles are rubbed and faradized. Thus weight and strength are gained without muscular degeneration. Few men will tolerate this method, and obese do not fare as well as lean women. In certain cases it is of distinct benefit. However, no greater mistake is made than to give all cases the rest-cure; they often want work and occupation, harmonized with rest.

Diet.—That no one dietary is essential is shown by the different recommendations; some physicians favor a meat diet, others avoid meat, especially in cardiac types, and give well-cooked vegetable albumins, cooked fruit, fats, milk and eggs. In mental anorexia, feeding by mouth or rectum must be forced. Water in abundance is indicated. *Gastric hyperæsthesia* should be relieved by small doses of carbolic acid and gelsemium. Eructations must be controlled, since the air admitted exceeds the gas expelled; asafœtida is excellent because it makes as strong an impression when swallowed as when eructed. Electricity in general is largely suggestive in its effects; cerebral galvanization gives uncertain results. With massage and vegetable diet, endorectal faradization relieves *constipation*. In cardiac types alcohol and tobacco are interdicted, and small doses of belladonna and bromide usually control *palpitation;* other vasomotor symptoms are relieved by these remedies, or by ergotin or nitroglycerine, according as they are angioparetic or angiospastic. Arsenic is valuable in vasomotor troubles with anæmia.

Sexual symptoms are treated by psychical measures and by sitzbaths. *Insomnia* is often difficult to relieve. In the order named, the following measures are valuable: avoidance of evening work, stimulants or excitement; a cool bath, the wet pack or an ice-bag over the heart; small doses of bromide with valerian for the "expectant attention" which wards off sleep; 20 grains of sulphonal in hot milk, which should be slowly sipped; codeine with hyoscine; and chloral (the continued use of which induces vasomotor disturbance). The habit of sleep is like the habit of insomnia; it must be cultivated, and the patient must not get up to read. Local, urethral, and uterine affections usually receive excessive therapeutic attention.

THE "TRAUMATIC NEUROSES."

This is a practical grouping of traumatic neurasthenia, hysteria, chorea or hypochondriasis. Hysterical forms were first described by Walton and Putnam, Erichsen (1866) wrote on the subject of trauma to the spine, on which the illogical name of railway spine was later conferred, and Oppenheim (1887) termed the group the "traumatic neuroses." All organic affections must be excluded, whether trauma may seem directly or indirectly causal, as vertebral caries, tumor, Bechterew's chronic ankylosis, Kummel's rarefying osteitis which Kocher refers to fracture; hæmatorrhachis, meningeal hæmatoma, or pachymeningitis; and cord affections, as poliomyelitis, myelitis, or lateral sclerosis. Experimental trauma may produce molecular nervous changes (Schmauss, Strümpell, Bruns, Vibert and Crocq), a fact which may be of clinical importance, and trauma is thought to hasten arteriosclerosis (Friedmann). Alcoholism and syphilis are predisposing factors. "*Traumatic neuroses" are purely functional affections caused by trauma*, which operates psychically rather than somatically. The symptoms are those of hysteria, neurasthenia, and hypochondriasis, singly or in combination. The patient may feel well for a day or two and then pain develops at the seat of injury or in the spine; anxiety, fear for his financial future, or for his family, a "fixed idea" of his incapacity, and the development of "litigation symptoms" are typical of hypochondriacal neurasthenia. The *psychical symptoms* are usually but not always prominent; they are accentuated by the effect on the patient of repeated, sometimes suggestive, physical examinations, visits of lawyers or adjustors, court room suspense, reversals of judgment or appeals. Vertigo, backache, headache, sleepiness by day, and nocturnal insomnia may cause great distress. *Sensory symptoms* may be present. Pain, which seems to be nervous, may prove organic on examination by the x-rays; it may be simulated, but is often real, as shown by persistence after award of damages. Tender areas are difficult to pass upon, because of exaggeration or simulation; Mann, Köpf and Rumpf found that pressure on areas which are actually tender increases the pulse rate by 10 to 30 beats; this sign is subject to several conditions: (*a*) it is not always present in real pain; (*b*) its absence is no proof of simulation; (*c*) in simulation, the agony and writhing which occur when certain areas are touched may increase the heart's action. Paræsthesia and hyperæsthesia of the skin or special senses are frequent. Anæsthesia indicates traumatic hysteria. Hysteric or neurasthenic contraction of the visual fields may remain the same from day to day, and not vary with approach or withdrawal of the test object; this is not a sign of simulation as suggested by some writers; Förster's type (*v.* NEURASTHENIA) cannot be assumed. In all cases the influence of suggestion must be borne in mind. The pupils may vary, especially by dull illumination; reflex immobility indicates organic disease. *Motor symptoms* may embrace all those of hysteria or neurasthenia, abasia, astasia, paralyses, contractures suggesting vertebral injury (Kummel's rarefying osteitis) or joint disease (Brodie's joints), pseudo-spastic or -tabetic gaits, tremor (*forme trepidante*), or sometimes muscular atrophy (especially about the joints). Hysterical convulsions are rare; the author has twice seen choreiform affections in telephone girls, who sustained slight shocks and once a genuine chorea. Patients must often be watched; one of the author's cases, with spastic gait, shuffled out of the office, but walked perfectly well around the corner. The *reflexes* are often increased, perhaps asymmetrically; ankle clonus, which is probably spurious, is sometimes reported. Cardiac symptoms may be prominent and cannot be simulated. When there is albuminuria and glycosuria, the question naturally arises whether they were present before the trauma.

Diagnosis.—The diagnosis concerns several points which are often difficult or impossible to establish, because objective findings are frequently few. (*a*) It is usually possible to state whether one of the neuroses is present or whether organic disease obtains. (*b*) Exaggeration and simulation are difficult to estimate, as shown by the change of professional opinion from the idea that all or most cases are simulated to the present statement of some writers that these factors are present in 4 (to 10) per cent. of cases only. (*c*) If disease is present, is it directly due to injury? (*d*) If so, the most difficult of all questions is presented, namely, the degree and duration of the disease, and the damages to be awarded.

Prognosis.—(*a*) *As to life*, the outlook is usually good, though some cases become insane or suicidal. (*b*) *As to complete recovery*, it is less favorable. In most cases it is surprising how soon symptoms disappear after satisfactory legal settlement is made; this is said without cynicism. Many persons do not then recover completely and symptoms may endure in those who persistently shrink from litigation. Hysterical symptoms are more favorable in prognosis than the neurasthenic or hypochondriacal. It is maintained by some that cardiac overaction may induce organic myocardial changes, and that arteriosclerosis may develop into precocious senility (dementia præcox).

Treatment.—Treatment is similar to that of hysteria or neurasthenia and is therefore largely psychical. The physician, as a rule, dreads the effect of litigation on the patient, assures him that the symptoms are seldom severe or enduring, that there is no organic lesion, and advises early settlement and speedy return to work.

EPILEPSY.

Definition.—Epilepsy, by derivation, means "being seized upon"; it has been called the "falling sickness," and morbus sacer. It does not include Jacksonian or symptomatic epilepsies, which will be considered in its differentiation. Though it is classified with hysteria and neurasthenia as one of the "three great diffuse neuroses," it will probably be finally shown to include more than one disease. It is a *disturbance of balance between the central exciting and inhibiting nervous processes* attended by (*a*) a typical *chronic convulsive disorder*, affecting all parts of the central nervous system, each attack being followed by *loss of consciousness*, (*b*) *partial or atypical manifestations* of these characteristics, or (*c*) concomitant or consecutive psychical or other symptoms. It occurs in from one to six persons per thousand.

Etiology.—The *ultimate causes* must be separated from the inciting causes (Nothnagel). Of the former, the *neuro-* or *psychopathic tendency* of Griesinger is the most important, being found in 35 to 50 per cent. of cases and causing lessening of the nervous resistance. As in tuberculosis, only the tendency is transmitted by inheritance, rarely the disease itself. Neurotic or psychopathic antecedents, parental alcoholism (51 per cent.), morphinism, plumbism, syphilis, tuberculosis, gout, arthritism, diabetes, or ovarian and testicular disease, lessen the nervous resistance of the offspring. Trauma during labor may be included under this head. Though the disease may appear at any age, it rarely occurs after the thirtieth year. Seventy-five per cent. of cases (Gowers) develop before twenty; 46 per cent. between ten and twenty, mostly at puberty; 28 per cent. before ten. The sexes are about equally affected.

Inciting causes include (*a*) *infections* as malaria or typhoid; syphilis may be hereditary, induce malnutrition in the secondary stage, of the Jacksonian type from cortical gumma, or it may be parasyphilitic; (*b*) *intoxications*, especially alcoholism; (*c*) *exhausting affections* as rickets, and diseases of the blood or of metabolism; and (*d*) *trauma* or fright. Inciting causes are found in only 37 per cent. of cases (Gowers).

Reflex epilepsy is said to occur from painful scars, nerve tumors, or foreign bodies in the ear, nose, nasopharynx or larynx; from diseases of the teeth, digestive tract, uterus, and heart; from onset of the menses or their monthly appearance, pregnancy, phimosis, or onanism; from gall-stones, colic or abdominal paracentesis. While these factors may appear in certain cases and attention must be given to the general examination and health, the importance *per se* of reflex disturbances is daily lessening.

Pathology.—The fact that there is no characteristic macro- or microscopic change is not proof that some cortical change will not later be demonstrated. The usual finding after death from an attack is that of any convulsion, *viz.*, venous engorgement and punctate ecchymoses. Induration of the cornu ammonis (Meynert) (3 to 38 per cent.), induration in the medulla, neurogliar increase in the cortex, thickening, asymmetry or deformity of the skull, or persistence of the thymus, have not yet been proven to exist constantly in epilepsy, nor are they proven causal.

Symptoms.—The symptoms are best studied from the *completely developed attack*, in which there are three component parts: (*a*) The *prodromal stage* is not always present. Its "warnings" are either distant or immediate. The distant warnings (in 4 to 17 per cent. of cases) may precede the seizure by hours or days, are possibly due to metabolic changes in the cortex, and take the form of restlessness, dispositional changes, depression, thoracic or cephalic oppression, insomnia, vertigo, nausea, grinding of the teeth, hyperæsthesia of the special senses, angioneurotic palpitation, or cerebral congestion. The immediate warning is the *aura* proper, which is the beginning of the convulsion rather than a warning. It is a central cortical irritation; its frequency ranges between 30 and 50 per cent.; it is rarer in sudden, stormy, generalized seizures. Its forms are varied. The psychical aura is rare, and is manifested by emotional or intellectual alteration and anxiety. The sensory aura consists of paræsthesia, and a sense of deadness in a limb or joint, attacks of migraine, and epigastric sensations (from the pneumogastric nerve, pressure or irritation of which sometimes inhibits the convulsion). The special senses may be irritated or blunted; for instance, there may be flashes of light or colors, hallucinations, blackness before the eyes, and less often anomalies of hearing, taste or smell. The motor auræ are circumscribed clonic or much less often tonic twitchings, as in the signals of Jacksonian epilepsy, from which they are distinguished by a rapid loss of consciousness; automatic movements, like stamping, or buttoning the coat, are rarer, and motor weakness and singultus, coughing or sneezing are least frequent. Vasomotor auræ may be localized or generalized, as rushing of blood to the head, palpitation, anæsthesia, increased temperature, increased arterial tension, pallor or chilling. The patient then falls, *pale and unconscious*, or is thrown with force, often on his face (Trousseau), when (*b*) the *convulsive stage* begins. There is first a *tonic* convulsion, which affects all voluntary muscles, and is due to infracortical irritation. The initial *cry* is gutteral or groaning, results from tonic abdominal, thoracic and laryngeal spasm and is heard in 50 per cent. of cases. The head is held back; the eyes are open and staring, the jaw set, the face flushed, the neck tense, the body usually opisthotonic, breathing is suspended, the muscles are hard but slightly vibrating to the palpating hand, the arms are turned tetanically inward, the hands are clenched with the thumb adducted and under the fingers, the legs are extended, the thighs adducted and the toes flexed or extended and spread apart. In some cases the head, eyes or even trunk deviate, and in a few instances the tetanic spasm spreads gradually, instead of the patient having the usual instantaneous tonic spasm. The tonic phase lasts ten, fifteen or thirty seconds, relaxing in inverse order from the limbs to the neck and face. The second phase of *clonic convulsion* occurs at once, appearing first in the limbs, which are not yet quite free of the tonic spasm. This stage is usually bilateral, highly irregular and violent, with very short remissions, leading perhaps to injury, fracture, luxation, breaking of teeth, laceration of skin, tongue and cheek, or rupture of muscles. It consists largely of alternating flexion and extension; the head strikes the ground, the body turns violently, the eyes jerk and protrude, the face is distorted, the tongue is bitten as it is thrust out, bloody saliva is expressed by the masseters, inarticulate sounds arise from the movements of the diaphragm and respiratory muscles; and gurgling results from intestinal movements; vomiting movements, filling of the stomach with air, cyanosis, swelling of the jugular veins, ecchymoses in the conjunctiva, skin, retina or larynx, involuntary and sometimes forcible evacuation of the bladder, less often of the rectum, and occasionally ejaculation, occur. The clonic phase lasts one-half to three minutes, ending in a fine generalized tremor. (*c*) The *comatose* stage often begins with a deep sigh; respiration and circulation become normal, the limbs relax and the condition resembles a sound sleep, from which the patient awakes in from fifteen minutes to several hours, sometimes suddenly, more often gradually, and in a more or less dazed condition. He recalls nothing of the attack and the loss of memory may extend back of the seizure (retrograde amnesia). He experiences pain in the muscles, joints or head, nausea, mental irritability or depression and finally a group of exhaustion symptoms, to which Féré has especially directed attention; these are (i) sensory exhaustion, expressed by hypæsthesia, anæsthesia, hypalgesia or analgesia. (ii) Exhaustion of the special senses, as concentric limitation of the visual and color fields, especially in abortive attacks, dulling of hearing, taste or smell, and (iii) motor exhaustion which, like (i) and (ii), is due to cortical fatigue. The reflexes are often weak or abolished.

The seizure has been described with special reference to the dominant motor changes, but during the attack other findings are observed. *Vasomotor* constriction often occurs, as shown by the initial pallor of the face, cardiac overaction or perhaps irregularity. The ecchymoses and retinal congestion are probably due to the convulsion. The *reflexes* are affected. The pupil is almost always reactionless, is often narrow at the onset, and usually dilates widely in the second stage; afterwards it may oscillate; the pupils are sometimes unequal (20 per cent., Féré). The tendon and skin reflexes are weak or lost in complete attacks and for hours afterwards, whence we may judge of the severity of the seizures. The temperature is usually considered normal, but may be elevated 2 or 3°. In the urine, Voisin found albumin in 50 per cent., though others consider it infrequent. The author has often noted transient albuminuria and cylindruria (granular casts) after marked paroxysms; glycosuria is uncommon. The toxicity of the urine, the phosphates and the nitrogen are increased; polyuria is common in the third stage. The sweat is increased.

Types of Epilepsy.—1. The completely developed attack (*epilepsia gravior, haut or grand mal*) consists of (*a*) the classic type (29 per cent.), as above described, with complete loss of consciousness, and tonic followed by clonic convulsions; or (*b*) the well developed but slightly atypical type (19 per cent.), in which all the components of (*a*) are present but vary somewhat; for instance, the spasm may not always be generalized.

2. *The rudimentary attack* (21 per cent.), consists of loss of consciousness, plus tonic or clonic spasms, but not both. This includes the apoplectiform cases, especially the senile (arteriosclerotic) form; the forms resembling myoclonus; the cases which do not fall but stumble along at the onset (epilepsia procursiva) or turn (epilepsia rotatoria).

3. The third form is the *abortive attack* (epilepsia mitior, *petit mal*, 31 per cent.). Its importance is underestimated by the profession and the public; two main types, with endless variations are distinguished: (*a*) one with loss of consciousness with few or no motor signs, and (*b*) motor and vasomotor signs with no loss of consciousness, or very slight clouding of the intellect. The loss of consciousness is often very slight, or may last for a few seconds, so that a scarcely perceptible break occurs in the patient's occupation, reading, speaking, playing the piano or a game of cards; it is described by the French as "*absence.*" He may move his lips inarticulately or there is slight nystagmus, a chewing or swallowing movement, tremor

or twitching of the face, monospasm, or vertigo. In children, a cry may be the sole symptom. Some patients seem to fall suddenly asleep. Nothing, or everything, may be remembered. Auræ are very common and may constitute the whole attack, which aborts without loss of consciousness or spasms. Vasomotor symptoms are very common. Some would include the rudimentary type under this head.

4. The *psychical equivalent* of Sammt, which is also known as transformed or larvated epilepsy or the epileptoid condition (Griesinger), is often mistaken. Mental symptoms may precede, succeed, alternate with or replace the ordinary convulsive type, which must be first recognized to establish this variety. The mental changes are slight (*petit mal intellectuel*) or marked (*grand mal intellectuel*) and include excitement, emotional outbursts, narcolepsy, incoherent conceptions, amnestic attacks or even mania. The patient may act like an hypnotized subject; he may take sudden long journeys, or commit assault, arson or murder, whence the great medico-legal significance of this variety. Milder forms are said to occur, as sweats, stupor or vacuity at the menstrual epoch.

The Interparoxysmal State.—If the patient is not to be seen during his seizures, evidences of epilepsy should be looked for carefully. Signs of psychical degeneration, poor differentiation of the ear, very large, small or prominent ears, hæmatoma of the ear, deformed teeth, lips or palate, coloboma, congenital strabismus, nystagmus, small eyes, irregular pigmentation of the iris, deformed genitals or fingers, disorders of speech or irregular facial innervation may indicate hereditary taint. Neurasthenic symptoms are not unusual. The expression is often dull. When idiocy occurs with epilepsy there are two possibilities; first, that both are coördinate results of a common cause, or second, that early, chronic and repeated attacks have initiated mental degeneration. However, many brilliant men have been epileptics, as Julius Cæsar, Napoleon, Carl V, Peter the Great, St. Paul, Mohamed, Petrarch and Rousseau. The importance of organic disease of the heart, lungs, stomach, and kidney, is difficult to estimate. It is possible that the future study of metabolism may establish the auto-intoxication theory. Epileptics often sweat profusely, are usually constipated and frequently become obese from inordinate eating and from the bromide treatment.

Diagnosis.—The diagnosis concerns the convulsive seizures and the abortive types. Similar convulsions occur in numerous other conditions and must first be excluded. They occur in (*a*) organic brain disease, as syphilis, tumors, vascular lesions, paretic dementia, multiple sclerosis, and infantile cerebral paralysis. These affections may produce generalized or Jacksonian fits. *Jacksonian epilepsy* is more clonic (from cortical irritation) than tonic (due to infracortical irritation), is often associated with paresis or sensory alteration, occurs less often with coma unless its generalization is unusually wide, and in 50 per cent. of cases is due to small cortical tumors. Epilepsy rarely (E. tarda) develops after thirty years of age, and 90 per cent. of cases which occur after thirty,—excluding alcoholism and uræmia,—are syphili (Fournier, H. C. Wood). After forty-five, vascular lesions are a ve common cause. In some cases true epilepsy appears to follow th Jacksonian type. (*b*) Convulsions may occur in altered conditions o the *blood*, as alcoholism, *uræmia* (eclampsia), plumbism or dru poisonings. (*c*) *Reflex epilepsy* results from dentition, worms, o digestive disorders; in some cases genuine epilepsy seems to follow the reflex form (which perhaps is epilepsy in itself).

Simulation of epilepsy is impossible. In the complete type, the tongue biting, cry, stertor or involuntaries may be absent. Scars are often of diagnostic value. Nocturnal epilepsy occurs in 33 per cent., often coming on when the patient drops off to sleep or on awakening in the morning; it easily escapes recognition for years, but the depression, bitten tongue, ecchymoses and involuntary evacuations are unmistakable.

Differentiation from hysteria, which is usually easy, may be difficult, indeed the two affections sometimes co-exist; the following table is combined from Gowers and Biswanger:

EPILEPSY.———*vs.*———HYSTERIA.

Distant warnings: few, mental.	Frequent, emotional.
Aura: more frequent, momentary, oftenest unilateral or epigastric.	Less frequent, longer, bilateral foo aura, globus.
Cause: none.	Emotional.
Onset: always sudden, often with pallor, cry which is never verbal, immediate falling and complete unconsciousness, with rigidity, etc.	More gradual, rarely with pallor or cr which may be repeated or verbal, a recurs later. Rarely brusque fallin slower and far less complete uncon-sciousness, and may react to sugges-tion or pressure on hysterogenous zones.
Tongue biting, frothing: very common.	Exceptional, or seemingly purposive.
Convulsions: generalized, tonic followed by clonic spasms, very rarely tonic alone; flexor spasm predominates.	Rigidity, to carry out semi-intentional movement; struggling; clonic, irreg-ular spasms, seemingly fragments of partial movements of emotion; ex-tensor spasm predominant "arc de *cercle*," clownism, passional attitudes, etc.
Reflexes: pupils contracted at onset, dilated later, immobile (in clonic period). Tendon and skin reflexes absent during and after attack.	Pupils usually wide and react (may be immobile, Westphal, Karplus). Sometimes convergent strabismus. Reflexes not absent or increased.
Involuntary evacuations: frequent.	Absent or most exceptional.
The urine: solids always increased, both nitrogen and phosphates.	All solids decreased and altered ratio between earthy and alkaline phos-phates.
Temperature: sometimes elevated 2–3°.	Practically never.
Coma: complete.	No real coma.
Duration of seizure: very short.	Longer, recurrent, relapsing.
Post-paroxysmal: sensory and motor exhaustion, ecchymoses, absent reflexes, mental incapacity, with no memory of transpired events.	Depression, fatigue, pain in head or stomach, nausea, some memory of attack

The most difficult forms to differentiate are *petit mal* and transformed epilepsy, in which the history or observation of major attacks is important. *Petit mal* may be confused with cardiac syncope, labyrinthine vertigo, etc.

Course and Prognosis.—When one attack has occurred, a second may be expected in 33 per cent. of cases in less than one month, in 33 per cent. in from one to twelve months, in 33 per cent. in over one year; and when epilepsy is once established, daily attacks occur in 10 per cent., in 50 per cent. they occur at intervals of less than two weeks, and in 75 per cent. they occur at intervals of less than one month (Gowers). The mode of life, stimulation, excitement, pregnancy, and sexual excesses may increase their frequency, and cessation during fevers is not uncommon. In some, though fortunately rare, cases, the attacks occur in quick succession (*status epilepticus, état de mal*), as many as 10 or 30 or over 100 occurring in one day. In one case 2,500 seizures occurred during a month; the stage of coma may last from two or three days to a week, with a temperature of 105–7° and death then results. The *outlook (a) as to recovery* is poor. Five to 10 per cent. of cases may recover spontaneously (Hufeland, Dana, Müller). The prospects are best in cases which begin before twenty, in males, in cases with long intervals, and, though it may seem strange, in hereditary forms. After two years' duration, the outlook is unfavorable. (*b*) *As to life*, the prognosis is more favorable. A few cases drown, suffocate from turning on the face, or are fatally burned during the seizure. Some patients die during the insult from vascular lesions of the brain, or less often from heart rupture, respiratory cramp, œdema of the lungs or vomiting into the larynx. The status epilepticus and delirium tremens are dangerous. Epileptics frequently become tuberculous. In 10 per cent. dementia occurs. The average duration of life is probably shorter than normal. Bodily health and intellectual vigor may, however, be remarkably preserved.

Treatment.—**1. Treatment of Cause.**—This has a limited scope. Syphilitic epilepsy, unless due to a gumma or to the secondary anæmia, does not respond to antisyphilitic remedies. Avoidance of alcohol may be almost curative in this form. Reflex factors should be removed. In Sweden epileptics cannot marry; 50 per cent. of hereditary epileptics transmit some transformation of the taint to their offspring.

2. Hygienic Therapy.—New cases in children should receive the general care outlined under the other neuroses. Work in school should be limited to a few hours, and country life is generally preferable to that in the city. For very poor subjects, institutional treatment is indicated. Erb uses galvanization through the head. Most neurologists greatly restrict the consumption of meat, although Gowers allows it once or twice a day. Toulouse and Richet withdraw salt, and give a milk and cereal diet, which they think promotes the affinity of the bromides for the brain cells.

3. Bromide Treatment.—This was introduced by Laycock (1853), and was developed by Voisin. The bromides act directly on the nervous tissue; they depress the cortical cells, with which they org ically unite, inhibit reflex action in the brain and cord, and les centripetal conduction. *Untoward symptoms may result even wit therapeutic limits.* Intellection may be dulled and muscular depr sion occur, but these may often be counteracted by administrat of coffee; the frequent stomach symptoms can be avoided by f dilution of the bromide in water; diarrhœa, which results especia from potassium bromide, is relieved by opium if marked, or if less, salicylate of bismuth and beta-naphthol, āā gr. x, after meals; in elimination by the skin, acne is common, which is lessened or removed by giving large quantities of water and small doses of arsenic, and by washing the skin with green soap. Belladonna in small doses will modify the respiratory catarrh or salivation. The anaphrodisiac effects cannot be prevented. In some individuals there are *marked idiosyncrasies*, as mental excitement, hallucinations, rapid emaciation ("bromide cachexia") with lessened physiological resistance to acute diseases. *Toxic symptoms* may be *acute*, resembling those of paretic dementia, but more commonly are chronic; *chronic bromism* is most likely to develop in those with weak digestion or circulation and when the drug is given carelessly for a long time, because its elimination is slow. In the former, hydrotherapy, massage and tonics should be used as preliminary or concomitant measures. Cardiac symptoms are due almost wholly to the potassium salt, but especial care in the use of all bromides is indicated in cases of senile epilepsy (myocarditis, arteriosclerosis, endocarditis) or of great nutritive exhaustion, in which, combination with digitalis or strychnia is indicated. In bromism the reflexes are decreased, particularly the pharyngeal reflex, but Englehardt has shown that it is weak in 17 per cent. and absent in 25 per cent. of normal individuals, whence Ziehen watches the corneal reflex, which is never normally absent. In advanced bromism there is low temperature, bronzed skin, vasomotor relaxation, somnolence, apathy, paralytic speech, muscular relaxation, ataxia, tremor, fœtor ex ore, anorexia, slow respiration (the usual cause of death), weak heart, abolition of sensation in the skin and mucous membranes and of all reflexes, and decrease or suppression of urine. The drug therapeutically unites with the nerve cell and depresses its activity and metabolism, and toxicologically it causes its degeneration or atrophy. The drug must be used for months or years, and the symptoms above enumerated must be watched for; it seems that epileptics have a certain tolerance for bromides, which Voisin considered almost as a food in this affection.

Results.—Gowers noted immediate relief in 43 per cent., improvement in a short time in 47 per cent., and no effect in 10 per cent. of his cases. He gives a dram a day of the potassium salt, for two years, for he finds that larger doses are no more efficacious; in nocturnal epilepsy a large dose should be given at bed-time. Seguin's results were: cessation of attacks in 23 per cent., notable decrease in 40 per cent., no great effect in 26 per cent., no effect in 10 per cent.

Charcot's method was to give a daily dose of one dram, which was gradually increased to nearly two, and was then slowly decreased to the original amount. Erlenmeyer's formula is extensively used, and gives the best results (sodium and potassium bromide each two parts, and ammon. bromide one part). The best results are obtained in cases which are treated early in their course and early in life, and in those in which there are long intervals between attacks. Opium, belladonna, nitroglycerine, digitalis and chloral are of but secondary importance, although with special symptoms they may reinforce the bromides. Nitroglycerine may be useful in *petit mal*. Children tolerate large doses of bromides (as well as of mercury, belladonna and arsenic).

4. Treatment of the Attack.—This is confined to cases in which auræ are present. The patient is placed on the bed or ground to avoid injury, and a towel inserted between the teeth to prevent biting of the tongue. Compression or manipulation of the limb in which the aura is felt may in a few instances avert the spasm. Charcot placed an ice-bag over the head and one over the heart. Inhalations of amyl nitrite, ether or chloroform are sometimes beneficial.

5. Surgical Indications in Traumatic Epilepsy (Jacksonian or generalized).—Biswanger's conclusions are (*a*) that results are disappointing; only a small percentage of cases actually recover; operation is indicated (*b*) when convulsions emanate from a small cortical focus; (*c*) when the convulsions are of a type intermediate between the Jacksonian and generalized form; (*d*) when, with generalized convulsions, there is a suggestive scar, bone defect or localized tenderness on percussion; (*e*) since the internal lesion does not always correspond with the external scar, the site of trephining should be governed by focal symptoms, motor or sensory auræ, or "exhaustion" signs; (*f*) cysts, tumors, bone, membrane or scars may be removed, but not the cortex (as recommended by Horsley); (*g*) operation must be followed by the bromide treatment; (*h*) operation is contra-indicated in ordinary epilepsy.

INFANTILE CONVULSIONS.

In young children the nervous tissues are incompletely developed, many axis cylinders are not yet clothed with myelin sheaths, and the lower dominate the upper segments, thus predisposing the child to disproportionate nervous reaction.

Etiology.—(*a*) *Rickets* as a cause was especially emphasized by Wm. Jenner. Many cases of carpopedal spasm, tetany and laryngismus stridulus have a rhachitic basis; this form occurs in infants between the eighth and twenth-fourth months of life, is likely to recur, and disappears after the second year. (*b*) *Reflex spasms* may be caused by peripheral irritation, as from dentition and worms, though the importance of reflex irritation has been greatly overestimated; gastro-intestinal irritation or auto-intoxication, phimosis, and otitis media, are possible causes. (*c*) They may be due to *gastro-intestinal intoxication*, which results in debility, malnutrition or a "hydrocephaloid" condition (Marshall Hall), which has been described above under various captions and is usually of most serious import. (*d*) *Nervous affections*, trauma during delivery, meningitis, tumor, sinus thrombosis, severe coughing, acute encephalitis (cerebral hemiplegia), or acute poliomyelitis, are etiological factors. (*e*) They may result from *acute infections*, as scarlatina, measles, or pneumonia, in which convulsions and vomiting rather replace the chill which is more common in adults. (*f*) Genuine *epilepsy* may begin in early life; 12 per cent., according to Gowers, and 40 per cent., according to Osler, occurs in the first three years of life.

Symptoms.—The symptoms resemble those of epilepsy in most of its phases, but they are, however, less developed in the eclampsia of children. It usually begins in the hands, and the seizure is partial, tonic, intermittent, and carpopedal rather than generalized. Screaming and grinding of the teeth may occur. The laryngeal spasm has already been described under affections of the vagus.

Treatment.—The occurrence of the *convulsion* is the first indication for treatment, and should be met symptomatically by inhalations of chloroform or nitrite of amyl, followed by grs. v to x of chloral, and ʒss of bromide by rectum. The bromide should be given for several days, for its effects are cumulative rather than immediate. Opiates should generally be avoided in children under five years, especially in nurslings; if given to them at all the dosage should be one-half the dose according to the rule of age, *i. e.*, $\frac{1}{26}$ of the adult dose in a child of one year, instead of $\frac{1}{13}$ of the adult dose. Opiates should not be used in weak or marantic infants. The hot bath is of little service.

The *cause is then determined* and treated, as (*a*) the rickets, and (*b*) the peripheral irritation; lancing of the teeth is practiced less and less; (*c*) in gastro-intestinal intoxication the stomach should be emptied by ipecac or by means of the stomach tube; lavage of the bowel is indicated in acute cases and stimulants should be given in cases of some standing. (*d*) The bromides should be administered as in epilepsy.

CHOREA AND CHOREIFORM AFFECTIONS.

The name is given to several convulsive maladies. Chorea, or St. Vitus dance, known to Paracelsus, was endemic in the middle ages and is now often known as chorea major; it was hysteria or possibly epilepsy. Chorea in its usually accepted sense was described by Sydenham and is known as Sydenham's chorea, chorea minor, or infective chorea.

Definition.—Sydenham's chorea is (*a*) a neurosis of late childhood; (*b*) it is characterized by subacute onset, limited duration and, usually, recovery; (*c*) it is probably cortical in localization, as indicated (*d*) by involuntary irregular spasmodic movements, and (*e*) increased

coördination on voluntary efforts; (*f*) it is always associated with
me psychical weakness, irritability or alteration in character and
) is frequently associated with rheumatism and (*h*) endocarditis.

Etiology.—(*a*) *Age*: 80 per cent. of cases occur in persons between
e fifth and fifteenth years; 90 per cent. in those between the fifth and
entieth years; it is most frequent at the sixth and seventh year of life,
at puberty. (*b*) *Sex*: Seventy per cent. of cases are in girls (Gowers,
sler). (*c*) *Rheumatism* has been considered a cause since Bright's
ne and this theory has been established especially by English writers.
he French writers probably overestimate its importance, and the
ermans apparently underestimate it. Rheumatism is a factor in at
ast 20 to 25 per cent. (Hughes and Sée); some consider it causal in
or even 70 per cent. of cases. *Endocarditis*, *rheumatism* and *chorea*
e too frequently associated to escape serious attention. Rheum-
ism occurs mostly before the chorea, but in 4 per cent. of cases
the two occur simultaneously. Their association is most frequent
the young, but their exact relation cannot be established until the
use of rheumatism is known. Rheumatism, endocarditis and chorea
e probably correlated, *i. e.*, are due to a common virus. (*d*) *Other*
ute infections are causes in 43 per cent. of cases (Neumann). Chorea
s been observed after gonorrhœa, puerperal fever, sepsis, pertussis,
easles, typhoid, etc. (*e*) *Pregnancy* is a predisposing cause in women,
rticularly before the twenty-fifth year. Chorea occurs in the first
ree months, especially in first pregnancies and is most severe in
aracter. It rarely occurs postpartum. Giles de la Tourette justly
lds that many cases classified as chorea are really hysteria. (*f*)
europathic heredity is observed in 16 per cent. of choreic subjects,
ich probably explains its frequency in Hebrews. This factor has
emingly been overestimated. (*g*) Other causes are sometimes
erative, as fright, trauma, and reflex irritation from nasal or digestive
sease, as worms. It occurs in the lower classes, is sometimes due to
doform or mercurial poisoning, apparently occurs when the baro-
eter is low, and is influenced by the poor hygiene, excitement,
hibition and competition of the school-room; this is called the
school-made chorea" by Sturges.

Pathology and Pathogenesis.—The disease is a provisional neurosis,
nce no constant pathological findings exist. The following changes
ve been recorded; hyperæmia, exudation about the arteries, areas
softening, punctate hemorrhages, round hyaline bodies in the peri-
scular lymph sheaths (Flechsig, Wollenberg, Jakomenko), and
elling and turbidity of the large pyramidal cells of the cortex (Tur-
r); but none of these are characteristic or constant. In fatal cases
docarditis is the most frequent finding (90 per cent. Osler, 94 per
nt. Sturges), and Osler notes that its frequency is greater even than
rheumatism. Kirke's theory of cerebral embolism has been rather
enerally abandoned. The bacteriological findings are negative or
onflicting. It is probable that the irritation, whether the result of

exhaustion or of toxæmia, is either cortical or in the basal ganglia, which are closely connected with the cortex or subcortex.

Symptoms.—1. Onset.—Prodromes are infrequent, but sometimes there is a subacute onset, with irritability, restlessness, depression, disturbed sleep, headache, anorexia, rheumatic pains or sensory irritation. The affection usually begins with motor disturbance, in the form of *involuntary movements* and *incoördinate voluntary movements;* the latter are often misunderstood and punished, for the child cannot sit still, or breaks or drops things.

2. Chorea Movements.—These are *involuntary,* although they may seem voluntary, as the thrusting out of the tongue or the reaching of the arm at an object. They are "*irregular in time, character* and *degree,*" are often long, are not usually attended by fatigue or pain, are incomplete and without motive, are increased by attention being drawn to them and by excitement and almost always cease during sleep. They usually begin in the arms, and later extend to the face and legs. Chorea very often begins on one side, especially the right, whence it extends to the other side but sometimes remains unilateral (*hemichorea*); both sides, however, are always involved to some extent. The face is contorted with varying expressions, now grimaces or again motiveless smiles; this is the *facies choreatica* of Sachs. The eyes roll, are disturbed especially when attempts are made to fix them, and diplopia is not uncommon. The pupils are often wide and react slowly. Tuckwell describes a case in which the teeth were broken. The author recently saw with Dr. Halbert a similar instance in which many of the teeth were broken. Speech is involved in 25 per cent. of cases; this is due rather to difficult articulation by the tongue and lips than to phonation. Speech is quicker than normal, like the movements of the arm, and may be aggravated by irregular movements of the glottis and respiratory muscles, resulting in panting, irregular inspiration or expiration or sobbing sounds in which the diaphragm participates. Aphasia is less frequent. The *arms are most involved;* they are flexed, extended, pronated and supinated in tireless succession. Bouillaud spoke of the movements as muscular insanity (*folie musculaire*). The *trunk* is sometimes involved in light cases, and distinctly so in the severe types. The movements are sometimes so violent as to throw the patient out of bed. The *legs* are usually much less involved; there is a zig-zag gait, and walking may be impossible. Besides hemichorea, other irregularities are at times encountered, as monochorea, chorea paraplegica, or chorea alternans. The *muscular power* and endurance are often said to be normal. Some writers, as Wallenberg, insist that paresis is no part of chorea, but Gowers and Osler speak of marked muscular weakness. Two forms are distinguished: (*a*) paresis with chorea, in which the muscular power is reduced, as in the usual case, and (*b*) limp or paralytic chorea (Todd, West, and the *chorée molle* of French writers). The paresis may be of the mono-, para- or hemiplegic type.

3. **Psychical Changes.**—These are constant, and resemble an acute cerebral neurasthenia in the undue irritability and ready fatigue which are manifested. The subject is wilful, forgetful, emotional, irritable, unable to concentrate the attention or to remember perfectly. Marked alteration in addition to these changes may occur, as complications, *viz.*, delirium, mania, melancholia or hallucinations; *chorea insaniens* has a most unfavorable prognosis. Combination with hysteria is frequent, with epilepsy infrequent. Hughlings Jackson found paroxysmal headache in 90 per cent. of cases.

4. **Sensation.**—Sensation is usually normal. Tenderness over the spine and peripheral nerves is not common, though S. Wier Mitchell and Triboulet described a painful chorea. Occasionally there is a tingling sensation and numbness. Blocq and others have insisted that sensory alteration is referable to hysteria, but Triboulet finds that there is usually some reduction of sensation.

5. **The Reflexes.**—These are difficult to study, and are often said to be normal. They are normal in 50 per cent., and decreased or absent in 50 per cent., although Joffroy usually found the patellars decreased or absent, and Oddo found the tendon reflexes (*a*) rarely increased, (*b*) normal in light cases only, and (*c*) as a rule decreased or absent (absent on one side in 80 per cent., on both sides in 50 per cent.). The bladder and rectum are usually normal save in the severest forms.

6. **Electrical Reactions.**—The electrical reaction and mechanical irritability of the muscles are with few exceptions unaltered.

7. **The Heart.**—Irregularity is common, and is often due to irregularity in breathing (pseudo-arrhythmia). A slight increase in rate is due to the general neurosis. It has been claimed that chorea may affect the "muscles of organic life"; Ollivier, Roger and Simon have written of cardiac chorea, which probably does not exist. *Functional murmurs* are frequent, especially in thin nervous children, from the altered condition of the blood, the toxæmia, and increased cardiac rate. They may be confused with incipient endocarditis. Great conservatism must be exercised in their prognosis and diagnosis (see DIAGNOSIS OF MITRAL INSUFFICIENCY).

Endocarditis has been mentioned in the definition of the disease and its causation and pathology (90 to 94 per cent. of fatal cases). It was found in 66 per cent. of Osler's cases without rheumatism. It is rarely malignant, but may produce embolism. Mitral lesions, especially insufficiency, are the most common form. The importance of latent endocarditis, *i.e.*, without clinical symptoms, has been emphasized by Osler, Oxley and Grosse. Pericarditis is not frequent, though it is found in 20 or even 26 per cent. of cases, in certain statistics. Heart disease is found in 50 per cent. of cases in which there have been two attacks of chorea, and in 100 per cent. of those having over three.

8. **The Skin.**—Herpetic eruptions and pigmentation result from medication by arsenic. Urticaria, erythema nodosum and purpura are sometimes observed, as well as the rheumatic nodes first described by Maynet and later by Barlow and Warner.

9. General Symptoms.—Emaciation occurs chiefly in the severer types. The urea and phosphates are often increased. Albuminuria is rare, except as a result of renal embolism. The temperature is not often elevated, save from cardiac and other complications; even in chorea insaniens it rarely passes 102°.

Course and Prognosis.—The average course of mild cases is from six to ten weeks, and six weeks to six months is the average duration of all varieties. In very rare instances the disease may last six (Gowers) or even sixty years (Meldner). Three main types are distinguished: (*a*) The *mild type,* in which the patient is nervous, irritable, has but slight choreic movements and is often dyspeptic and sometimes anæmic; (*b*) the *severe form* in which the spasmodic movements are severe and general; (*c*) the *maniacal type* (*chorea insaniens*), which is most often observed in adult or pregnant women, and sometimes at puberty. The movements are incessant and extreme, and are often associated with fever, constant insomnia and great psychical disturbance. As a rule, the shorter the duration the more intense are the symptoms. The tendency to *recurrence* was noted by Sydenham. Relapses occur in 25 to 33 per cent. of cases. One year is the average interval, and when there is no recurrence within one and one-half years, it is probable that none will be observed (Gowers). Germain Sée held that relapses were most likely to occur at the sixty-ninth day. *Recovery* is the rule; the mortality averages between 2 and 3 per cent. Twenty to 25 per cent. of pregnant cases die. Fatality is greatest at the age of puberty and in adults. As Charcot remarked, death occurs less often from complications, as endocarditis, than from toxæmia, fever, mental changes, emaciation, delirium, and coma. In all cases rise of temperature or obstinate insomnia is ominous. Though the immediate outlook is not influenced by endocarditis, it is a most important feature in the ultimate prognosis of a complete recovery. *Intercurrent fevers* may dissipate the spasms. While complete recovery is generally expected, in rare instances distinct psychical changes (Jolly) or permanent chorea may remain (*v.s.*).

Diagnosis.—Recognition of the disease is usually most easy, after one has seen a typical case, and when one bears in mind the conception (definition, *q.v.*) of the disease.

Differentiation.—Simple tremor or the intention tremor of multiple sclerosis is easily separable. The hemichorea of hemiplegia (see DIAGNOSIS OF CEREBRAL HEMORRHAGE) or the bilateral chorea of *infantile cerebral paralysis* (*q.v.*) is easily distinguished by the history and course. Friedreich's ataxia is differentiated by its slow, irregular, incoördinate movements, nystagmus, its frequent family incidence, the scoliosis, talipes and slow, scanning speech. *Hysterical chorea* (so-called) is rhythmic, more regular and frequently produces sudden ("electrical") movements, as bowing or salaaming, nodding the head, etc. *Chorea major* is easily distinguished. Chorea in the original sense is chiefly of historical interest; it was a pandemic or epidemic *hysteria* provoked by religious excitement. Its victims made pilgrimages to the shrine of St. Vitus whence the name of the disease. Similar outbreaks occurred in Kentucky among the early settlers. This form closely resembles the saltatoric spasm described by Bamberger in 1859, in which the legs contract on attempting to stand, causing jumping or springing movements. It may be transitory, or may last for years, and occurs chiefly in neurotics. Beard and Thornton, quoted by Osler, have described a similar neurosis which was endemic in Canada among the "jumping Frenchmen." The "holy rollers," and jumpers of Russia and Java probably come under this head, or under impulsive tic (*v.i.*).

Huntingdon's chorea has no connection with Sydenham's type. Chronic chorea comprises two forms: (*a*) The *senile form*, in which the mind is less involved, the course more benign than in (*b*) the *hereditary form* of Huntingdon. Although it was described by Walters (1842) and Lyons (1863), Huntingdon in 1872 brought out the cardinal features of the disease as observed on Long Island. These are (i) the late and gradual onset after the thirtieth year, more marked in males than in females; (ii) its hereditary character, sometimes involving 25 or 50 per cent. of the family, sometimes skipping generations, or lasting for generations or even for two centuries in a family (Osborn); (iii) its progressive character, and (iv) the resulting mental degeneration, ending in slow dementia, often with epileptiform attacks or suicidal tendencies. The precise *pathology* of Huntingdon's type is not known. Pachymeningitis, hæmatoma of the dura, or less often chronic encephalomeningitis, vascular disease and atrophy of the convolutions have been present in the isolated autopsies made. The *etiology* is unknown. *Clinically* it is characterized by irregular movements, which usually begin in the hands, are somewhat controlled by voluntary effort, and are more irregular, incoördinate and slow than in acute chorea. Slow spasmodic facial contortions are observed, and the speech becomes first slow and hesitating, and later quite indistinct. The gait is unsteady and swaying. Writing is interfered with, the reflexes are moderately increased, sensation is normal save that the muscular sense is somewhat blunted, and dementia develops. The disease is incurable, although somewhat benefited by opium and bromides.

Tic must be differentiated. The term is now used to designate an "habitual, conscious, convulsive movement resulting in the involuntary contraction of one or more muscles, abruptly reproducing some reflex or automatic action of every-day life" (G. Guinon). There are two forms.

(*a*) In *simple tic*, (i) localized spasm, *habit spasm*, or habit chorea, which is said by S. Wier Mitchell to occur in girls between seven and fourteen years of age, may result from lesions of the nose or throat or possibly from eye-strain, and includes movements such as winking, drawing of the mouth, sniffing, shaking the head, and shrugging the shoulders. It usually ceases, but may last into adult life, and the "child's attention should not be drawn to the movements," although the writer believes that judicious reminders and discipline inhibit these habits. Arsenic may be beneficial. (ii) The *electric chorea* (Henoch and Bergeron) consists of short, lightning-like contractions in special muscles, as those of the neck or shoulders, as though the muscles were electrically stimulated. (iii) *Dubini's disease* (1846) consists of contractions of the arms and legs, followed by hyperæsthesia, pain in the head and back, muscular atrophy, paresis and sometimes fever and convulsions. It is endemic in Lombardy, its cause is unknown, and it is usually fatal from apoplexy or coma. (iv) *Paramyoclonus multiplex*, or *myoclonia*, which was described by Friedreich in 1881, is an affection of sudden onset, with symmetrical, involuntary, arrhythmic, quick, clonic contractions of the upper parts of the arms, legs and trunk, sometimes of the face and tongue, and occurs without loss of consciousness. The contractions number from 10 to 150 a minute, and may terminate in a tetanic contraction. It affects male adults chiefly (75 per cent.) and follows mental or emotional agitation, so that some would classify it under hysteria. The myotatic irritability and reflexes are increased, the mind is usually normal, although sometimes fixed ideas are observed, hiccough is occasional from involvement of the glottis or diaphragm, voluntary movement is decreased or sometimes arrested, there are no sensory symptoms and the outlook is usually good, although the course may be long and relapsing. Treatment consists of administration of voltaic electricity, nervines and bromides. The family form, described by Unverricht, with epilepsy and spasms of the tongue, and deglutition, is probably a subtype of hereditary chorea.

C. Oddo gives the following general differentiation between

TIC	and	CHOREA.
Form of movement: pseudo-intentional, with repetition.		Incoördination and great variability.
Rhythm: rhythmic, brusque, short, synchronistic.		Arrhythmic, slow, not synchronistic.
Will: suspends movements.		No action.
Accessory signs: muscular power preserved; normal tendon reflexes and sensation.		Myasthenia; modified reflexes and sensation.
Etiology: hereditary degeneration.		Neuropathic tendency.

(*b*) *Tic with explosive utterances*, impulsive tic or Gilles de la Tourette's disease, is closely affiliated with hysteria and monomania. It occurs in neurotic individuals between the sixth year and puberty. The *cardinal features* of the psychosis are: (i) Involuntary movements in the face and arms, or in all the muscles in severe types, when the movements become violent or explosive. (ii) Explosive utterances, resembling a bark, occur; the patient repeats words he hears (echolalia) or mimics movements (echopraxia); the child may use obscene or profane words (corprolalia), which was considered by de la Tourette to be most characteristic. (iii) The subject has fixed ideas. These include: arithmomania, in which every action is preceded by counting or doing a certain thing a given number of times; a fear of contamination (*délere du toucher*); onomatomania, or repetition of a given word or name; *folie pourquoi*, demanding the reason for everything. The prognosis is usually unfavorable.

(*c*) In *complex coördinate tic*, as thumb sucking, rocking in the bed, and bumping the head, the outlook is favorable (Gee and Hadden) when it does not occur in feeble-minded children.

Treatment of Acute Chorea.—1. Prophylaxis.—This may produce results; sensible methods of education should be adopted, the hours in the overheated, foul school-room should be short, and the useless forcing of children by marks, prizes and exhibition should be abandoned. Children can be taught more in an hour or two of judicious individual instruction than in the five or six confining hours which their wandering attention must endure. Prevention may concern other etiological factors (*q. v.*); for instance, the nose should be examined for sources of irritation, as adenoids.

2. Rest in Bed.—The results obtained by rest in bed and isolation are as rapid as those of any medicinal therapy. It not only saves tissue waste, but is the best prophylactic and symptomatic treatment of possible or actually established endocarditis.

3. Skin.—The skin is very often dry and harsh, whence it must be given special attention. Diaphoretics frequently produce beneficial effects. The stimulation of the skin by proper covering and by hydrotherapy is the simplest and least injurious method. The refrigerant diaphoretics, and full warm baths, give the best results. The skin must be kept clean, and in severe cases, the water-bed must be used.

4. Trauma.—Trauma is prevented in severe types by padding the beds by mattresses and pillows, since the violent movements may even throw the patient to the floor. Sepsis is a frequent occurrence, and is most dangerous. The temperature must be taken by rectum.

5. Exhibition of Drugs.—(*a*) *Fowler's solution* is the most frequently used and the oldest remedy, and apparently gives the best results. It should be given in increasing doses (see TREATMENT OF PERNICIOUS ANÆMIA, for detailed action); children tolerate large doses of arsenic exceptionally well. It may be administered hypodermatically or by clyster when the digestive tract is irritable. Arsenic is not without its dangers. C. Gamble, Jr., records an instance of fatal arsenical poisoning in a case to whom about 1½ ounces of Fowler's solution was given in a month (including an intermission of one week). It appears that rest in bed, and administration of a placebo, as nux vomica, has given equally good results, but any individual series of cases may be misleading. (*b*) *Salicylates* are of benefit only when rheumatism actually co-exists, and acetanilide is indicated only in the initial stages. In other words, the antirheumatic remedies are less important in the treatment than is rheumatism in the etiology. (*c*) *Depressomotors*: The *bromides* rarely cure the movements, although they often quiet the mild psychical manifestations. Chloral has been recommended and given in large and continued doses, to produce sleep or continuous sleep. This method merely demonstrates the tolerance of the body for dangerous remedies, and as stated before, chloral cannot be given safely, in amounts exceeding a dram for adults in three divided doses at intervals of an hour. Morphine is generally considered dangerous and inferior to chloral. Small doses often accentuate the mental agitation, but if combined with chloral, hyoscine and bromide, it may give at least temporary relief. (*d*) *Strychnine* is valuable (Trousseau), especially in the later stage, when recovery is slow. (*e*) In *chorea gravidarum*, abortion or premature delivery is indicated.

TETANY.

Definition.—Tetany, which was first described by Steinheim (1830) and Dance (1831) and named by Corvisart, consists chiefly of (*a*) peculiar tonic, intermittent, bilateral, painful spasms, mostly in the extremities, without involvement of the sensorium and (*b*) increased mechanical and electrical excitability of the nerves.

Etiology.—(*a*) It occurs *endemically* or epidemically, especially in Vienna, Heidelberg, Berlin and Syria, and it is prevalent during March and April. It is rare in America, England and Italy. Though once frequent (1830–1860), it is now uncommon in Paris. In the Vienna outbreaks 46 per cent. of cases occurred in shoemakers, and 24 per cent. in tailors who were otherwise healthy. Tetany occurs among the poorer classes. Eighty-three per cent. occurs in persons between sixteen and twenty-five years of age. It may develop in an entire family. (*b*) *Digestive affections* may produce it, as dilatation of the stomach (Kussmaul, 1871), dyspepsia, ulcer or cancer of the stomach or duodenum, often with pyloric obstruction; auto-intoxication is the most probable cause of this group. Only 80 cases of gastric tetany are on record. (*c*) *Rickets* is causal in 85 to 90 per cent. (Seeligmüller) and in 96 per cent. (Cassel) of the infantile types. Some writers—incorrectly, we think—consider that the infantile carpopedal spasm is tetany. (*d*) *Tetania strumipriva* is a type of tetany which follows removal of the thyroid in 15 per cent. of cases (Albert, Mikulicz, Kocher). It disappears when a new thyroid is implanted, and reappears on its removal. Animals deprived of the thyroid present symptoms practically identical with those of tetany. If one-fifth of the gland is left, it usually prevents tetany (von Eiselberg), but this fraction must be healthy. It may be true that the thyroid is less important than the parathyroid glands. (See pages, 895 and 896). (*e*) Tetany occurs also in the *acute infections*, sepsis, nephritis, puerperal or nursing women and rarely in intoxications with ergot, morphine or lead. There are recorded only 32 cases of the puerperal form. The only two cases which the author has seen in this country occurred with florid secondary syphilis. It may develop with other nervous disease, and French writers have attempted to class it under hysteria.. No pathological changes have been observed in the brain or nerves; in a few cases there has been cloudiness with swelling of the anterior horns. Fifteen cases are reported in America (Howard, 1906).

Symptoms.—1. The earliest and chief symptom is *the muscular contracture*, which usually commences in the hand, with stiffness, numbness, and pain, and culminates as a tonic, symmetrical, painful contracture. The muscles of the hand are hard; the attitude is that of the accoucheur's hand, that of the hand in writing, or that observed in paralysis agitans; it is produced by contraction of the ulnar flexors; less often it resembles the clenched hand of old hemiplegic contracture, and the nails even enter the palm; rarely the fingers are tonically spread apart. Except in light forms, the spasm cannot be overcome by force. The wrist may be flexed. The foot is affected less often than the hand, and the spasm draws the foot into the equino-varus position, with the toes flexed and the sole hollow. The spasm is usually limited to these parts, causes pain, lasts from minutes to hours or even days, disappears, and usually recurs later. In rarer and more severe cases, the parts above the elbows and knees may participate, with adduction of the arms and thighs, cramping of the face, neck, chest, tongue, diaphragm, and ocular muscles, with diplopia, spasticity of the sphincters with retention, dysuria, etc.

Laryngismus stridulus (Clark, 1815) is common in infantile rickets, in which clonic contractures also occur. The muscles are somewhat weak and tremor is frequent.

2. *Trousseau's sign* consists in producing the spasm by pressure over the nerves, for example, just above the elbow or over the peroneal nerve, for a few seconds or from one to five minutes. It is wholly due to nerve pressure, not to pressure on the vessels, and *is pathognomonic.*

3. There is *increased electrical irritability* (*a*) of the motor nerves to the galvanic current (*Erb's sign*) with early anodal closing and cathodal opening tetanus. Anodal opening tetanus is found in this disease alone. There is also increased electrical irritability (*b*) of the sensory nerves (Hoffmann) and (*c*) of the nerves of special sense (Chvostek, Jr.), to faradic and galvanic currents.

4. *Increased mechanical irritability* of the motor nerves (*Chvostek's sign*) is observed on tapping or pressing on the facial, median, ulnar or other nerves, which produces contraction of the muscles supplied by them. It is quite constant but may be absent in children and is not pathognomonic, having been found in normal persons, in cases of bulbar paralysis, in the major neuroses, and in convalescing cases of facial paralysis. (Some of these may have been atypical tetany). The muscles themselves are not overexcitable.

5. *Other symptoms* may be present. The sensorium is usually clear, except in rare cases with pyloric stenosis. The special senses are

normal; optic neuritis is extremely rare. Headache and malaise are frequent. In contrast to the pain and paræsthesia, objective sensory findings are absent. In rare cases epileptiform seizures are seen, which are probably due to thyroid changes. The reflexes are usually normal. The temperature is elevated in 10 to 15 per cent. of cases, but may be normal or subnormal. In very rare chronic cases, trophic, vasomotor and secretory disturbances may occur, as œdema, polyuria, glycosuria or falling out of the hair.

Diagnosis.—The diagnosis in typical cases is most easy from the cardinal signs: (*a*) the peculiar spasms; (*b*) Trousseau's, (*c*) Erb's and (*d*) Chvostek's signs. In epilepsy these signs are absent, and the aura, the cry, falling, tonic and clonic convulsions, biting of tongue and involuntary urination are characteristic; in very rare instances the two affections co-exist. Hysteria mimics everything, but the cardinal symptoms, except the spasms, cannot be reproduced; it has its own stigmata; certain reported epidemics of tetany, as at Gentilly, were clearly hysterical. In adults atypical forms without spasm are recognized by the other cardinal signs (*b*, *c*, *d*) which easily separate tetany from the occupation neuroses and acroparæsthesia (*v. i.*) In children meningitis and brain tumor are distinguished on careful examination. The rhachitic relations are important even though the extreme statement that all tetany in children is rhachitis, cannot be admitted.

Prognosis.—The outlook is good in most cases, and the course covers from two to four weeks as a rule. Recurrences are not infrequent. The etiological prognosis naturally varies, but generally speaking, death occurs not from tetany but from dilated stomach, or ablation of the thyroid. Cases may die from laryngospasm. In a recent review of his cases, Fränkl-Hochwart concluded that the prognosis is worse than is usually stated; 20 per cent. of his cases died, 20 remained well, and the balance had recurrences and serious disabilities.

Treatment.—The treatment varies with the cause. (*a*) *Rest and quiet* are indicated on general principles, although cases of tetany are influenced very little by excitement. Massage, strychnia and electricity should of course be avoided. Change of occupation and location, at least temporarily, are advisable in the endemic form. (*b*) In the *digestive form*, washing out the stomach and bowels in suspected cases of auto-intoxication may induce severe spasms. These cases are both dangerous and obstinate. The spontaneous mortality in gastric tetany is 70 to 80 per cent.; with operation for pyloric stenosis, these figures are reduced to 37.5 per cent. mortality. (*c*) In the *rhachitic form*, dietary regulation and improved hygiene are necessary, and phosphorus and iron are indicated. Inhalations of chloroform in some cases irritate the sensitive larynx. (*d*) In *tetania strumipriva*, thyroid feeding is usually of no value, as the mortality (80 per cent.) shows; successful thyroid implantation gives relief. (*e*) Cases following *acute infections* usually have a spontaneously short course; those occuring in pregnancy rarely indicate abortion; in nursing women, weaning gives prompt relief; for intoxications there are obvious indications.

In general, drugs are of little value; chloral is indicated *a priori*, because of its influence on the motor nerves, but it is inferior to the bromides, as is also morphine, except for pain. Sweating and digitalis in nocturnal forms of tetany have apparently helped some cases, but the disease usually runs its limited uninfluenced course.

OCCUPATION OR FATIGUE NEUROSES; WRITER'S CRAMP.

The continuous, hard, improper use of coördinate, functionally associated muscles may result in their cramping. *Writer's cramp* (graphospasmus, mogigraphia) was first described by Charles Bell (1830), and especially studied by Duchenne and Benedict. Neuropathic tendencies are noted in most cases; 83 per cent. occurs in males, 93 per cent. occurs in those between twenty and sixty years of age, and most of these cases develop between twenty-five and thirty-five. Worry is a frequent factor; cold, trauma, neuritis and neuralgia are possibly factors. The disease practically never occurs in those who write properly, *i. e.*, move the arm from the elbow or the shoulder, but develops in those who use the little finger or wrist as the basis of movement. The affection is located in various centres; some hold that it is in the coördinating cortical or subcortical centres; it is clearly not in the muscles or nerves, for their other movements are often normal; the occasional atrophy and paresis may suggest localization in the cord.

Symptoms.—The symptoms come on gradually, with fatigue, pain and cramping in the muscles of the fore-fingers and thumb, which may extend to the forearm or shoulder. There may be the "lock spasm" of Mitchell, in which the pen is spastically held or it may be thrown from the fingers. On rest, the tonic spasm disappears but reappears with attempts to write. In Benedict's classification we find the spasmodic, paralytic, tremulous and neuralgic forms, which are of less interest as types than as showing the various symptoms, which are often combined in a single case. Vasomotor symptoms may occur, as flushing, or glossy skin, and sometimes the faradic irritability is decreased and the galvanic increased.

Differentiation, according to the type, from hemiplegic weakness, paralysis agitans, musculospiral paralysis, and neuralgia, is usually easy. *Similar cramps* are observed in telegraphists, milkmaids, weavers, cigarette rollers, tailors, shoemakers, watchmakers, and violin and piano players. Public speakers may suffer from similar neuroses of the throat, and Archibald Church also mentions spasms of the jaw in gum chewers and of the lumbar muscles in physicians who drive much.

The *outlook* is poor, because the unfortunate sufferer is often compelled to write until the affection is firmly fixed, and when able, usually returns to the same work. In some cases the muscles may waste.

Treatment.—Rest is essential to recovery, because treatment for the usual nervous symptoms alone never succeeds. Plaster casts are sometimes successfully employed. Even when the subject learns to write with the other hand, it often develops there. Prevention is possible by writing free-hand from the shoulder; holding the pen between the first and second fingers, use of pens with large handles, of quills and of forearm splints with the pen attached to the extremity, are also helpful. Vigouroux and Wolff use with success systematic gymnastics, including passive manipulations and active exercises.

MYOTONIA. THOMSEN'S DISEASE.

Myotonia is usually called myotonia congenita from its leading causal element, heredity. It was first described by Leyden, but more fully by Thomsen (1876), who was a victim among 20 cases in his own family, in five generations. This rare affection, of which there are about 100 reported cases, occurs chiefly in males and in Germany and Scandanavia. Its pathology is obscure, for the only necropsy which has been performed (Déjérine and Sottas) showed no nervous alteration. Muscle fibers, excised during life, show hypertrophy, decreased striation, nuclear multiplication and slight interstitial increase, leading Leyden and others after him to consider the process myopathic. Some offer the objection that these changes are artefacts.

Symptoms.—A peculiar *transient tonic muscular rigidity* develops when the muscles are first used, which limits or arrests movement, but wears off as their use is persisted in. It recurs, or indeed is most intense after rest or when a new kind of movement is undertaken. It appears early in life, perhaps "in the cradle," but generally between the ages of four and ten, it increases as the muscles develop and then becomes stationary for life. In one case improvement was observed. The legs are more affected than the arms, but any muscle may, in exceptional cases, be affected, as those of the face, tongue, or bladder or the respiratory muscles. They are usually stronger and sometimes larger than normal, although some writers speak of muscular weakness. The longer the muscles are rested, the more severe is the initial spasm on exertion. Rigidity does not follow passive muscle movement, although firm pressure or a sharp blow initiates it.

Though the tendon reflexes and the mechanical and electrical excitability of the nerves are practically normal, application of the galvanic current to the muscles produces sluggish, prolonged closing contractures, which are easily and equally obtained with either pole, and become tetanic with strong currents; this is the *myotonic reaction of Erb.* Undulatory contractures follow strong faradic stimulation. Subjects of myotonia are frequently irritable or hypochondriacal.

Treatment is of little value. Thomsen believed that exercise retarded the disease. Cold, dampness, excitement and fatigue, are to be avoided.

The following are allied, if not identical, affections: the congenital *paramyotonia* of Eulenberg, consisting of tonic spasms, which last from fifteen minutes to several hours, occur especially in the arms, and often affect the eyes and mouth; it is excited by exposure to cold (vasomotor spasm in the muscles); and the *ataxic paramyotonia* of Gowers, in which tonic spasms may last the entire day, with muscular weakness, ataxia especially in the hands, and disturbed sensation or anæsthesia in the fingers.

PARALYSIS AGITANS.

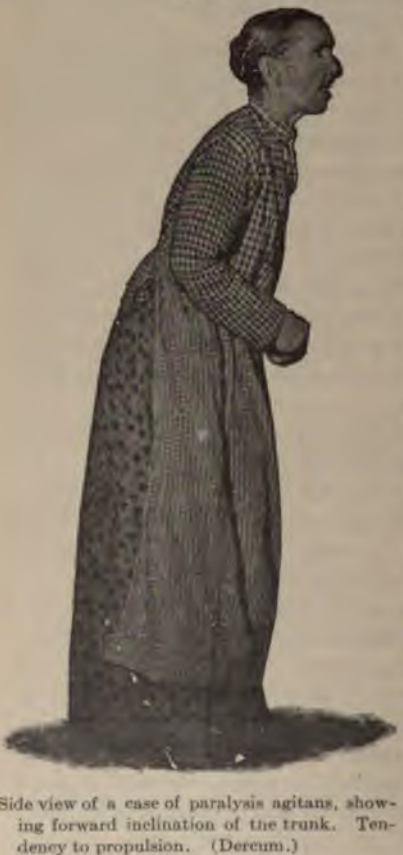
This affection is also known as shaking palsy or Parkinson's disease; it was first described by Parkinson in 1817.

Etiology.—(*a*) Most cases occur in persons between the ages of *forty and sixty-five.* (*b*) Sixty-six per cent. of cases are *men.* (*c*) A *neuropathic tendency* is present in 15 per cent. (*d*) It is doubtful if trauma, acute infections or thyroid disease are causes.

Pathology.—It is classed among the neuroses, because of the absence of any constant pathological finding. It is not strange that arteriosclerosis and perivascular thickening are often found, since paralysis agitans occurs in "the early degenerative period." Gordinier's collection of cases examined by recent histological methods, shows that (*a*) general arteriosclerosis is less marked than that of the nervous tissue; (*b*) the arteries in the cord are markedly changed, and around them are (*c*) increased neurogliar sclerosis and (*d*) pigmentation and atrophy of the nerve cells and fibers.

Symptoms.—The cardinal symptoms are (*a*) tremor; and (*b*) stiffness, slowness of movement, shortening and weakening of the muscles. (*a*) *Tremor* in 66 per cent. of cases is the first symptom; it may develop after the muscular rigidity, and is sometimes absent (paralysis agitans sine agitatione). It usually appears first, and is most marked in the *hands.* It affects first one hand, and usually next reaches the leg of the same side, then the opposite hand, the opposite leg, and lastly the trunk, so that at different periods of development it is successively a monoplegic, hemiplegic, or finally a diplegic tremor. It is slow (4 to 7 oscillations per second), rhythmic, fine at the onset, but somewhat coarser later and in the large muscles is more marked in peripheral than distal parts, *persists during rest, ceases during sleep,* and ceases momentarily on voluntary action. In rare cases, it appears only on movement, as intentional tremor; Gerhardt found it increased by movement in 50 per cent. It is difficult to define precisely the muscles involved. In the hands it may affect chiefly the flexors and extensors at the metacarpal joints or the interossei, which give the *spinning or pill-rolling movement.* The wrists may be abducted and adducted, rarely supinated and pronated, and the arms and shoulders are implicated but little. The writing is tremulous. The feet are mostly involved at the ankles, and in the toes but little. The thigh adductors often tremble. Trembling is infrequent in the face, neck and back, while the abdomen entirely escapes. The head and body may of themselves tremble, but much of it is imparted to them from the limbs. In the rarest cases, the tongue, palate, larynx and epiglottis tremble.

FIG. 80.



Side view of a case of paralysis agitans, showing forward inclination of the trunk. Tendency to propulsion. (Dercum.)

(*b*) *Muscular rigidity* and *shortening*, weakness, and *slowness of movement* usually follow the tremor; slight symptoms are detected early, and anticipate the tremor or exist without it. These changes result in the following: (i) The *Parkinson facies*, which is *mask-like* and devoid of lines or expression; the eyelids seldom wink. The forehead shows transverse folds because of the bowed head and body. The eyes move tardily in reading, and the entire body must be turned to enable the patient to look laterally. There is also slow, monotonous, *monosyllabic speech*, with hesitation at first, but hurried at the finish. (ii) The *attitude* is characteristic, because of the flexor positions assumed, the head and the chin are held forward, the neck is extended and rigid, the body is inclined forward, the elbows are fixed and brought somewhat forward and away from the body; the forearms are brought toward the body, the trembling thumb and index finger are apposed, or the fingers deviate toward the ulnar side as in arthritis deformans, and in standing the knees are somewhat bent and at times closely apposed. The body expression, we may say, is like that of the face. (iii) The *gait* is equally characteristic. Like the speech, it is initially hesitant, and later hurried. The patient, when once started, may not be able to stop himself. Trousseau spoke of the patients as "chasing their centre of gravity." This *festination* or *propulsion* is probably purely mechanical from the forward bodily inclination, although it was interpreted by Charcot as a kind of forced movement. To counteract the throwing forward of the centre of gravity, the arms may be carried behind the back. In some cases, if the patient is pushed backward or sideways, he may not be able to stop for some steps; this is *retro- and latero-pulsion*. (iv) The *palsy* is often slight and rarely extreme, although it may be simulated by the helplessness caused by rigidity. The patient is not inclined to

xertion, "moves like a piece of machinery," as Parkinson describes
, and may not be able to turn over in bed. Patients have drowned
om falling on the face in a small puddle of water. Muscular reaction
quires 40 per cent. more time than normal.

Other symptoms are subordinate. The mind is clear, but we may
y *stiff* like the face and limbs, and a certain restlessness, irritability
apathy may be detected; insomnia is common and headache
casional. Sensation is essentially normal, although pains, pruritus,
aræsthesia and hypæsthesia are seen in 35 per cent. of cases. Vaso-
otor disturbance most often assumes the form of a feeling of heat;
deed the peripheral temperature may be increased several degrees.
evere sweats are quite common. The pupillary, cutaneous, tendon
nd organic reflexes are normal.

Course and Prognosis.—The onset is sometimes sudden, but usually
radual, and the course covers several decades. The outlook is more
vorable in atypical than in typical cases. Remissions may occur,
ut the disease progresses steadily. Death very rarely occurs from
e affection itself, but usually from intercurrent pneumonia or ad-
ancing arteriosclerosis, cerebral softening, myocardial insufficiency,
ephritis and kindred lesions.

Diagnosis.—When there is clear development of the cardinal symp-
oms, the disease is unmistakable. Confusion is possible in the early
emiplegic forms, but the absence of a paralytic stroke, and the later
ourse are distinctive. Charcot and Ordenstein (1868) clearly differen-
ated the affection from *multiple sclerosis* (*q. v.*). The fine, *senile*
emor, the *hereditary tremor* of Dana, tremors due to overexertion or
citement, to toxic causes (alcohol, nicotine, lead, mercury, coffee,
ium, or chloral), weakness of convalescence from acute infections,
retic dementia, or the neuroses (epilepsy, Graves' disease, hysteria,
urasthenia) are easily excluded by the absence of the Parkinson
gidity, facies and festination.

Treatment.—Treatment of the probably fundamental arterioscler-
is may possibly retard the disease, but prophylaxis is possible in the
rly stages only. All mental and physical fatigue should be avoided.
he administration of full doses of iodide, chloral, opiates, bromides,
yoscine, ergotin, conium or duboisin may mitigate the tremor, but
ways lower vitality and disturb function.

PERIODIC FAMILY PARALYSIS.

This affection was first described by Cavaré (1853), and more fully
y Westphal (1885); Audibert and Oddo collected 64 cases (1902),
hich Holzapple brought to 70 (1905). It is a family affection; Goldflam
llected 11 cases in one family, and E. W. Taylor 12 in another.
ost cases occur in adolescence, and nearly all of them in persons
nder twenty-five; it recurs every few weeks or months, possibly every
ay, or only at intervals of years. Its *symptoms* are almost wholly

motor, producing a *flaccid paralysis* in the legs and arms, less often in the trunk, and exceptionally in the face, neck, tongue and throat. It occurs without apparent cause, usually during sleep, from which the patient awakes with a sense of fatigue, weakness, or dulness which develops into paralysis within twenty-four hours. Mental and sensory symptoms and those of the cranial nerves and special senses are rare. There is no fever, the pulse is often slow and cardiac dilatation has been observed. The deep reflexes are decreased or abolished, often also those of the skin, and the faradic excitability of the nerves and muscles is lessened or absent. The patient is well in a few hours or days, until another attack occurs. The disease usually subsides spontaneously after the fiftieth year of life. Its *nature* is not determined. Goldflam's theory of auto-intoxication is supported (*a*) by J. K. Mitchell, Flexner and Ebersoll, who found increased secretion of kreatin before and during the attack; (*b*) by Crafts and Irwin, who isolated toxins from the fæces; (*c*) possibly by the fact that potas. citrate sometimes lessens or aborts the seizure. Dana thought that the disease was hysterical, but Bernhardt and others consider it affiliated with myotonia and progressive muscular atrophy. Holtzapple administers potas. bromide in doses of thirty grains.

MIGRAINE.

Migraine (hemicrania, megrim, sick headache) is an hereditary degeneration or psychoneurosis, with the paroxysmal occurrence (in a complete attack) of auræ, headache, nausea and vomiting.

Etiology.—(*a*) *Heredity* is a conspicuous cause (90 per cent. Möbius), either directly as migraine, or indirectly from neurotic, insane or epileptic antecedents. Joint affections, as gout, are often noted in the family history. (*b*) Thirty-three per cent. of cases begin between the fifth and tenth, 40 per cent. between the tenth and twentieth years, and the remainder usually before the thirtieth year. Females are affected a little more frequently than males. (*c*) *Exciting causes* are acute infections, constipation, dyspepsia, overexertion, alcohol or sexual abuse, emotional factors, pelvic disorders, and lactation. It is attributed, though probably incorrectly, to adenoids. Refractive errors may explain some cases, but as Möbius states, "this cause is epidemic in the United States."

Symptoms.—Preliminary symptoms are often absent, although the patient may feel depressed, or, in some cases, especially well and energetic. They occur in cases in which the sensory symptoms (see below) are least marked. Sometimes the pupils are dilated. Most attacks develop during the night.

AURÆ.—In over 50 per cent. of cases, auræ are (*a*) sensory, as visual auræ, a cloudiness before the eyes, sparks, lightning, zig-zag forms, etc.; they are most often bilateral, and affect the homologous half-fields; taste and smell may be similarly affected; paræsthesia may occur, usually as hemiparæsthesia; (*b*) motor auræ are much less common, as aphasia, dysphasia or motor weakness on the contralateral side; in the very rare form, ophthalmoplegic migraine, there is fugitive ptosis, outward strabismus, double vision and a dilated immobile pupil; mental auræ are uncommon and not usually pronounced, as restlessness, anxiety, confusion, and double consciousness. The auræ last for ten or fifteen minutes.

THE ATTACK.—(*a*) *Headache* is the cardinal symptom. As first it is usually intense and circumscribed, as over the eye, temple, occiput or forehead; then it spreads over one side (*hemicrania*), sometimes affects the opposite side, or rarely reaches the neck or arm. The pain is deep, varies in character, being most often boring, and its intensity may attain that of the pain in brain tumor or meningitis. It is increased by movement, stooping or lying down. The special senses are hyperæsthetic but the nerve trunks are seldom tender. The mind is usually clear and often acutely active, sometimes there is double consciousness, and in rare cases stupor. (*b*) *Nausea* follows, often with vomiting of undigested food and finally of bile; this is the so-called "bilious headache." Feeding and medication are quite impossible in severe cases. Vomiting may be attended by hyperacidity or moderate collapse, as in sea-sickness. (*c*) *Vasomotor symptoms* may be present. The *spastic type* was described by Du Bois Raymond in his own case; with pallor, coldness, wide pupils, and retraction of the eyeballs; Thoma found the temporal artery sclerotic on the side of lesion, and de Giovanni noted a marked arcus senilis on the same side. The pulse may be slow and tense and the breathing superficial. In the *paretic type* observed by Möllendorf in himself, there are redness, small pupils, sweating, and increased flow of saliva or tears. Very rarely is there erythromelalgia, œdema or ecchymosis. The paretic may follow the spastic symptoms. It is best to regard these sympathetic symptoms rather as a result than as a cause of the attack.

The *attack ends* in various ways. Relief may come with the vomiting, after which the patient frequently sleeps. Sometimes the ending is almost critical, with sweating, lachrymation, polyuria or epistaxis. The attack lasts from ten to twelve hours; the term *status hemicranicus* is used for cases of long duration, or those in which the attacks overlap. Though the typical seizure comprizes the aura, lasting fifteen minutes, and the headache, lasting ten or twelve hours with nausea and vomiting, *most attacks are atypical*. There is most often headache alone, or headache with vomiting; in a few cases the attack aborts with the aura, or vomiting exists alone. The auræ, pain, gastric and cardiac inhibition, vasomotor symptoms and vomiting can only be explained on the assumption that migraine is a *functional cerebral affection*.

Course and Prognosis.—When the disease has once been established in youth, its attacks become more frequent in adolescence and usually last until the forty-fifth or fiftieth year. The periodicity, which was noted in early cases described by Tissot, Trousseau and Liveing, has not been explained. Overwork, worry, menstruation, or change of routine may precipitate an attack at a regular or irregular interval. Attacks may cease during an intercurrent pregnancy, tabes or gout. The school of Charcot has emphasized the fortunately rare danger of transformation into epilepsy, labyrinthine vertigo, or some psychosis. Complete cases are more obstinate and dangerous than the ordinary light form.

Diagnosis.—When there is a history of heredity and development in childhood, the full attack is most characteristic. "Sick headache" is practically always migraine. In atypical forms there may be diagnostic confusion, chiefly with various forms of *headache*, which may be classified as follows:

1. Those which result *from nervous affections*, as (*a*) cerebral neurasthenia, in which there is a sense of pressure rather than actual headache, and which is often constant, without nausea, but with paræsthesia, and in location is frontal or usually general, but sometimes occipital or temporal; (*b*) hysteria, which is less common, (clavus headache, "neuralgic headache"), and sometimes is precisely like migraine (Charcot); (*c*) epilepsy, which some consider as identical with migraine; in epilepsy the aura is shorter, and loss of consciousness is the most constant symptom; (*d*) meningitis, which in all types and in all cases is early and severe; (*e*) hydrocephalus, which is often severe, especially in acquired forms; (*f*) brain tumor, which is constant, generalized, often very severe, and sometimes typically hemicranic; (*g*) paretic dementia, in which migraine is frequent; care is always necessary when migraine occurs after syphilitic infection.

2. Those which result from affections of the *special senses*, as eye strain, hypermetropia; glaucoma, which most often produces frontal headache; nasal disease, which may cause involvement of the sinus with frontal headache; this form is not periodic and is often relieved by iodide, which has no effect in migraine.

3. Those which result *from toxæmia and autotoxæmia*. (*a*) Those due to acute infections are usually frontal and dull, but sometimes occur in other locations and are severe, deep-seated, and increased by stooping; in typhoid or malaria they are often frontal or supra-orbital; (*b*) intoxication by alcohol, nicotine, lead, nitroglycerine, and opium, and (*c*) constitutional states, as gout, or arthritis. (*d*) In nephritis, they are usually frontal, remittent or continuous, but sometimes sharply hemicranic. (*e*) In gastric affections from toxæmia or congestion, they are exceedingly common, mostly occipital, but sometimes vertical or frontal.

4. Those resulting *from circulatory causes*, as from wearing a tight collar, repeated coughing, cardiac disease or arteriosclerosis (with vertigo), are usually increased by lying down.

5. Those which result *from anæmia*, especially from chlorosis as a "tugging" sensation back of the eyes, or frontal or generalized headache, are often relieved by lying down.

Treatment.—*Prophylaxis* is said to be of little value, but many subjects learn to recognize the warnings and avert the attacks, by r noting unusual mental or physical capacity for work, or an appetite, which usher in some attacks. Migraine subjects bserve *moderation* as do other neurotics; moderation in eating, y of meat, in work, in exercise or recreation, in use of narcotics lants as coffee or tobacco, and in sexual matters. Immoderaeed, is almost a neurosis in itself. Overwork, hurry, worry, ion must be avoided, and their first manifestations must be nd treated. The extract of *cannabis indica* gr. ¼ to ½, given s or months, after Seguin's plan, is almost the only remedy lps the condition, and then only in some cases; the author a dozen cases in which migraine has been benefited or wholly under its use. Möbius finds that *salicylate of soda*, gr. xv, coffee, given the evening before the attack, often wards it off. cerine in full doses (Gowers) sometimes aborts it, as may out the stomach and following the lavage with a saline (Her-

treatment of the established attack, bromides constitute the best they are given not only during the attack in the mild type, nuously, as in epilepsy, when the type is severe and frequently (Liveing and Charcot). Acetanilide and the kindred coal-tars p the mild forms and may in combination with ergotin, lessen bing pain, but soon lose their effect. Morphine should be avoided. The stomach sometimes rejects all medication ust then be given by rectum. Application of cold to the head, baths and sinapisms to the nape of the neck and epigastrium rdinate helps.

NEURALGIA.

n the nerves is only a symptom, usually of an affection of a al sensory nerve, although in rare cases centric disease may ripheral pain, as affections of the cortex, pons, medulla, or r the thalamus. The pain of neuralgia, in which there is little ganic change in the nerve, and the pains of neuritis may be

gy.—(*a*) Neuralgia occurs mostly in *adult life* (twenty to sixty), ly before puberty or after sixty, in which case it is usually d intractable. (*b*) The majority of cases occur in *women*. (*c*) is an apparent factor in 25 per cent. of cases, especially in hic, weakly or gouty families. (*d*) *Debility* from exhausting s, as lactation, anæmia, overwork, sensory fatigue, as from n, sexual or other excesses, emotional conditions and trauma, se it. (*e*) It may result from *toxæmia;* (*a*) acute infections, *influenza*, typhoid, especially at its onset, *malaria*, the causal ce of which has been much overestimated, rheumatism (from to cold) particularly of the fascial type, and sometimes may cause it. (*b*) Alcoholism, *diabetes*, plumbism, gout, and may produce it; most of these are also causes of neuritis, differentiation from which may be difficult. (*c*) *Nerve affections* as sciatica and neuritis, herpes zoster, reflex irritation, as radiated from a carious tooth, compression of a nerve trunk by varices, aneurysm or neuromata or the root pains of tabes, may cause neuralgia. (*d*) Arteriosclerosis may affect the nutrient nerve vessels; this was first asserted by Putnam and Dana, and lately by Thoma and Dehio.

General Symptoms.—The *pain* usually presents the following characteristics: (*a*) It is always *paroxysmal*, with intervals of total cessation, or at least of relative relief; intense pain is never constant. (*b*) It *recurs* either irregularly at intervals varying from hours to months, or with surprising periodicity on a given day or hour, perhaps at the menstrual period; periodicity is as frequent in non-malarial as in malarial cases. (*c*) It is usually *unilateral*, is generally constant in its seat, at least for a time, and is most often found over the distribution of a single nerve, or several nerves, from which it sometimes radiates into other regions; if pain is bilateral, it is usually symmetrical. The pain may shift from place to place, especially when due to systemic causes. It is probable that the spinal cord may be the medium of radiation of pain from one region to another and that pain may follow the distribution of the spinal segments rather than that of the peripheral nerves as in herpes zoster. However, the exact pathology of neuralgia is still uncertain. (*d*) Its *onset* and *cessation* are *sudden and spontaneous;* an imminent attack may be precipitated or existing pain may be aggravated by muscular movements, a breath of air or a simple touch; the character of the pain is *sharp*, shooting, *stabbing*, rarely pulsating; if it travels along the nerve trunk its shooting direction is usually centrifugal, rarely centripetal; if it is localized it is commonly burning or boring. (*e*) The pain is *usually deep*, but the skin is very hyperæsthetic. (*f*) The "*tender points*" of Valleix (1811), later called maximal points by Head, are found in the nerve (50 per cent.) both in the attack and the free interval, especially where the nerve emerges from the bone or fasciæ, lies on hard surfaces, divides or anastomoses. The diagnostic value of occasional tenderness over the spine at the origin of a nerve (Trousseau's *point apophysaire*) is overestimated, because it occurs in many other conditions. In neuritis, the nerve trunk along its entire accessible course is tender. (*g*) *Vasomotor constriction*, followed by dilatation, is fairly common. Œdema, erythema, blanching or falling out of the hair, secretory disturbances, herpes, pigmentation, numbness or tingling, trophic alteration of the skin, localized hypertrophies and reflex twitchings of the muscles are much less frequent than they are in neuritis. Persistent pain or its etiological factors may decidedly reduce the general nutrition.

Local Clinical Varieties.—1. TRIFACIAL NEURALGIA (prosopalgia, *tic douloureux*) is the most important, severe and frequent type, because the fifth nerve is most exposed to trauma, compression and infection, by way of the eye, nose, and ear. The entire nerve is not usually affected, though pain may radiate from one division into another; ndeed only the secondary branches of a single division may be involved. The general features of neuralgia are present, as already described. (*a*) Involvement of the *first branch* is the most common form; pain occurs over the eye, forehead, and frontal sinus (in which the familiar pain is experienced after eating ice-cream); it sometimes radiates into the second and third branches; there are "tender points" over the lower edge of the nasal bone, the supra-orbital notch, less often over the supra-orbital nerve, even to the vertex, sometimes over the occiput or over the cervical spines; cold, carious teeth, disease of the sinus, glaucoma, and errors of refraction (hypermetropia) are causal factors. (*b*) When the *second branch* is involved, there is pain over the side of the nose and cheek from the infra-orbital region to the upper lip; there are "tender points" over the lower border of the nasal bone, the malar bone, the infra-orbital foramen, the gums over the canine teeth, and sometimes in the hard palate; among other causes due to sclerosis is the dental alveolitis in the aged. Diseased teeth may cause neuralgia without local dental symptoms; therefore in every severe case the dentist should be consulted. (*c*) Involvement of the *third branch* is the least frequent form; inframaxillary pain, along the lower teeth, tongue, chin, external ear and maxillary region; and tenderness over the auriculotemporal nerve and inferior dental foramen may occur. In old persons the loss of the teeth brings the jaws closer and therefore stretches the inf. dental nerve; it is relieved by employing proper plates. In severe forms vasomotor symptoms and increased secretion from the eyes, nose and salivary glands are usually present. Hyperalgesia is usual so that the face cannot endure simple contact, and drinking, mastication and wiping the nose cause great suffering. Herpes and trophic changes are probably neuritic rather than neuralgic symptoms. In rare instances facial spasm, transient reflex ptosis, internal strabismus or myosis co-exist. *Differentiation.* In headache and migraine there are no tender points; intracranial disease, as tumor or syphilis, is usually excluded by systematic examination. The *prognosis* is doubtful in severe forms, which are often lifelong in duration and agonizing in degree.

2. In CERVICO-OCCIPITAL NEURALGIA; there is pain in the first four cervical nerves, especially the inferior occipital and auricularis magnus; this pain sometimes radiates into the fifth nerve; there is tenderness half way between the mastoid and spine, over the parietal eminence and over the cervical plexus between the sternomastoid muscle and trapezius; this form is rarely primary, is often bilateral, and is less intermittent than other neuralgias, which facts are explained by its frequent connection, especially with cervical caries and syphilis, and also with tumors, meningitis, root disease and affections of the ear.

3. PHRENIC NEURALGIA is rare, though it sometimes occurs in pericarditis and diaphragmatic pleurisy; it is most often left-sided, with pain and tenderness over the insertion of the diaphragm and nerve trunk in the neck; movements involving the diaphragm are painful. Diagnosis and differentiation are usually uncertain.

4. In CERVICOBRACHIAL NEURALGIA, any nerve may be involved, but the ulnar and circumflex are especially affected. Pain may radiate over the entire plexus and into the chest; the tender points are most often in the axilla, over the shoulder and over the ulnar distribution; it is most often confused with neuritis from trauma, compression or other organic disease; it may be due to rheumatic arthritis.

5. INTERCOSTAL NEURALGIA ranks third in frequency after trifacial and sciatic neuralgia (neuritis). Pain is felt along the intercostal spaces and tenderness over the *points* of emergence of *its three branches*, over the angle of the ribs, in midaxilla and near the mammary line. It is more easily distinguished in the upper than lower interspaces. Differentiation may be difficult because of muscular rheumatism in which movement is more painful. Peritoneal disease, gastric ulcer, spinal caries, aortic aneurysm, and tabes must be distinguished. It is most common in gastrohepatic affections, and in neurotic women.

HERPES ZOSTER is probably an acute specific inflammation of the posterior root ganglia, as shown by Bärensprung and Charcot and later by Head and Campbell. It is mentioned in this connection because of the acute pain preceding and associated with the rash, which appears after three or four days; the pain is often protracted and intractable; the pain on the trunk follows the distribution of the spinal segments rather than that of the intercostals. The ganglion is the seat of a cellulohemorrhagic exudation and its fibers to the posterior column of the cord degenerate.

6. LUMBAR NEURALGIA is infrequent; it occurs in the iliohypogastric, ilio-inguinal and genitocrural branches, sometimes in the ext. cutaneous, obturator and crural nerves. The pain is felt over the iliac crest, in the inguinal canal, labium or testes (irritable testicle).

7. SACRAL NEURALGIA is also infrequent. Coccygodynia is probably neuralgic, and occurs mostly in women. The pain is increased by sitting, coitus, and defecation; it is obstinate and resists even operative measures.

8. In neuralgia *of the feet*, there is pain in the heel, which was called PODODYNIA by Gross; plantar neuralgia, which is mostly neuritic, occurs in acute infections, and in caisson disease. Morton's metatarsalgia, which was regarded by him as due to bony compression of the fourth metatarsophalangeal nerve, usually occurs in one foot, and mostly in women; operation is usually indicated.

9. VISCERAL NEURALGIAS are considered under diseases of the heart, stomach and kidneys and under hysteria and neurasthenia. Under this caption may be given *Head's areas of pain* referred to visceral disease: the organ is first given and following it, is the area to which its pain is reflected: *Heart*: 1st, 2nd, 3rd dorsal segments. *Lungs:* 2nd, 3rd, 4th, 5th dorsal segments. *Stomach*: 6th, 7th, 8th, dorsal; cardiac end from 6th and 7th. Pyloric end from 9th. *Int tines:* (*a*) Down to upper part of rectum, 9th, 10th, 11th and 12 dorsal. (*b*) Rectum, 2nd, 3rd and 4th sacral. *Liver and gall-bladde* 7th, 8th, 9th, 10th dorsal; perhaps 6th dorsal. *Kidney and urete*

0th, 11th and 12th. The nearer the lesion lies to the kidney, the nore is the pain and tenderness associated with the 10th dorsal. The ower the lesion in the ureter, the more does the 1st lumbar tend to ppear. *Bladder*: (*a*) Mucous membrane and neck of bladder, 1st, 2nd, 3rd, 4th sacral. (*b*) Overdistention and ineffectual contraction, 11th and 12th dorsal and 1st lumbar. *Prostate*: 10th, 11th, 12th dorsal, 1st, 2nd, 3rd sacral and 5th lumbar. *Epididymis*: 11th and 12th dorsal and 1st lumbar. *Testis* and *ovary*: 10th dorsal. *Appendages*: 11th and 12th dorsal and 1st lumbar. *Uterus*: (*a*) In contraction. 10th, 11th, 12th dorsal and 1st lumbar. (*b*) Os uteri. 1st, 2nd, 3rd and 4th sacral (5th lumbar very rarely).

Treatment of Neuralgia.—Logically the treatment of the cause (*q. v.*) should precede that of the symptoms, but practically, pain is the prime indication for therapeutic measures.

1. Local Measures.—Pressure on tender points may diminish or bolish pain. As in sciatica, blisters and sinapisms (both of which nay leave permanent pigmentation on the face), the cautery, local eat, acupuncture or injections of water or cocaine may give relief. Freezing the skin by ethyl chloride (Richardson, Deddard) often gives better results. Menthol, camphor-chloral (equal parts) and iniments are of some value (tr. aconiti, tr. chloroformi, tr. belladonnæ āā ʒij, linimentum saponis ℥iv). Injections of 1 per cent. solution f osmic acid into the nerve trunk are very successful; they were ecommended by Bennett (1889) and later by Murphy; paralysis ometimes results. Massage is often beneficial, for the muscles and erves have common veins.

2. Nutrition.—A full diet, with milk, and cod-liver oil, and also with rest, the Mitchell cure, hydrotherapy and massage, is often eneficial, especially with change of climate or surroundings. Alcohol, which is always dangerous, is most often indicated in older subjects. The beneficial effects of castor oil are often great, probably because it promotes elimination. Gouty subjects may thrive on a vegetable diet.

3. General Drug Treatment.—There are numerous remedies. Iron and strychnia, and possibly phosphorus are valuable in anæmic subjects. Dana gives strychnine hypodermatically in doses of $\frac{1}{30}$ gr. even up to $\frac{1}{8}$ gr.). Arsenic is excellent in anæmic, malarial and degenerative types; quinine is beneficial in non-malarial as well as malarial types and in trifacial neuralgia especially, although its value is considerably overestimated. Valerian, cannabis indica and bromides should be given for irritability; salicylates for rheumatism and olchicum and cimicifuga for gout; iodides are sometimes of benefit o non-syphilitic subjects; full doses of nitroglycerine are frequently elpful.

4. Analgesics.—Acetanilide (gr. v) and the other coal-tars should e used first in fresh cases. Extr. cannabis indicæ (gr. $\frac{1}{4}$) and fluidextr. gelsemii (♏ ii to iv) should be administered every three to six ours in the less severe cases. In acute febrile or rheumatic cases, fluidextr. aconiti (♏ i to ii) should be given every two hours until tingling in the mouth or extremities and slowing in the pulse intervenes. Liebreich advocates croton-chloral (gr. v), or with care even (gr. xx) for several doses. Finally, in severe cases, morphine may be given but as far as possible, it should be avoided; sometimes its administration is inevitable; though some writers find that it tends to prevent recurrence, the pain often develops again, intensified; it should be combined with atropine, which may be given alone, as it sometimes more permanently controls the pain than does the morphine.

5. **Electrotherapy.**—This sometimes relieves the pain, but it more often fails. Galvanization with a weak current, which should be just strong enough to produce a sensation of burning, and should be applied with the sponges warm and with the anode to the painful area, may be beneficial, especially in newly developed cases.

6. **Surgical Therapy.**—This includes section, resection, stretching, twisting or divulsion of the nerves; resection should be tried first, but all of these proceedings may fail. In incoërcible trigeminal neuralgia, the Gasserian ganglion may be extirpated (Hartley, Krause, Keen, Cushing), but this operation is severe, and loss of the eye may result. Horsley opens the skull and divides the root of the fifth nerve above the ganglion, but this also is distinctly dangerous, and Spiller and Frazier therefore suggest its division without opening the cranial cavity.

VASOMOTOR AND TROPHIC NEUROSES.

Vasomotor centres exist in the cerebral cortex and medulla. The fibers probably run in the lateral columns of the cord and leave by the anterior roots. *Vasomotor paralysis* occurs independently or with functional or organic disease. Paralysis of the cervical sympathetic may result from trauma, aneurysm, tuberculous apical or glandular disease, or disease of the cord. It is attended by redness, rise of temperature, pulsation and tortuosity of the vessels, sweating, unrest, ringing in the ears and palpitation. In paralysis of the cervical sympathetic there is pupillary narrowing from the unopposed action of the third nerve, slight ptosis, narrowing of the interpalpebral fissure from paresis of Müller's muscle, salivation, lachrymation, and in cases of some standing, retraction of the eye and flattening of the cheek. The pupil reacts somewhat to light but not to stimulation by pain or pinching of the skin of the neck (Möbius); the pupil can be widened by atropine, but to a less degree than in the normal eye.

Vasomotor spasm presents converse symptoms, pallor, coldness, small vessels, and frequently a sense of tingling or arterial pain. Two examples are nervous angina pectoris, and Raynaud's disease; spasm (contraction, irritation) of the cervical sympathetic produces wide pupils, an increased interpalpebral fissure and exophthalmos.

Little is known of the *trophic nerves*, if indeed separate nerves subserve nutrition. If they exist, they are probably closely related to the vasomotor tracts. Trophic changes may be symptomatic of disease of the nerves or cord, as in neuritis or nerve trauma, hemifacial atrophy, tabes, syringomyelia, and myelitis, under which their special features have been already discussed.

Erythromelalgia was described first by S. Wier Mitchell (1872); the term indicates the chief clinical characters, *redness and pain in the extremities*. It is rare, though much more frequent than is shown by Rost's series of 40 cases. It occurs mostly between the years of twenty-five and forty and is slightly more frequent in males than in females. Its pathology is not wholly clear, although Mitchell and Spiller find neuritis and sclerosed arteries. The toes (less often the fingers), and balls of the toes, are the seat of severe, usually paroxysmal pain, swelling and redness; the arteries throb and the veins are engorged; the color is sometimes blue. Pain is sometimes experienced in the head and neck, and vertigo and muscular weakness may be noted. The symptoms occur mostly in warm weather, and generally disappear in winter. The clinical course is long, the prognosis doubtful and the treatment consists of electrotherapy, cool baths, acetanilide and iodides.

Acroparæsthesia is closely related to erythromelalgia. It was described first by Nothnagel, and F. Schultze (1890) named this very frequent affection; its derivation signifies *paræsthesia of the extremities*. More than 90 per cent. of cases occur in women between thirty and sixty years of age. The etiology is doubtful (stasis or arteriosclerosis). Its chief feature is an unpleasant crawling, tingling sensation, sometimes amounting to actual pain in the fingers or hands, less often in the toes or feet. The discomfort is greatest at night and in the early morning; it is peripheral and symmetrical but does not correspond with any nerve area. Generally there are no objective findings, other than occasional vasomotor disturbance or slight sensory blunting. The outlook is favorable, the course is chronic and treatment is symptomatic; the faradic brush frequently affords relief.

Spontaneous symmetrical gangrene, or **Raynaud's disease** was first described by Raynaud (1862). Sixty-six per cent. of cases occur in women, who are usually delicate, anæmic and neurotic, and most cases develop in persons between twenty and forty-five years of age. In 10 per cent. it is hereditary. Other causal factors are uncertain; exposure to cold and dampness, exhaustion, worry and acute infections are sometimes considered of etiological importance. Occasional co-existence is noted with hysteria, neurasthenia, epilepsy, tabes, syringomyelia, myelitis, angioneurotic œdema, peripheral neuritis and acute mania. Its pathology is not wholly established, but arterial spasm, endarteritis and neuritis are said to exist. There are *three stages;* (*a*) *Local ischæmia* (or syncope), begins with pallor and coldness of one or two fingers, perhaps of the entire hand, or of the toes, and is attended by numbness or severe pains. The "dead fingers" or toes appear on exposure to slight cold or on emotion, and reaction occurs after a variable period. The process may not pass this stage. (*b*) *Local asphyxia* (or cyanosis) develops in the parts involved, at times with some swelling or anæsthesia. It is most severe in winter, and chilblains are regarded as the mildest type of this stage. (*c*) *Local gangrene*, which is spontaneous, symmetrical and fortunately rare, is the last stage. It is dry, sometimes vesicular, and its extent is always less than the area of ischæmia and asphyxia. The tips of the fingers or toes dry up, fall off and scars mark successive attacks. Less often the tip of the nose, ear, buttock, heel, shoulder or abdomen is involved, and very rarely the tongue, penis, elbow, or knee. Fever is usually absent. In some instances there is severe general disturbance, as fever, chill, enlarged spleen, colic, hæmoglobinuria, uræmia, stupor, coma, convulsions, transient hemiplegia, neuritis and spastic retinal vessels. The severe or fatal cases occur especially in children. The *course* is chronic and consists of repeated attacks, which often abate in summer. *Differentiation* is usually easy from hysterical gangrene (self-injury), diabetic, arteriosclerotic or embolic gangrene, syringomyelia, leprosy, ergotism and occlusion by acute arteritis, as in typhoid.

In *treatment*, narcotics should be given for pain; warmth and elevation promote venous return, and nitroglycerine dilates the arteries. Cushing advises that Esmarch's constrictor be applied for a few minutes, after which the circulation is restored. In actual gangrene, surgical interference should be postponed until complete demarkation develops, because the area is usually much more circumscribed than at first indicated.

Acute angioneurotic œdema was first fully described by Quincke (1882), although Milton (1876) described "giant urticaria." It occurs most frequently between the years of twenty and thirty-five and is slightly more frequent in women than in men. Hereditary forms are known; 22 cases occurred in 5 (Osler), and 22 cases in 6 (Milroy) generations of one family. It is allied to urticaria, erythema nodosum and possibly to some forms of purpura, and consists of a circumscribed œdema, which is sudden in onset and in disappearance, is tense and pale, does not pit on pressure, sometimes causes burning, and appears mostly on the eyelids, lips, cheek, tongue, hands or genitalia, sometimes on the buttocks, pharynx, larynx, lung or digestive tract. It sometimes attacks several areas simultaneously. Gastro-intestinal symptoms occur in 33 to 50 per cent. of cases, as epigastric pain, vomiting, colic or diarrhœa (see page 867). Hæmoglobinuria, albuminuria and polyuria are less frequent. The outlook is good, except for the dangerous laryngeal or pharyngeal localization which may cause sudden death. *Recurrence* is not uncommon, and sometimes occurs with considerable periodicity. *Treatment* is symptomatic and general; nitroglycerine has been recommended. Bloodgood employed deep incisions with great benefit.

Twenty-two cases of **chronic hereditary trophœdema** in six generations were described by Milroy (1893) of Omaha, and 8 cases in four generations by Meige (1898). A firm, pale, painless œdema, chiefly below the hip or knee, develops toward puberty, without cardiac, renal or apparent local causes. It may appear in the arm and leg of the same side, including probably some of the so-called local hemihypertrophies. The first cases recorded were initially thought to be phlebitis. Health and life are not threatened, although treatment produces no results.

Hydrops articulorum intermittens (hydrarthrosis intermittens) or intermittent hydrops in the joints, occurs with equal frequency in both sexes, and 71 per cent. occurs in persons between ten and forty years of age. Its exact etiology is not known. Some cases present malarial, gonorrhœal or traumatic antecedents. The knee is usually involved when several joints are affected, and in 33 per cent. is alone involved. The effusion wholly disappears after two or three days, but it reappears obstinately every one to four weeks. There is no fever and usually no pain. Treatment is general.

Scleroderma was described by Lusitanus and Alibert, though Thirial (1845) gave the first full account of it. It is a chronic thickening and hardening of the skin, followed by atrophy. It is a rare affection, but is more common than is generally recognized. The *localized form* usually develops before, and the *generalized form* after, the twenty-fifth year. Seventy-five per cent. of cases occur in women. The *causation* is obscure; erysipelas, trauma, cold, and neuropathic disposition are sometimes considered etiological, and association with other neuroses, general and vasomotor, is frequent. *Anatomically* the skin and all the contained structures are fibrosed. In the localized form it occurs in spots or streaks, which often correspond to the course of given nerve trunks or spinal segments. In the general form the body may be literally "hide bound." Its distribution in order of frequency is (*a*) the upper extremities; (*b*) the trunk; (*c*) the head and face; (*d*) the lower extremities; (*e*) generalized. The skin is usually first painful and sensitive, then reddened and thickened, and finally pale, glossy, dry, atrophic and parchment-like. It is rigid and without folds, inhibiting the mimetic play of the features. The nose is peaked, the lids cannot close over the eyeball and may become everted. The mouth is so small as to sometimes necessitate extraction of some of the teeth, and rigidity in the neck may render swallowing difficult. In the fingers it occurs in 5 per cent. of cases, the condition being known as *sclerodactylia* (Ball). The close skin produces decided deformity and loss of function. Sensation is usually preserved, the secretion of sweat is usually reduced, and pigmentation and trophic changes in the hair and nails are occasionally seen. In some cases it occurs with herpes; this is the herpetiform morphea of Hutchinson. Circumscribed atrophy of the bones is said to occur. Only 30 per cent. of cases improve, and 15 per cent. recover, whence the outlook is generally unfavorable. *Treatment* is unsatisfactory. Improvement has followed administration of salol and inunctions with oil; thyroid extract (Osler, Lustgarten, Sachs and Bramwell), especially if used early in the disease, may promote recovery.

Ainhum was first observed by Clark (1860) then by Da Silva Lima (1866). It is a chronic affection, usually limited to the great toe, which at its plantodigital fold suffers a slow, painless, non-inflammatory circular constriction and spontaneous amputation. Ainhum is chiefly found in the colored races of Brazil, Africa, India and sometimes in this country. Its nature is disputed; an ingrowth of the epidermis and scleroderma have been thought to be the cause. It is not amputating leprosy, as has been claimed.

VI. DISEASES OF THE MUSCLES.

1. MYOSITIS.

Inflammation in the muscles may be single or multiple; it may be an independent affection or a secondary one. It may reach the muscles by contiguity from adjacent disease, may result from trauma, trichinosis, infection with cysticercus, echinococcus, sporozoa (myxo-, sarco- and microsporidia), sepsis, puerperal fever, rheumatism, scarlatina, gonorrhœa, syphilis, or erythema multiforme. The muscles are the seat of serous or sometimes hemorrhagic or diffuse purulent exudation in septic forms, and of fatty degeneration. There is a form, endemic in Japan, in which the Staphylococcus pyogenes aureus occurs in pure cultures. The muscles are usually friable and sometimes fragmented. Polymyositis first described by Wagner (1887), Hepp and Unverricht, occurs most often in youth and adolescence and is attended by pain especially near the tendons, swelling and often œdema in the muscles, the electrical irritability of which is decreased as also are the tendon reflexes. The skin may participate, and herpes, erythema, bullæ or purpura (*dermatomyositis*) may be present; the infection may involve the tissue in which the afferent nerves originate (muscle spindles) and topographically a neuritis and myositis may co-exist or coincide (*neuromyositis*, Senator). In some cases there is arthritis or phlebitis. Oppenheim describes a case of *dermatomucomyositis* with stomatitis, iritis and conjunctivitis. The inflammation occasionally reaches the tongue, pharynx, larynx, or respiratory muscles, and may even cause difficult mastication or swallowing, ptosis or dyspnœa. The systemic reaction is usually severe, is marked by fever, chills, sweats, splenic tumor, sometimes by angina, bronchitis, albuminuria or leukocytosis. The course is usually fatal.

Differentiation concerns *multiple neuritis* (tenderness over the nerve trunks, motor and sensory findings); trichinosis (finding trichinæ in the tender, swollen muscles, increase of the eosinophile cells, and digestive symptoms); and the *periarteritis nodosa* of Kussmaul and Maier (*q.v.*)

Treatment is symptomatic, and is identical with that of sepsis. Sweats and administration of aspirin are beneficial.

Myositis ossificans is characterized by a myositis which usually develops acutely in the muscles of the neck or back before the fifteenth year, and leaves residual foci of infiltration, which gradually calcify. The thigh and arm are most severely and permanently affected. The process occurs in the strongest muscles and at an age when they develop most actively; it may affect many muscles and runs a chronic course, perhaps of several decades. One hundred and thirty-four cases are recorded (Cahier); there were 101 traumatic cases (Werner, 1904). Massage is sometimes beneficial.

2. MUSCULAR RHEUMATISM (MYALGIA).

This includes many vague but important affections. It occurs particularly in men, after exposure to cold and dampness, and is obviously promoted by certain occupations. Muscular overexertion, as straining and lifting, is frequently a part of the history. Rheumatic and gouty subjects are specially exposed and to recurrent seizures. Disorders of metabolism or mental as well as physical overwork mark some cases. Other forms are, as Leube insists, probably infectious, for fever occurs in 33 per cent., and is occasionally followed by endocarditis; muscular rheumatism in this sense does not usually involve many muscles, but usually one muscle or a single group. The pain is not paroxysmal, as in neuralgia, and is experienced in the muscle or reaches it from its tendons or fasciæ. In some cases it is apparently a mild neuromyositis. Its varieties are; *Myalgia capitis*, affecting the fascia of the head; *omalgia*, involving the muscles of the shoulder, or rheumatic *torticollis*, which is usually unilateral; *pleurodynia*, which affects the chest muscles, pectoralis, intercostals, and serratus, limits respiratory movements and is likely to be confused with intercostal neuralgia, pleurisy, periostitis of the ribs, or vertebral disease; *lumbago*, which involves the lumbar muscles, and is the most frequent, painful and obstinate form, causing great pain on movement or on efforts of the uninvolved muscles to maintain quiet; it is probably due in some cases to muscular rupture, as after lifting. In the abdominal muscles and glutei, it may simulate pelvic disease. In *treatment*, rest is urgently indicated, for use of the affected muscles aggravates pain and protracts the condition. Involuntary movement is prevented by careful strapping with adhesive plaster, especially when there is pain in the chest. A hypodermic of morphine, into the muscle, is often indicated.

Deep injection of water into the muscles of the back may relieve pain. Ringer's method of acupuncture with long needles which are pushed three inches into the back and left there for from five to ten minutes is beneficial, but is usually vigorously opposed by the patient. Very energetic massage, with the local application of heat, probably gives the best results. The actual cautery, the application of the constant current, and the use of Tesla's coil give good results. The use of belladonna plasters gives relief. Gelsemium, acetanilide and salicylates often produce no effect, but may instantly relieve certain cases.

Chronic cases, with vague, fugitive pains which come on with change of weather, are treated in the same way—usually without result. Confusion with chronic alcoholism, tabes, gout, and diabetes is frequent.

SECTION X.

INTOXICATIONS. SUNSTROKE.

(A). ALCOHOLISM.

I. Acute Alcoholism.

(*a*) The *highest brain centres* are first affected. The judgment is first involved (emotion predominating), motility next (as shown by the disordered speech, gait and vision), then the lower centres (respiratory, circulatory and spinal). (*b*) The *circulation* is increased and the pulse becomes more rapid, though it is held that alcohol does not stimulate the heart. Full doses may make the pulse slow and small. The vascular tone is lax and the face is therefore flushed; for this reason alcoholics suffer little shock from coincident traumatism. (*c*) *Respiration* is slow, deep, and seldom stertorous. (*d*) The *pupils* are variable, often dilated or sometimes narrow and reactionless. (*e*) The *temperature* may fall markedly, from vasoparesis, and usually with some cyanosis and failing of the bulbar centres; Reincke states that temperature has fallen as low as 75°! Involuntary evacuations occur in the severest cases and there may be subcutaneous hemorrhages.

Dipsomania is intermittent acute alcoholism, an hereditary psychosis in which, after intervals of abstinence and of no craving for liquor, the patient becomes intoxicated.

Diagnosis.—The alcoholic breath is usually suggestive but epileptics and uræmics may drink, and while drunk, a man may have his skull broken or suffer an apoplexy. Many mistaken diagnoses result from carelessness, but many are unavoidable, and the apparently drunk patient should be treated for alcoholism, while other possibilities should be carefully considered. As a rule the patient can be aroused, but in "dead drunks" this is not the case, and incomplete coma may occur from other causes. The condition of the pupils is not of certain diagnostic value, though they are usually dilated in alcoholic, contracted in opium and uræmic, and unequal in apoplectic, coma. Stertorous breathing is strongly indicative of apoplexy or uræmia. Convulsions following delirium and muscular twitchings are less common than in other similar conditions, but at the time of writing, the author has a case which showed uni- and then bilateral convulsions. The urine rarely contains alcohol, as but one or at the most 5 per cent. of ingested alcohol is excreted by the lungs and kidneys. One-third of all the fatal poisonings in New York and Berlin are acute alcoholism.

Treatment.—Gastric lavage is sufficient in most cases. Even the convulsions require little attention, but when severe, yield to a few whiffs of chloroform. Acute mania yields to a hypodermic of apomorphinæ hydrochloras (gr. $\frac{1}{6}$). Collapse, cold extremities, and cardiac and respiratory failure are relieved by hot coffee, strychnine, atropine, aromatic spirits of ammonia and local heat, as will be more fully outlined under acute opium poisoning (*v. i.*).

II. Chronic Alcoholism.

This is less an hereditary tendency toward drinking than a lack of character. The constant use of alcohol has varying effects; some people can take beer, wine or spirits throughout life without apparent injury; in others, even the mildest alcoholic drinks may induce visceral alterations, so that as in liver cirrhosis, it would seem that there is some other etiological factor; however, alcohol in health is a bad food, an injurious narcotic or stimulant, a protoplasmic poison to the parenchymatous organs and arteries, and a check on metabolism.

Symptoms.—(*a*) There may be *nervous* manifestations. Functional changes include the alcoholic tremor, which is a most common symptom; mental dulness and weakness; nervousness, jumping from slight stimuli, irritability, impaired judgment and feeble will power; neuralgias and muscular rheumatism. Quinquad's sign is elicited by having the patient place his fingers vertically to the examiner's hand or body, when a series of jerking shock-like movements are noted; Fürbringer decides that (i) if it is absent, alcoholism is absent; (ii) if it is moderate, there is uncertainty and (iii) if it is marked, in 3 cases out of 5, the patient is an alcoholic. Of organic nervous manifestations, multiple neuritis is the most common; it may result from small amounts of alcohol, when there is a coincident metallic poisoning; it is much more often a promoting factor than an actual cause of paretic dementia; epilepsy may result from alcoholism; hemorrhagic pachymeningitis, chronic leptomeningitis, acute polioencephalitis, degeneration in the anterior horns, or Clark's columns, degeneration of the cerebral nervous tissue and slight optic neuritis may be more or less direct sequences. (*b*) The *circulation* is affected. Arteriosclerosis often develops with its train of sequences, cardiac hypertrophy and dilatation, angina pectoris, cerebral softening or hemorrhage. (*c*) *Chronic bronchitis* and emphysema are frequent collateral findings. There may be chronic pharyngitis and laryngeal thickening. (*d*) The *digestive organs* are involved. Alcoholic gastritis (*q. v.*) is common; the morning vomiting (vomitus matutinus) is quite characteristic, and is quieted only by another dose of its cause. Beer drinkers usually have large stomachs. Alcoholic cirrhosis has been already discussed; fatty degeneration, arterial congestion and hypertrophy may antedate or accompany cirrhosis. Beer drinkers have lax bowels, while consumers of spirits are constipated. Hemorrhoids are common. (*e*) The *kidneys* undoubtedly suffer. Dickinson, Formad and Pitt think that renal disease is less common than is usually believed; Formad and Pitt found that the kidneys were hypertrophied without other change in 43 per cent. As in cirrhosis, several factors may be necessary to produce indurated kidneys. (*f*) *Metabolic maladies*, as gout, obesity, and, according to Strümpell, some forms of diabetes may result from chronic alcoholism. (*g*) The *skin* frequently presents the acne rosacea, a very common but not, as Trousseau says, an "indelible stigma." The vessels of the nose, cheeks and often of other parts are suggestively dilated; the conjunctivæ are injected and the eyes water.

Prognosis.—The prognosis is unfavorable, both from the standpoint of curability and complications. English life insurance companies estimate that the risk is 25 to 43 per cent. less in abstainers than in alcoholics. The general resistance is lowered, thus favoring infections, as tuberculosis, and raising greatly the death rate, most notably in pneumonia. Twenty per cent. of suicides are alcoholics. The offspring of inebriates are disposed to numerous nervous affections.

Treatment.—*Treatment* consists of correction of the habit and care of the complications, the latter of which have, in great part, already been considered. The cure of the habit is largely personal. Its prophylaxis is better than its cure. No person, however intellectual or strong-willed, can safely drink with any regularity. Institutional treatment is generally necessary. The vomiting should be treated in accordance with instructions given under acute and chronic gastritis; lavage is probably the most reasonable measure; a mixture of bismuth (see TYPHOID, VOMITING) and small doses, (♏ i to ii) of Fowler's solution and large ones of tr. nucis vom. (♏ x) are valuable just before the administration of hot milk with tr. capsici, ♏ x. Hyoscine (gr. $\frac{1}{100}$) should be given two or three times at 3 hour intervals, chloral hydrate (gr. x) and sod. bromide (ʒ ss) for three or four doses, before bedtime, or sulphonal (gr. xx) in hot milk before bedtime, in order to induce sleep. Atropine (gr. $\frac{1}{100}$) and strychnine (gr. $\frac{1}{30}$) four or six times daily, hypodermatically, quiet the nervousness, which occurs in the first days of abstinence. Sipping ice-water, sucking lemons, or drinking water with tr. quassiæ, may satisfy the desire to "drink something." Cocaine and morphine should be avoided, lest a substitution or succession of habits develop. Relapse is very common.

III. Delirium Tremens.

Delirium tremens (*mania a potu*) is an acute accident of chronic alcoholism, and almost never follows an acute debauch in an otherwise temperate individual. It was noted among sailors by Sutton of Greenwich in 1813, but was probably best described by Ware of Boston in 1831. Sixty-six per cent. of cases occur in men, while multiple neuritis is more common in women (Gowers); most victims are between thirty and forty years of age. The stronger spirits, rather than beer or wine are the cause of delirium tremens as they are of alcoholic convulsions. It may be precipitated by trauma, mental shock, the shock of onset of an acute infection, or by sudden failure of the stomach to digest and assimilate food.

Symptoms.—The symptoms develop with restlessness and insomnia, which renewed drinking fails to allay; for a day or two the mind wanders, but can be brought back to realities. The patient may be facetious, but is soon disturbed by horrible hallucinations. There is "great versatility of ideas" in delirium tremens. The patient talks volubly, turns suspiciously to the head of his bed, drives his horses vigorously, and brushes away the vermin from the coverlet. He at first knows that the bugs and snakes are unreal, but they soon become actual torments, so that unless watched he will leap from the window after waiting for his opportunity. The pulse is rapid and soft, the tongue is tremulous and is coated white, the urine is scanty, and sleep is constantly absent. There may be moderate fever, 101 to 103°, which some attribute rather to complications.

Diagnosis and Prognosis.—The diagnosis is rarely uncertain. The chief danger of error is in overlooking coincident causal trauma and infection, as pneumonia. In heavy and constant drinkers, a delirium may be delirium tremens or an "associated delirium," as from apical pneumonia.

The *outlook* is always uncertain. The early convulsions are of no great moment (Gowers). The prognosis is determined by the pulse, the general nutrition and complicating alcoholic or other lesions. Rigidity of the muscles of the neck is ominous (Putnam), and indicates œdema of the brain. The average mortality is 15 to 20 per cent., but varies as the affection is (*a*) uncomplicated (relatively good prognosis), (*b*) associated with other affections (grave outlook) or (*c*) occurs in the aged or debilitated (probably fatal outlook). Recurrence after recovery is frequent, and 27 attacks in one case have been recorded. In fatal cases death usually results from exhaustion and heart failure; the tongue becomes dry, the delirium constant, and the skin cold and clammy. The autopsy shows œdema of the brain ("wet brain").

degenerative changes in the brain and cord (Bonhoeffer), arterial alteration (Collins), and hypostasis of the bases of the lungs.

Treatment.—(*a*) The patient should be *kept in bed* and watched constantly lest he escape. Strapping the ankles and wrists and confining him by a straight jacket is a harsh procedure, and invites trauma to the hands and feet, but is inevitable in cases where the family is indulgent, or when the nurses are negligent. (*b*) *Withdrawal of alcohol* is usually advocated but is a severe measure; the continuance of moderate doses seems to hold the pulse steady, and favors the digestion and is, in the author's belief, indicated. (*c*) *Sleep is induced* with great difficulty and as Ware first pointed out, comes when the disease runs its course. Excessive medication should be avoided. Sod. bromide (ʒss), tr. capsici (♏ v) and whiskey (ʒii), in hot peptonized milk should be given every (two or) three hours, by mouth if possible, or by the nasal catheter, for nutrient enemata are difficult to give and are seldom retained. Chloral is always dangerous in inveterate alcoholism. Hyoscine with strychnine may be beneficial (*v. s.*) Morphine should be given only by the hypodermic method, for its absorption can then be measured; it was formerly given to excess. Protracted warm baths may quiet the patient, but they are hard to administer. In 6 cases in which the violent delirium became ominous, the writer has etherized the patient, and in 3 instances with excellent results, though he does not advocate its wide use. (*d*) The *circulation* should be treated as in pneumonia. Large doses of digitalis are not indicated; they are tolerated only because they are not absorbed, as the original treatment by digitalis proves; it is based on the fact that a nurse by mistake gave a toxic dose of digitalis; the patient improved and the "digitalis treatment" was thus instituted. (*e*) *Cold affusions* upon the neck and chest are recommended by Broadbent.

(B). OPIUM POISONING.

I. Acute Poisoning.

This is important chiefly from a diagnostic standpoint. In the second stage it is strongly characterized by profound sleep, dry, flushed skin, narrow pupils, slow, strong "digitalis pulse" and slow deep stertorous breathing. The third or terminal stage is different; the skin becomes cyanotic and clammy, the narrowed pupils may dilate, the pulse become weaker (though the heart centers live after the other bulbar centres have died), respiration halts, and is very slow. Mistakes in diagnosis are made by attributing undue importance to the "pin point" condition of the pupils. Morphine may be detected in the stomach washings and in the urine.

Treatment.—This consists of (*a*) *mechanical evacuation* by repeated gastric lavage. (*b*) *Chemical antidotes*, such as large amounts of tannic acid, should be given with the lavage, and removed as the tannates formed may dissolve; 1 to 1,000 permanganate of potash (Moor, of New York) may be given by mouth or hypodermatically. (*c*) *Respiration* must be sustained by walking the patient (the ambulatory treatment), but fatigue must be guarded against; phrenic faradization is indicated, but care is necessary lest too strong a current produce paralysis instead of stimulation; strychnine plus atropine may be given hypodermatically, but the total dose of atropine should not exceed gr. $\frac{1}{30}$ to $\frac{1}{20}$; pulling slowly and rhythmically on the tongue may help respiration; Sylvester-Hall's artificial respiration and oxygen inhalation may be used. (*d*) *The heart* should be stimulated by coffee, strychnine, heat and digitalis.

II. Chronic Morphinism (Morphinomania).

The use of morphine for pleasure only, so common in Asia, is rare with us. The habit is contracted by its use for insomnia or for mental or physical pain. Forty-seven per cent. of morphine victims are said to be physicians; women and—in Germany—officers rank next. It may be taken for years without mental or physical deterioration, but morphinists are usually less happy, as increase in the daily dose becomes necessary and the half-oxidized alkaloid produces symptoms (*v. i.* TREATMENT) which are only allayed by larger doses. Morphine acts as a nerve poison. The victim becomes restless and irritable without his dose. The mental and moral attributes are anæsthetized and there is a "moral insanity." The patients lie by choice, neurotic symptoms are common, muscular and mental asthenia develop, the pupils are contracted, the sweat and saliva are decreased, the appetite fails, the skin becomes sallow, itchy and dry, the hair coarse and gray, constipation is usual, and slight ataxia may develop.

Prophylaxis.—Physicians should not give opiates too freely. When they are administered for severe pain the habit is less often contracted than when given to neurotics. At least 70 per cent. of cases relapse after a cure.

Treatment.—Treatment is almost impossible without (*a*) *institutional confinement*, which must of course be voluntary. (*b*) The *morphine* may be *withdrawn* abruptly, rather suddenly or very gradually; the second method is the best, stopping the morphine entirely in four to seven days. Its withdrawal is attended by awakening of dormant anæsthetized functions; there is at first paroxysmal yawning and sneezing; roughening of the voice, paræsthesia, neuralgias especially in the legs, clammy skin or rapid pulse, wide pupils, tremor, sexual excitement, extreme irritability, vomiting, colic and diarrhœa also occur. The symptoms were attributed by Marme to di-oxymorphine poisoning, the antidote to which is morphine; Hitzig believes that they are largely due to hyperchlorhydria, for the gastric nerves under the influence of morphine secrete little hydrochloric acid; relief is obtained by washing out the stomach with a solution of

Carlsbad salts. The patient will obtain morphine if possible. (*c*) The *patient should be kept in bed* for a week to obviate vasoparetic cardiac collapse. (*d*) *Symptomatic treatment* is indicated (i) by the *collapse* (aromatic spts. of ammonia, ♏ xx, strychnia gr. $\frac{1}{30}$, heat); (ii) by *nervousness* (warm baths, sodium bromide in doses of one dram every four hours); (iii) by the *insomnia* (hyoscine gr. $\frac{1}{100}$ every two hours for two to six doses, sulphonal or trional, gr. xx); (iv) of the *gastralgia* (gastric lavage with alkaline salts); (v) by *diarrhœa* and colic (as in enteritis, but vegetable astringents, large doses of bismuth, and no opiates should be used); (vi) by *neuralgia* (acetanilide, gr. v, fluidextr. gelsemii., ♏iii). Whatever treatment is instituted, alcoholism, chloralism, bromism or cocainism should not be substituted.

(C). LEAD POISONING.

Etiology.—Plumbism or saturnism is very common, and its importance is daily increasing. (*a*) *Industrial plumbism* is very common. Workers in white or red lead factories usually acquire the disease. Miners of the metal generally escape, but smelters of lead ore, and even animals and birds near the furnaces, are frequently affected. Painters, plumbers, glaziers, less often tinners, printers, cameo polishers, cartridge makers, etc., may be affected. (*b*) *Accidental contamination of food or drink* may occur. Poisoning may result from the action of acids in canned foods on the tin or solder, as in cans of sardines; wine or cider may become poisonous in the same way, as happened in Devonshire, and in Poitou (*colica pictonum*). Chromate foil, covering hams or candy, rubber nipples, bullets in pickled game, bread ground in lead vessels, chromate used to color buns (D. D. Stewart's report of the fatal Philadelphia epidemic), Seltzer or other charged waters, wine, and *eau de vie* sweetened or cleared with litharge, are a few of the many subtle causes of saturnism. Drinking water conveyed through lead pipes is generally protected by the deposit of lime and other salts, but poisoning is possible when the water is exceptionally pure or soft, thereby depositing little lime in the pipes, or when it is saturated with organic matter or collected from lead roofs. (*c*) Less frequent sources of plumbism are in the *environment*, and in such things as hair-dyes, powders or cosmetics, linen, freshly painted walls, candles, sealing wax, brightly tinted toys, false teeth, and thread. (*d*) *Therapeutic* and *criminal plumbism* is rare; it may result from the acetate of lead when given internally or externally, and from impure subnitrate of bismuth; its criminal use is very uncommon. (*e*) *Predisposing factors* are alcoholism, age (it occurs most often in persons from thirty to forty) and sex (75 per cent. of cases are females, Oliver). Susceptibility varies greatly; plumbism may cause death within a week, or may not develop until after forty-two years of work in the metal (Tanquerel des Planches). *Absorption of lead* usually occurs (i) by the digestive mucosa; most of the metal is precipitated by the albumin of the food and eliminated without absorption; (ii) by the respiratory mucosa, by inspiration as dust in white lead factories; or (iii) by the sound or diseased skin (cosmetics) or even the conjunctiva or vagina (lead douches). It is *eliminated* chiefly by the urine; Putnam stated that he found lead in 25 per cent. of 150 persons who had no evidence of plumbism; the liver and alimentary mucosa eliminate smaller quantities.

I. Acute Poisoning.

This is uncommon, though it may occur from therapeutic, criminal or professional causes. Its symptoms are (*a*) gastro-intestinal; a styptic burning taste occurs in the mouth, thirst is present, there is a lead line on the gums in some cases, though less than in chronic poisoning, nausea and vomiting sometimes of white lead chloride, abdominal colic, retraction of the abdomen, diarrhœa with black (lead sulphide) or bloody movements, or sometimes constipation. On postmortem examination, the catarrhal gastro-enteritis may be wrongly considered a postmortem change; white spots may be seen (Orfila's spots). The symptoms may be (*b*) circulatory; the pulse may be slow (40 to 50) and irregular; anæmia, cyanosis and collapse may occur; (*c*) they may be nervous; vertigo, stupor, delirium, neuralgia, cramps, convulsions, and even cerebral and peripheral paralysis may develop; (*d*) nephritis with suppression of urine may develop.

Treatment consists of full doses of sodium and magnesium sulphate, in order to precipitate an insoluble lead sulphate, and to remove it, demulcent drinks (eggs and mucilages), opium for pain, and cardiants.

II. Chronic Poisoning.

Symptoms.—(*a*) *Colic* is the most common symptom; it occurred in 88 per cent. of Tanquerel's 1,390 cases. It is due to local deposit of lead in the intestines (muscles or mucosa), spasm of the bowel, changes in the nerves or angiospasm. It may develop early or late; it may be preceded by malaise, lumbago or pain in the legs, or may be brought on by alcoholic excesses. It is a colic accompanied by constipation (dry colic), rarely by diarrhœa; it is umbilical, whence it spreads over the abdomen, perhaps to the legs or scrotum; it is relieved by pressure, though sometimes the abdominal wall is hyperæsthetic; sometimes vomiting and absolute constipation may simulate obstruction, and in one case the author observed intussusception as a result of lead poisoning. There is no fever, the pulse is small and hard (Riegel) and the urine is scanty. Without treatment colic is protracted; its relief is usually marked by a bowel movement. Relapses are very frequent. (*b*) The *gingival lead line*, which is present in over 90 per cent., is a gray-black line, 2 to 3 mm. wide, which is seen most clearly on the lower gums and is due to deposition from the blood of lead sulphide, precipitated by the sulphuretted hydrogen of the tartar. The coincident anæmia brings it out very clearly. Similar deposits may occur under the buccal mucosa opposite the molars. It is very frequently mistaken for tartar, which occurs on the teeth, sometimes for the black carbon line seen in miners, or rarely for the silver line in argyria. (*c*) *Other digestive symptoms* are coated tongue, fœtid breath, parotitis, saturnine dyspepsia and rarely icterus. Test meals show lactic acid and no HCl (Sailer and Speese). (*d*) *Anæmia* is very frequent in the "lead cachexia." The hæmoglobin and erythrocytes decrease moderately; the red cells show basophilic granules (Grawitz) and quite commonly nucleation, even in mild cases (Cadwalader, 1905). The leukocytes may be increased. (*e*) The *heart and vessels* are affected. The heart is very often hypertrophied, as is evidenced by a loud second aortic tone and aortic regurgitation of atheromatous origin; the arteries are often sclerosed, occasionally causing angina pectoris, frequently myocarditis, irregular heart action and perhaps some of the brain symptoms (*v. i.*). The asthma saturninum is probably cardiac or uræmic. (*f*) The *kidneys* are often sclerotic, as a result of the elimination of the metal. It is said that the change does not begin around the vessels but in the tubules, which are the seat of a necrosing deposit of carbonate of lime. Nephritis, gout and plumbism may co-exist (*v.s.*). (*g*) *Nervous manifestations* may develop. Peripheral degenerative *neuritis* occurs in 7 per cent., usually as a later symptom. About one-tenth of these cases have not had colic. It is characterized by the paralysis of muscles with the same function, by muscular atrophy and cramps, tremor, cutaneous hyperæsthesia, neuralgia, arthralgia (55 per cent.), myalgia and by varying degrees of reaction of degeneration (*v.* MULTIPLE NEURITIS). The musculospiral type, "*wrist drop,*" is the most common and is bilateral; the supinator longus (a flexor) and the small extensor of the thumb escape involvement; tenderness and anæsthesia are rare. Gubler's tumors are swellings of the extensor tendon sheaths and are due to backward dislocation of the carpus or perhaps to a trophic change; they are hard, ovoid, indolent, seldom painful or larger than a pigeon's egg, and regress with the paralysis. A second but rare form is the *brachial,* involving the deltoid, biceps, brachialis anticus, and supinator longus, rarely the pectorales. A third rare type is the *Aran-Duchenne,* which is especially observed in tailors (Möbius), and involves the small muscles of the hand, the thenar and hypothenar eminences; it resembles that of chronic anterior poliomyelitis (which indeed has been found in a few cases at autopsy). A fourth type, the *peroneal,* involves the peronei and toe extensors; according to Tanquerel, but 13 per cent. of lead palsies affect the leg. Lastly, rarer types may involve the larynx, vagus, and face; generalized paralyses, either slow or rapid like Landry's paralysis, are very rare; primary muscular atrophy may occur, is most often of the Aran-Duchenne type, and shows fatty and fibroid muscular changes.

Cerebral forms (*encephalopathia saturnina*) occur in 5 per cent., they are often preceded by headache, colic, and insomnia, and are attended by amaurosis, limitation of the visual field, or other disturbance of the special senses, retinitis albuminurica or saturnina (with inflammation, engorgement and possibly ultimate atrophy), and paralysis of the eye muscles. The most frequent type is the *delirious* encephalopathy, which is variable or mobile in character, and sometimes resembles delirium tremens; the next type is the *convulsive* (epilepsia saturnina) which occurs without auræ, is usually generalized, often fatal, and is always to be considered when epilepsy develops in an adult; the third type is the *comatose*. Hysteria frequently occurs with plumbic encephalopathy. Neglected or unrecognized cases may become dements (pseudodementia paralytica saturnina). Deposit of lead in the protoplasm of the brain is the usual cause; sometimes the cerebral symptoms are arteriosclerotic (as hemiplegia) or uræmic.

Lead-poisoning is usually *recognized* (*a*) by the etiology, (*b*) by the lead line, (*c*) by lead in the urine, (*d*) by colic and (*e*) by wrist-drop. In a case of long coma with choked disk, in the Cook County Hospital, no cause was found, until after a second examination, the lead line was seen. The man was a barber, who regularly dyed the hair of a number of people, and probably absorbed the lead through his hands.

Prognosis.—The acute forms are more favorable. Vascular and renal changes are largely incurable. Atrophic paralyses are serious. The wrist-drop usually regresses, though it is sometimes permanent. Coma, convulsions and other cerebral symptoms are serious; about 25 per cent. of cases die, and after apparent recovery, mental degeneration may develop.

Treatment.—(*a*) *Prophylaxis* should be observed. A mask should be worn in white-lead factories to avoid inhalation of lead dust; workers are careless and poisoning is frequent despite precautions. Painters and others may often avoid poisoning by carefully cleaning the hands and nails before eating. (*b*) *Lead colic* is treated by hypodermic injections of morphine and atropine to control pain and spasm. Riegel uses nitroglycerine for the supposedly causal spasm of the vessels. Spts. of chloroform (♏ xx) with other aromatics may be given by mouth. (*c*) The *constipation*, and removal of any lead salts, unabsorbed in or excreted by the alimentary mucosa, necessitate catharsis. Full doses of castor oil (℥iv or more) with paregoric (℥i) evacuate the bowels, and relieve pain and spasm; sodium and magnesium sulphates purge and coincidently precipitate the lead as an insoluble sulphate. (*d*) In order to *eliminate the lead* from the tissues, with the protoplasm of which it has united, potassium iodide, which breaks up this union and eliminates the metal, should be given. In acute or severe chronic cases it should be given with care, for it transforms the fixed tissue metal into free metal in the circulation. Nevertheless, *small doses of potassium iodide* (grs. iii to v) should be given, and some writers obtain even better effects from potassium bromide (grs. v to x).

Warm baths and pilocarpine (gr. ⅛) promote excretion by diaphoresis, and plenty of water stimulates the renal elimination. (*e*) *Iron* (not arsenic) should be given for anæmia. (*f*) For treatment of *neuritis,* see MULTIPLE NEURITIS.

(D). ARSENICAL POISONING.

Acute poisoning has been considered under pernicious anæmia.

Chronic Poisoning.—(*a*) *Criminal poisoning* is of great medico-egal importance, as in the unfortunate Maybrick case. (*b*) Poisoning by reason of occupation is uncommon. (*c*) *Therapeutic poisoning* s not common, but may occur in chorea, leukæmia, and pernicious anæmia. (*d*) *Most cases are accidental.* The dyes in garments, toys, glazed paper used in kindergartens, wrappers, artificial flowers, and wall paper are important sources of intoxications. The action of moulds (pencillium and mucor) may liberate volatile arsenical gases. Contaminated glucose used in beer caused the Manchester epidemic. Arsenic is absorbed by the lungs and alimentary tract, and is chiefly eliminated by the kidneys, which may degenerate. Putnam found arsenic in the urine of 30 per cent. of persons who showed no other ymptoms of arsenical poisoning but in this connection it may be emarked that arsenic is widely found in plants, sea water and springs. Smaller amounts are eliminated by the bowel, milk, and other secre-ions.

The *symptoms* are (*a*) *gastro-intestinal,* as dry throat, vomiting, purging, or colic; the stools may be like "rice water" and contain lecks of mucus; and (*b*) *respiratory,* as dry cough or chronic bronchi-is. (*c*) *Emaciation,* weakness, fever and anæmia may occur. (*d*) *Cutaneous* symptoms, as erythema, keratosis, pemphigus, herpes and brown pigmentation and (*e*) *nervous* manifestations, as weak memory, vertigo, headache and multiple neuritis may develop. The neuritis has the same general features as the saturnine form, save that sensory changes are more frequent, and the legs are more often affected than he arms; paralysis of the peronei and foot extensors causes the "steppage" gait. The small muscles are less often affected than in plumbic or alcoholic neuritis; paralysis, ataxia, sensory and trophic disturbances and the reaction of degeneration occur as in other neuri-ides. *Treatment* is that of lead poisoning or neuritis.

(E). FOOD POISONING.

Bromatotoxismus (Vaughan) exists in several forms:

I. Meat Poisoning (**Kreatoxismus**).—Sausages and head cheese are more dangerous than beef or mutton. Sausage poisoning (botulism or allantiasis) was known in Germany a century ago. Van Ermingen has isolated an anaërobic organism, the *Bacillus botulinus;* it contains spores, is flagellated and mobile and grows only on alkaline media at a low temperature (18 to 25° C); it is a saprophyte and causes symptoms by its toxins only, which are not destroyed by the gastric juice. Meat may seem to be normal to the taste and smell and yet contain ptomaines; their nature is still undetermined though Cobert considers them promatropin. Canned meat has often caused severe symptoms. In one instance botulism was caused by eating salad (Collatz). The symptoms of botulism appear in twelve to forty-eight hours, with nausea, vomiting and sometimes diarrhœa. Paralysis of the soft palate, larynx and œsophagus rapidly develop, and somewhat later paralysis of the bowel, mydriasis, ptosis, disordered vision, adynamia and weak heart action; the sensorium is clear and fever is unusual. The *mortality* ranges between 20 and 50 per cent.; death occurs in four to ten days and the autopsy shows parenchymatous degenerations and ecchymoses but especially degeneration of the ganglionic cells, as those of the vagus or oculomotorius; if the patient survives, convalescence is established only after months. In the Wellback cases, Ballard noted as early symptoms, headache, chilliness or rigors and pains in the chest or back.

The *symptoms* of other forms of meat poisoning are either those of *toxæmia* (the typhoid type) or *gastro-intestinal* inflammation (cholera type); the typhoidal symptoms, which appear after an incubation of four to six days, are fever, status typhosus, and even roseolæ; the choleriform symptoms, which develop in two to twelve hours, are vomiting, incessant purging, watery stools, clammy skin, collapse and cyanosis. Mild or rudimentary forms occur in most epidemics.

Treatment is unsatisfactory. *Early gastric lavage* and *purgation* are indicated; calomel followed by castor oil is the best aperient. Otherwise symptomatic medication as in cholera or typhoid is indicated; *champagne*, *strychnia*, hypodermatoclysis, and opium may be given. The resemblance of botulism to the diphtheria toxins has suggested the use of antitoxine; its effects in animals have been remarkable, but to the author's knowledge it has not been used in man.

II. Poisoning by Milk (Galactotoxismus) and *Cheese* (Tyrotoxismus) —The former produces symptoms analogous to those of the infantile affections of the bowel, as vomiting, bloody diarrhœa, fever, and collapse. In cheese poisoning Victor C. Vaughan has found tyrotoxicon and other more frequent toxins, which are probably albumoses. Similar ptomaines were found by him, by Novy and by Perkins in re-frozen ice-cream, and custard. In Norway where tyrotoxismus is most prevalent, the colon bacillus has been repeatedly found. Home-made cheese is more dangerous than the factory product. Various forms of toxæmia result; some are cholera-like, and others resemble belladonna poisoning. Treatment is that of kreastoxismus.

III. Poisoning by Fish (Ichthyotoxismus) and *Shell Fish* (Mytilotoxismus).—(*a*) Ichthyotoxismus, described by Comby (1827), may be caused by poisonous fish or poisonous glands in certain fish, as the roe or testicles, the muscular parts being harmless; the barben in

Europe, and the tetrodon in Asia are poisonous (*v.* BERI BERI). The poisoning due to putrefaction is more common. Paralytic symptoms are most frequent. (*b*) Mussel poisoning (mytilotoxismus) may cause dyspeptic symptoms in mild infections and cholera-like symptoms or symptoms of belladonna poisoning in severe forms. Rashes are common. Brieger isolated a mytilotoxin, which is found largely in the liver. The danger lies in placing mussel- or oyster-beds near river mouths, so that they feed on poisonous excreta. In restaurants, certain glands near the heads of lobsters, which are considered toxic, are carefully excised.

IV. Grain poisoning (Sitotoxismus).

1. *Ergotism,* which was described by Thuillier (1830) and carefully studied by Tüczek (1879), results from eating grain mixed with the ergot fungus (claviceps purpurea). Ergotized grain is found largely in bad harvests, resulting from wet springs and hot summers. Epidemics have occurred in Europe, and sporadic cases are not rare. *Acute ergotism,* with digestive symptoms, colic, cyanosis, weak heart and generally lethal outcome is less common than *chronic ergotism,* of which *two forms* exist; the first is the *convulsive* form, attended by paræsthesia, lightning pains, headache, delirium, melancholy, dementia, muscular relaxation and muscular spasms, either as generalized epileptiform attacks, or as local contractures (flexion of the arms and extension of the toes and feet); it is said to result from the alkaloid cornutin. The *second form* is the gangrenous, due to sphacelinic acid and marked by localized gangrene, usually in the phalanges, and less often in the tips of the ears and nose. Chronic ergotism is characterized by a number of *symptoms which suggest tabes,* as lightning pains, ataxia of station and movement, and lost knee jerks; and in five autopsies Tüczek and Siemens found degeneration with, later, sclerosis of the posterior columns; degeneration in the anterior cornua, and minute foci of softening or hemorrhage also occur. The clinical course is chronic, and death is frequent from convulsions or cachexia.

Treatment includes avoidance of diseased grain, and symptomatic therapy; opium should be given for convulsions, and nitroglycerine for gangrene.

2. *Pellagra* or maidism is a chronic nutritional and toxic affection, due to the use of maize which has been improperly stored, or is decayed, unripe, or infected with the aspergillus. An alkaloid (pellagazein) is thought to develop. Pellagra is endemic, chiefly in Italy, France, Spain, Hungary and Mexico. It occurs among poor farmers. There are *three stages*; the *first* consists of the *vernal erythema,* which is followed by exfoliation and sometimes by suppuration; dyspepsia and diarrhœa may develop; mild cases may recover, or may pass into the *second stage,* in which headache, pain in the back and muscular spasms occur, but the most characteristic symptom is the *ataxic paraplegia*; autopsies reveal a posterolateral sclerosis, sometimes with atrophy of the anterior horns, leptomeningitis, and variable brain findings. The *third stage* occurs after repeated attacks and is usually incurable;

cachexia, *epilepsy* and *dementia* develop; it is said that 10 to 35 per cent. of Italian insanity is due to pellagra.

In rarer acute cases, it may resemble typhoid in symptoms and pathology (status typhosus and swelling with ulceration of the intestinal lymph structures), or meningitis (foci of acute meningomyelitis). Other findings at autopsy are atrophy of the digestive mucosa, parenchymatous degeneration and pigmentation, the latter of which is thought to occur from adrenal inflammation. *Treatment* is prophylactic (proper storage of the grain); change of diet or of locality is indicated, and arsenic should be given internally.

3. *Lathyrism* (lupinosis) is produced by meal made from vetches, chiefly the *lathyris sativus* and *cicera*. Irving described the disease in India, and it has been also seen in Algiers and Italy. Like pellagra, it probably produces a lateral and possibly some posterior sclerosis of the cord, though no autopsies are reported. Clinically there is a *spastic paraplegia* with increase of reflexes and some paræsthesia, but without atrophy.

V. Potato Poisoning.—Solanin is contained in potatoes in small amounts (0.06 per cent.) but larger amounts (0.4 per cent.) occur in potatoes which lie for some time on the ground, or sprout in cellars. Fever, chills, general and cardiac weakness, alimentary irritation and jaundice may develop.

(F). SUNSTROKE.

Two forms are recognized, (1) sunstroke, and (2) heat exhaustion; the difference between them was first recognized by Dowler of New Orleans.

Sunstroke, also known as siriasis, insolation, *coup de soliel* and thermic fever, is due to exposure to the sun. A very frequent factor is alcoholism. There is thought to be a paralysis of the bulbar heat centres, leading to excessive production of heat and deficient elimination of it. The theory of infection has also been advanced.

Symptoms may develop very abruptly, and cause early, or even immediate death from asphyxia and paralysis of the heart; this type was long confused with apoplexy. In most cases there is an interval lasting from a few minutes to an hour, in which there are headache, vertigo, visual disturbances or nausea, and perhaps vomiting.

During the coma which develops rapidly, and in which the physician usually sees the patient, the following signs are observed: the face is first flushed, later rather cyanotic, and sometimes stained with petechiæ. The preliminary pupillary dilatation passes into myosis, and the skin has a peculiar odor. The pulse is rapid, usually over 100, and is bounding. The rectal temperature ranges from 107 to 112° and respiration is deep, labored and stertorous. Save for the laboring chest, the patient is usually quiet; in about 25 per cent. of the author's cases, they struggled during treatment by the cold bath, and in about 20 per cent. of the severe cases there were epileptiform convulsions.

Lewis and Packard (1901) in 92 cases found convulsions and unconsciousness in all severe cases; immobile pupils and absent knee jerks were common. Involuntary evacuations are usual, and the thin fæces have a peculiar odor. There is a leukocytosis in about 75 per cent. of the cases. Recovery is quite frequent; consciousness returns and the fever falls. Despite treatment, the Cheyne-Stokes breathing may develop, the pulse may grow smaller and more rapid, and death may follow, usually in a little more than a day. The autopsy shows early rigor mortis, fluidity of the blood, great venous hyperæmia, particularly in the brain and lungs, and dilatation of the right and contraction of the left ventricle.

After sunstroke, the patient becomes extremely susceptible to hot weather. Epilepsy, multiple neuritis and mental symptoms are among its after-effects. The author saw one case with a temperature of 109° in which the fever fell with hydrotherapy but rose to 104°; in a few days, the complication was recognized to be typhoid.

The *diagnosis* is easily made. Practically, there is but one possible cause of confusion, which is pontine hemorrhage, but the latter never develops nearly as high a temperature, and death is almost instantaneous; should the patient survive for some time, the paralysis, which is usually of the crossed type, is obvious.

Heat exhaustion may result from exposure to the sun or to high temperature with humidity; the cases which the author has seen came largely from laundries, basement stores, or engine rooms; heat exhaustion occurs also among stokers on steamers. The symptoms are those of a centric vasomotor paralysis. The skin is cool and livid, rather than red, and the rectal temperature is often 3 or 4° subnormal; the pulse is rapid and weak and the breathing is rapid but not labored. In short, the symptoms of collapse are present. Restlessness, anxiety and sometimes delirium are noted, though the sensorium is practically normal in most cases.

Treatment.—In *sunstroke* the chief indication is reduction of the retained heat. It should be relieved by immersing the patient in a bath which is gradually cooled by large pieces of ice. Shock is averted by this means; cerebral congestion is lessened by placing a block of ice under the neck, an ice-bag on the vertex, and an ice-bladder over the anterior neck; active friction of the body and limbs with the open hands brings to the surface the blood, which is cooled by the bath and the rubbing with pieces of ice. With this treatment, few more than one-third of the cases should die. The patient should be taken from the bath when his rectal temperature is 102°, for the fall usually continues after cessation of treatment. Laxatives are generally unnecessary, for the bowels move freely. Heart stimulants should be given *pro re nata*. For convulsions, a little chloroform by inhalation, and a hypodermic of morphine are indicated. Asphyxia and distention of the right heart are relieved by phlebotomy.

In *heat prostration*, the treatment is diametrically the opposite: a warm bath or hot bricks for subnormal registration, diffusive cardiants as ammonia, camphor and strychnine, saline solutions under the skin, and applications of mustard are indicated; cold and venesection are obviously contra-indicated.

SECTION XI.

DISEASES DUE TO ANIMAL PARASITES.

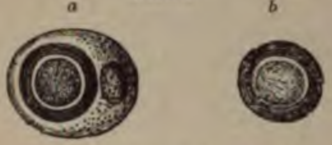
(A). DISEASES CAUSED BY CESTODES.

Tapeworms were known to the Egyptians, to the Hindus and probably to the Jews. They may cause symptoms by the presence of the mature parasite in the bowels or of the larval form in the viscera.

Tænia Solium.

I. The Tænia solium or pork tapeworm exists as the cysticercus cellulosæ in hogs, and rarely in dogs, rats, or deer, as "measles" (measle or bladder worms). Cysticerci (the larvæ) are found in the intermuscular tissue, particularly of the under surface of the tongue, in the masseters, shoulders, neck and diaphragm. They are easily seen with the naked eye and very easily with a low-power lens, as small opaque white bodies. They are much more frequent in Asiatic and German than in American pork. Man is infected by eating raw or partly cooked pork; the cysticerci develop in the upper ileum into the tænia ("band or ribbon-like") solium (referring to its usual solitary appearance, though 2 to 3, and even 20 to 41, have been found). Its head (*scolex*) is round, often gray or black in its upper part, is smaller than a pin head, and is attached to the upper ileum; it has 4 sucking disks and 20 to 30 hooklets, arranged in an anterior more numerous row, and a posterior row, whence the name "armed" tapeworm; it has no real mouth. See figs. 81, 82, 83, 84. Its neck is thin and not jointed, to which the segments ("*proglottides*") are attached. These number 800 or more; the segments measure 1 cm. in length by 7 mm. in width; those near the neck are narrower and shorter. About a yard from the neck they become squarer, and lower down they are smaller again. Each proglottis is hermaphroditic, containing male and female reproductive elements. The uterus is central and vertical, with 8 to 10 lateral branches; only a few of them mature, chiefly those in the lower links; the ova (0.036 x 0.03 mm.) are very numerous in each maturing segment and each one shows a shell and an embryo armed with six hooklets. The tænia reaches its full length of two to four yards in three to four months, when the lower segments are detached and voided; if the worm should break and a yard or so appear in the fæces, the segments will not reappear in the fæces until three or four months later. An ovum ingested by a hog or man, loses its shell in the stomach and the freed embryo passes into the tissues, where, encysted, it becomes again the cysticercus cellulosæ (or larva).

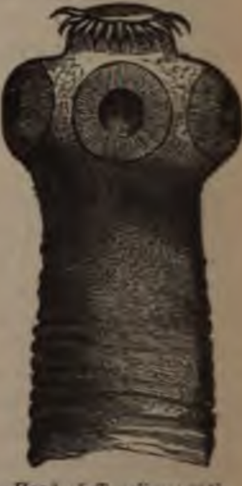
Fig. 81.



Ova of *T. solium: a*, with yolk; *b*, without yolk, as in mature segments. The hard brown shell is indicated. (Leuckart.)

Symptoms of the Tænia Solium in the Human Intestine.—Many, possibly most, cases present no symptoms; they may first develop after passing segments are found in neurotic subjects. In other cases dyspeptic disturbances arise, as anorexia (or less often increased or ravenous appetite), nausea, abdominal pains, constipation or diarrhœa. Stiles, in an experimental infection on himself, thought that he could feel the parasite move. Vomiting of segments is rare, though several feet of tapeworm have been vomited; in these instances the ova (embryos) may enter the tissues as cysticerci; this is a rare condition in which one individual suffers from both the intestinal and the larval stages. Anæmia is rather uncommon. In children reflex convulsions are possible. The parasite may live for even 10 to 15 years. The *diagnosis* is determined by the segments and ova, and the *prognosis* is practically always good.

Fig. 82.

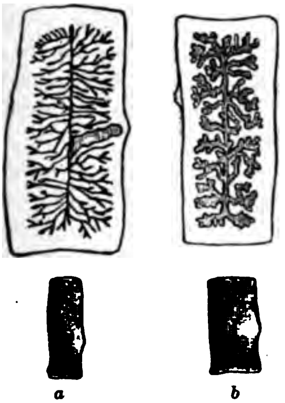


Head of *T. solium*: ×45.

Treatment.—As to *prophylaxis;* segments of the tapeworm should be burned; meat inspection should be more general than it is, though "interstate" meat is well inspected; pork should always be thoroughly cooked.

To cure the disease, a light and chiefly fluid diet should be given for two days, and then food should be withheld for 12 to 18 hours; this often causes the parasite to release its hold on the mucous membrane; some writers advise that a meal of onions and herring be given just before drugs are administered. Of these *pomegranate* ranks first; a decoction is made of 3 ounces of the root with 8 ounces of water; this is reduced to 4 ounces by evaporation; it is taken in divided doses within 3 to 4 hours; its sole active principle is the expensive pelletierine, which is given in doses of 3, 5 or 10 grains, and is followed by a purge in an hour. *Male fern* (felix mas) is almost equally effective; it is given as the oleoresina aspidii, or extr. felicis maris æther., ʒiss to ijss, may be given in syrup; both are followed by a saline purge in three to four hours. Castor oil is never used, because it promotes absorption of the male fern, which causes toxic symptoms as icterus, or amaurosis; Sidler collected 78 such cases of poisoning with 15 per cent. mortality and lasting blindness in 25 per cent.; Leichtenstern advises (*a*) that more than 10 gm. (ʒiiss) of the ethereal extract should never be given to adults (ʒi or less to children); (*b*) that it never be given on an empty stomach and (*c*) that it never be given two days in succession. The drug decomposes easily, whence reliable preparations must be obtained. Combinations may be made by giving with or after the decoction of pomegranate, spts. chloroformi (♏ xv) for four doses, or croton oil (♏ ii) for one dose, but pomegranate and male fern produce the best results. Search should be made for the head of the parasite, without removal of which treatment is unsuccessful. If it is not found, another trial should be made after a number of days.

Fig. 83.



Proglottides of (*a*) *Tænia saginata* and (*b*) *T. solium*: natural size, and enlarged three times to show arrangement of uterus. (After Leuckart.)

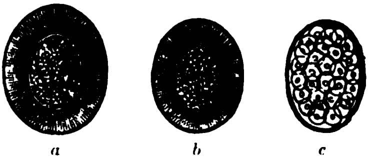
℞

Oleoresinæ aspidii	
Tr. vanillæ	āā gtt. xlv.
Acaciæ	
Sacchari albi	āā ʒi.
Aquæ	℥i.

M. et ft. emulsio.
S.—Take in one dose.

Symptoms of the cysticercus cellulosæ. These result when ova of the tænia solium are taken into the stomach; ova may reach the stomach from the intestines by antiperistaltic movements during vomiting, or indirectly by handling worms voided from the bowels. Cysticerci in the hog produce few symptoms. In man, symptoms depend largely on the number and localization of the larvæ. (*a*) In *the muscles and skin*, a few cysti-

Fig. 84.

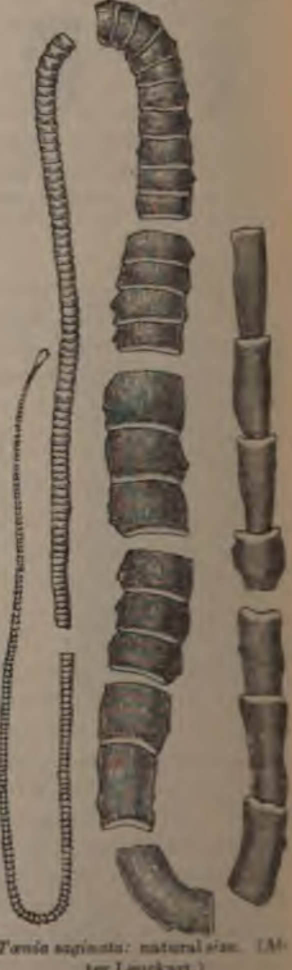


Eggs of (*a*) *T. saginata*; (*b*) *T. solium*; (*c*) *Bothriocephalus latus*: ×300. (After Eichhorst.)

cerci cause no symptoms; numerous cysticerci cause pain, tenderness and difficult movement. Diagnosis is possible only on excision of the subcutaneous nodules. In Stiles' collection of 155 cases, the localization was muscular in 20 per cent. and cutaneous in 3 per cent. (*b*) If they are in the *brain* (in 75 per cent. of Stiles' series) symptoms depend on their location; they are often absent; if the location is in the cortex, Jacksonian epilepsy is frequent, and if in the medulla, diabetes may develop. (*c*) There may be *other localizations*. They may be found clinically in the eye (v. Graefe), and a few other localizations are found at autopsy, as pulmonary, renal, or hepatic. There is no *treatment* except surgery.

II. Tænia Saginata (Mediocanellata).

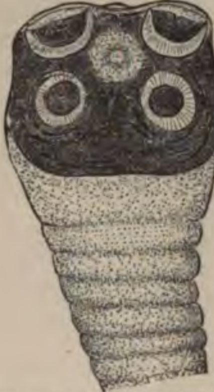
Fig. 86.



Tænia saginata: natural size. (After Leuckart.)

This form is most frequent in America, but is becoming more common in Europe. It is the "fat tapeworm," the "unarmed," or beef tapeworm. Its *cysticercus* is found in masseters of beef, nearly seven times as frequently as in other muscles, or in the heart, or brain. They are not so readily seen with the naked eye as the cysticerci in pork. Ingestion of rare beef containing cysticerci produces the tænia saginata in man.

Fig. 85.



Head of *Tænia saginata*. (Eichhorst.)

The intestinal parasite differs from the T. solium in the following points: (a) its *head* is larger, measuring 2 mm. and is square. It has no hooklets but four sucking disks, which are larger, more forward and are surrounded by pigment. (See figs. 83, 84, 85 and 86.) (*b*) Its neck is much shorter. (*c*) Its *proglottides* are longer (16 to 22 mm.) and broader (5 to 10 mm.). (*d*) The *uterus* contains 20 to 30 branches, which are less dendritic than pronged or forked. (*e*) The *ova* are rather longer. (*f*) The *parasite* is larger and longer, measuring 5 to 7 meters or yards. Its symptoms and treatment are those of the tænia solium. Ingestion of the ova or segments produces the cysticercus mediocanellata, which is common in beef but extremely rare in man.

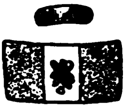
III. Other Tæniæ Occurring In Man.

The *Bothriocephalus latus*, or the Russian tapeworm is found chiefly in Russia, Poland, Norway, Sweden, Switzerland and Japan. Its cysticerci are found in the muscles and peritoneum of the pike, trout, salmon, and perch and in man's intestines develop into the bothriocephalus. Its head is flat or ovoid, possesses no hooklets and attaches itself to the intestines by two lateral grooves (suckers). Its neck is long and its segments broad, square, and may even number 4,000. It is long, measuring 8 to 10 yards. It occurs in America in imported cases. It may produce a profound anæmia which in severe cases closely resembles the pernicious type in its general symptoms and blood findings. (See figs. 84, 87.)

The *Tænia elliptica* or cucumerina (Dipylidium caninum) is a form, the larvæ of which develop in the lice of dogs, and the adult form exists in the intestines of dogs, sometimes in those of cats, and rarely in man. (See fig. 88.)

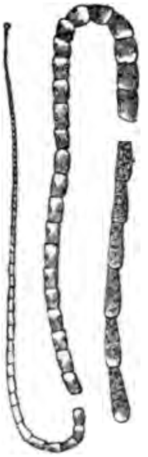
The *Tænia nana* (Hymenolepsis nana) is the dwarf tapeworm. It measures but ⅓ to 2 inches in length. Its cysticercus

FIG. 87.



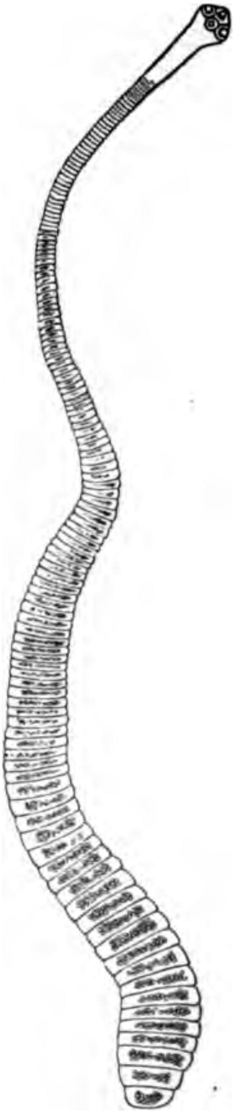
Proglottis of *Bothriocephalus latus*: natural size, and enlarged three times. (After Eichhorst.)

FIG. 88.



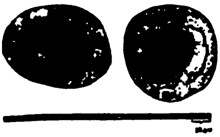
Tænia cucumerina: natural size. (After Leukart.)

FIG. 89.



Hymenolepsis nana: X 12 (After Leuckart.)

FIG. 90.



Ova of *Hymenolepsis nana*. (Smith.)

stage is in the intestinal wall of the rat and its adult stage in the rat's intestine. Stiles has recently decided that it is much more frequent in man than previous accounts would indicate. Its head contains four suckers and one row of hooklets. It may occur singly or in myriads. Aspidium alone is of therapeutic benefit. (See Figs. 89 and 90.)

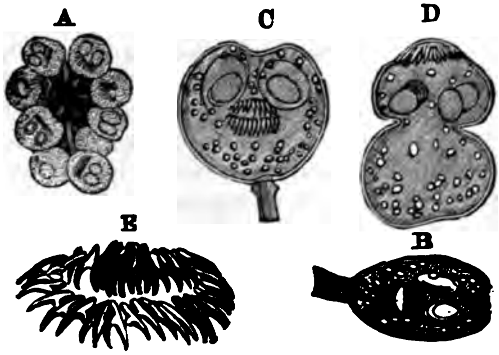
Tænia Echinococcus.—Echinococcus Disease.

We may first consider the *adult parasite as inhabiting the intestine of the dog* chiefly, and rarely of the wolf, fox or jackal. The head is small, and is provided with four suckers, and from 30 to 50 hooklets arranged in a double row. The segments number only three or four, and only the end one matures; it measures but 0.6 by 2 mm. but contains even 5,000 ova. The worm is small, measuring but four to five mm. in length, is very delicate and white, so that it readily escapes detection. When the ovum reaches the stomach of another animal, the hog and ox chiefly, and less often the horse, sheep or man, it loses its shell and migrates to the various organs (*v.* SYMPTOMS). The *geography* of echinococcus disease and mode of infection is as follows: In Iceland, where dogs and men live together, the disease in man is common, every seventh person becoming infected. In Australia both men and sheep are often infected. In Europe the malady is much more common than in America, where the reported cases in 1901 numbered 241 (Lyon); most of these were foreigners. The Icelanders brought the disease to Manitoba in 1874.

Infection occurs by licking of the hands by the dog, the tongue and anus of which often come in contact. Infection by contaminated drinking water or vegetables is less common.

General Symptoms and Pathology.—The small embryo, freed of its shell, penetrates the intestinal wall and wanders in various directions, into the muscles or peritoneum or into the radicles of the portal vein or cava, so that almost any tissue or organ may be infected, and the embryo merely stops when it reaches vessels too small for it. Once lodged, its hooklets disappear and a cyst develops, which is small at first but gradually reaches large or enormous proportions; there are records of cysts containing 30 and 70 pints of fluid. Its layers consist of an inner endocyst which is granular, and an outer finely lamellated structureless capsule; a fibrous layer develops outside from reaction of the tissues to the foreign body. After a while a number of "daughter" cysts arise from the endocyst; these are first "buds" and then miniatures of the original mother cyst; from these daughter cysts, which number about a dozen, "granddaughter" cysts arise by an identical process. Allen in one case found 8,000 daughter cysts. Finally from the endocyst of the maternal and daughter cysts there develop "brood capsules," in which small buds arise, which gradually become scolices (the heads of which will be intestinal tæniæ in the animal which ingests them). Some cysts do not develop scolices and are therefore called sterile. In animals the daughter cysts may grow outward (exogenous cysts). The well-grown cyst contains fluid (*v.* LIVER AND LUNG ECHINOCOCCUS); it is clear and neutral, has a specific gravity of 1005–10–15 and contains succinic acid, inosite (possibly sugar) and much sodium chloride but no albumin, unless the parasite dies and cystic inflammation develops. Then the fluid may become cloudy, buttery, gelatinous, purulent, brownish or grumous; the cysts disappear as well as the membrane, though the hooklets of the scolices remain a long time. When the parasites are alive a toxin seems to be present, for collapse often develops when the cyst ruptures or is punctured (see LUNG ECHINOCOCCUS).

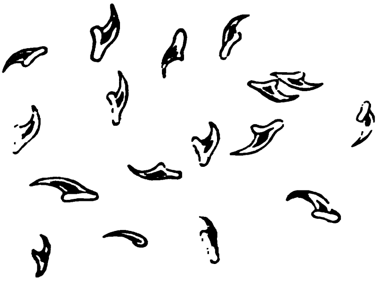
FIG. 91.



Human echinococci. (From Finlayson, after Davaine.)

A, a group of echinococci, still adhering to the germinal membrane by their pedicles. ×40.
B, an echinococus with head invaginated in the body. ×107.
C, the same compressed, showing the suckers and hooks of the retracted head.
D, echinococcus with head protruded.
E, Crown of hooks, showing the two circles. ×350.

FIG. 92.



Hooks from Tænia echinococcus. ×350. (Cort.)

The parasite is said to live for years, in one instance it survived for 20 years; after a variable time, unless suppuration intervenes, the cysts become cheesy and perhaps calcified or ossified.

The parasite may rupture into the serous sacs, bloodvessels (causing embolism), into the air passages, urinary tracts or externally (see ECHINOCOCCUS OF THE LUNG, LIVER AND KIDNEY).

Visceral Localization.—In 1,912 cases (the total reported by Davaine, Finsen, Mosler, Neisser and Cobbold) the liver was involved in 51 per cent., the genito-urinary organs in 10 per cent., the intestinal canal in 9 per cent., the lungs or pleura in 9 per cent., the brain or cord in 7 per cent., the bones in 3 per cent., the heart and vessels in 3 per cent., and other organs in 8 per cent. Vegas and Cranwell (1902) reported 970 Argentine cases of which 64 per cent. occurred in the liver and 7 per cent. in the lungs. In statistics from single observers, percentage of cases with localization in the liver often runs higher (69 to 72 per cent). Thomas, in 809 Australian cases of single hydatid cyst, found the lungs involved in 16.5 per cent. Hooklets have been obtained by lumbar puncture (Jacobs). There are reported 142 cases of renal echinococcus, 78 cases with localization in the bones and 23 in the pelvic bones (Baradulin, 1906). Grulee in 1905 found 55 cases with cardiac or pericardial localization. Echinococcus cyst of the thyroid has been reported 23 times (Ehrhart, 1905).

There is an unusual form of echinococcus, *the multilocular form*, probably due to a separate worm, the clinical features of which are described under hydatid disease of the liver to which it is almost exclusively confined. Virchow first described it in 1856. It occurs in Bavaria, Würtemburg, Switzerland, Austria, Austrian Tyrol and Russia. Few imported cases have occurred in America. It is unknown where the ordinary form is most common. It is rare, few more than 100 cases being recorded. Sixty per cent. occurs in males between 20 and 50 years of age. The ova are spheroidal and measure 100 to 170 micro-millimeters. The great difference from the ordinary form is the outward (exognous) growth of the cysts. The irregular and diffuse cystic proliferation is an unfavorable feature. The cysts contain a gelatinous material (which sometimes causes confusion with colloid cancer), surrounded by intersecting fibrous strands, which gives the liver a porous appearance, like a sponge or cheese with air cavities. In some vesicles there is cheesy material, with some resemblance to the granulomata. The vesicles sometimes suppurate.

Echinococcus cysts which cause symptoms belong largely to surgery, though as described elsewhere, spontaneous recovery is not infrequent.

(B). DISEASES CAUSED BY NEMATODES.

I. The Ascaris Lumbricoides.

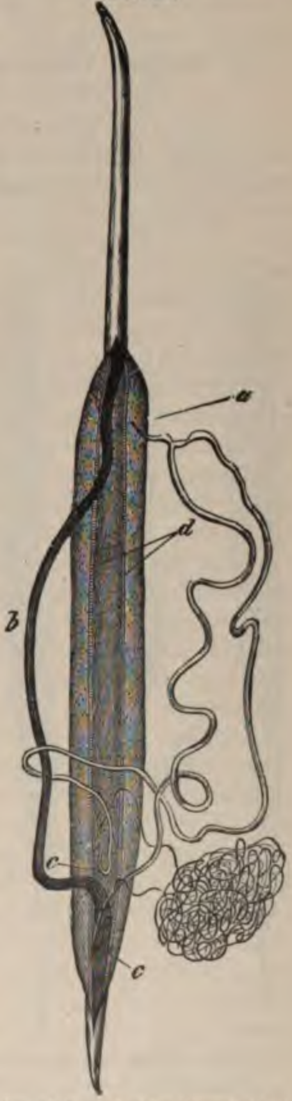
This is the most common intestinal parasite in man. It occurs oftenest in children and with relative frequency in the insane. Unlike the cestodes, no intermediate host is required. The female measures 7 to 12 and the male 4 to 8 inches in length; it is smooth, pointed at both ends and has transverse rings and four longitudinal ridges, a white one dorsally and ventrally, and two brownish ones laterally. (See fig. 93.) The ova measure 0.075 x 0. 058 mm., are oval and reddish and have a thick capsule. It is said that 60,000,000 eggs may develop in a single female. It lives in the upper small intestine; its mode of infection is obscure. Though they usually occur singly or in pairs, they may exceptionally develop in such numbers that by intertwining, they may obstruct the intestine. The ascaris may rarely rupture through an intestinal ulcer or perforate the sound gut. It has passed through a perforated appendix. With or without vomiting, ascarides may pass from the stomach into the œsophagus, nose, bronchi, lungs or middle ear, causing asphyxia, gangrene of the lung, etc. They may migrate into the common or hepatic duct.

Symptoms are not usually pronounced, though anorexia, salivation, offensive breath, constipation, colic or diarrhœa occur in some cases. Malnutrition is sometimes marked. The parasite secretes an irritating substance which is often noted to emanate from it. It is suggested that this secretion, if absorbed, may cause constitutional symptoms (Peiper).

Such manifestations as irritability, grinding the teeth, muscular twitchings and convulsions may develop in nervous children; they are said even to resemble meningitis. The author always doubted the existence of the *typholumbricosis* of Chaffard, Marie and Tauchon, until in one adult case, in which there were fever and nervous toxæmia, with otherwise negative findings, instantaneous subsidence of all symptoms occurred when the parasites were removed; this strongly suggested a relation between the fever and other symptoms and the lumbricosis.

Treatment.—After a fast of one-half to one day, *santonin* should be given in doses of gr. ss–i, for a child, and gr. ii, iii, or v, for an adult; it should be followed by a saline cathartic; if unsuccessful it may be repeated on two or three successive days, though toxic symptoms sometimes develop, as xanthopsia (yellow vision), urticaria (which

Fig. 93.



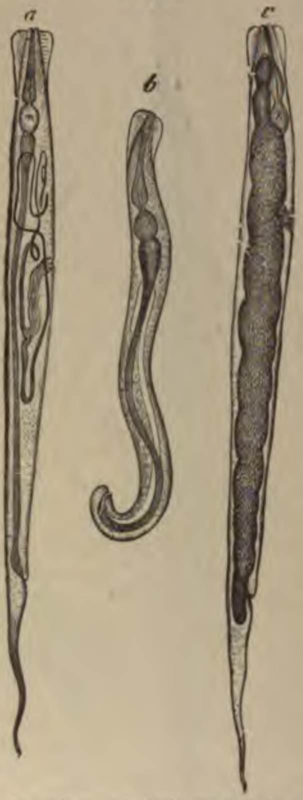
Ascaris lumbricoides, dissected and walls thrown back: *a*, genital orifice; *b*, intestine, *c*, oviducts; *d*, longitudinal band; *e*, ovaries. (Heller.)

may result from the parasite itself), vomiting, delirium or convulsions. The *extr. spigeliæ fl.* (ʒi) may be given with or without the santonin. These remedies do not kill the parasite, but merely render it uncomfortable.

II. Oxyuris Vermicularis (Seat Worm, Pin or Thread Worm).

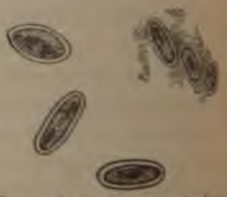
Children are more often affected than adults, and women more often than men. They are relatively frequent in the insane. The female in length measures 10 and the male 4 mm. (See Figs. 94, 95 and 96.)

Fig. 94.



Oxyuris vermicularis, magnified: *a*, young female; *b*, male; *c*, mature female, full of eggs. (Payne.)

Fig. 95.



Eggs of Oxyuris vermicularis ×275. (After Eichhorst.)

Fig. 96.

Oxyuris vermicularis: natural size. (Eichhorst.)

Ingested ova develop in the small intestine, where the worms mature and cohabit; afterward they exist chiefly in the lower colon and rectum. The eggs remain in the mother until the worm is expelled, though she occasionally "aborts" in the bowel. Infection occurs in those who are the most uncleanly, as in children and the insane. Infection may occur from water and green vegetables, and reinfection is not uncommon from scratching the anus during sleep and thus crushing the parasite; Eichhorst frequently found ova in the minute fæcal particles about the anus and Zenker demonstrated repeatedly their presence under the finger nails. As the parasites migrate at night, the symptoms are greatest at that time; they may be found on the bed sheet. The anal itching and rectal irritation are often excessive; periproctal suppuration may be excited. If any vaginal discharge moistens the perineum (and only then), they can wander into the vagina, causing irritation, sexual stimulation or masturbation. Disturbance of the sleep and appetite, nervous symptoms and anæmia may result. Diarrhœa is due to large numbers of parasites.

Treatment.—Santonin may be given in obstinate cases, but local measures are usually sufficient, as high enemata containing small proportions of carbolic acid, quassia, and turpentine. They should be repeated daily for two weeks. Itching is palliated by application of ½ per cent. carbolic salve to or within the anus.

III. Trichina (Trichinella) Spiralis; Trichinosis.

Though Tiedemann (1822), Hilton (1832) and Paget (1835) saw the parasite, Owen (1835) first fully described it. Leidy saw it in the hog in 1847. Zenker (1855–60) first described its pathological and clinical bearings; he found 4 cases in 136 autopsies.

Swine are infected in a manner which is not wholly clear, but infection most probably results from eating offal. The disease is rare in some countries, as France, and more common in others, as Germany. The hog is more frequently diseased in America than in Germany (a ratio of 1 to 17 [or 48] in the former and but 1 to 18,000 in the latter). From the clinical and etiological standpoint, the (*a*) *muscular or larval stage in hogs* should be considered first. The small encapsulated, "hair-like" (trichina) worm lies in the abdominal, diaphragmatic, psoas, laryngeal and other muscles. They live for a long while, even twenty to twenty-five years, without apparently causing any symptoms; calcification rarely occurs in swine. *Man is infected* by eating smoked, "cured" or insufficiently cooked pork; animals may be accidentally or experimentally inoculated, especially the rat, guinea-pig, rabbit, less often the cat, and rarely the dog. In much of the literature relating to its alleged presence in fish or worms, the trichina has been confused with other parasites. (*b*) The *adult or intestinal stage in man* is the next phase. When it is eaten by man, the small capsules (larval form in swine) are digested and the worms liberated; in the small intestine they mature (the females measure 3 to 4 mm., and the males are half as long) and the females are fecundated in two to three days. The female trichinæ produce one to two thousand embryos at once or possibly in succession, which stage requires another week (from fecundation of the female to birth of the embryos). At the end of the second week the embryos commence to migrate; they enter the lymph vessels and then the veins, by which they reach the voluntary muscles; some writers, as Graham, maintain that there is a primary direct entrance into the venous system. They lodge between the muscle fibers, enter them and attain (*c*) *the mature larval form*, which is identical with the larval stage (*a*) in hogs, and differing only in that man is their host. They measure 0.5 to 1 mm. in length. As a result of tissue reaction against the foreign bodies, a capsule develops in about six weeks, which surrounds one or more parasites. The capsules are at first transparent, but grow more opaque and, after months, calcify and later the trichinæ calcify also.

Frequency.—Though trichinæ are apparently about 40 times as frequent in American as in German swine, the disease is more common in North Germany where raw sausage, Westphalian ham and smoked ham are freely eaten. Fränkel denies that German trichinosis results from American pork. Williams of Buffalo, in 505 autopsies, found trichinosis in 5 per cent. Müller of Dresden found it in 0.98 per cent. of 1,939 postmortem examinations. Other statistics usually show lower figures than Müller's, and like the above, are taken from accidental postmortem findings. The disease occurs sporadically or epidemically. Perhaps a thousand cases have occurred in America. In Germany the epidemic at Hedersleben numbered 337, and that at Emersleben 250 cases.

Symptoms may be absent or slight, particularly in sporadic cases, and with moderately severe infection, as evidenced by accidental autopsy finding of encapsulated parasites.

In clear cases the symptoms are divisible into *two stages*; (*a*) the preliminary or *gastro-intestinal* stage, which begins in two to three days after eating the diseased meat; the symptoms are not constant either in sporadic or severe epidemic cases; they are anorexia, nausea, vomiting, colic and sometimes diarrhœa, which may resemble the evacuations of cholera. (*b*) The *invasion symptoms* begin in one or two weeks, and correspond to the parasitic migration. (i) *Fever* is usual, but its onset seldom begins with rigors. It results from the parasites and probably from some toxin which they elaborate; it may rise to 102, 104 or 106°, and its course is remittent and intermittent. Symptoms which are attendant on any fever may be present, as headache, febrile urine, or quickened pulse. In severe cases, as in Zenker's, typhoid may be suggested by delirium, epistaxis, dry tongue, status typhosus, Ehrlich's diazo reaction of the urine (in 80 per cent.), bronchitis, albuminuria and kindred toxæmic symptoms. In two cases typhoid and trichinosis co-existed (McCrae). (ii) *Acute diffuse myositis* is always most suggestive. Its intensity varies from mild forms of "muscular rheumatism" to the most severe and characteristic involvement; there is pain, tenderness, swelling, flexion to relieve muscular tension, and œdema in the face and other locations. The worms most curiously select only the striated muscles, so that the œdema which is observed in the abdomen and thighs, for example, never invades the scrotum or vulva. The eyelids are often affected greatly and early. Invasion of the eye muscles causes pain on moving the eyes; pain during mastication, phonation and deglutition mark penetration of the masseteric, laryngeal and pharyngeal muscles by the parasite; dyspnœa results from involvement of the diaphragm and intercostal muscles. The tendon reflexes may disappear. (*c*) *The blood* presents two striking features: first, the *leukocytosis* (15,000 to 30,000) and second, the eosinophilia (T. R. Brown); the eosinophiles normally constitute 0.3 to 4 per cent. of the leukocytes; their number rises as high as 50 or 68 per cent. and though they increase in the other parasitic diseases, this rise is most peculiar if not absolutely pathognomonic. The eosinophilia is greatest at the time when the trichinæ enter the muscles. (See Plate XIV, Fig. 5.) (*d*) *Profuse sweats,* polyuria, paræsthesia, urticaria and anæmia are inconstant manifestations.

Diagnosis.—This is based on (*a*) the possibility of infection, as by eating poorly cooked pork, (*b*) the discovery of trichinæ in the uneaten portions of suspected meat, (*c*) the detection by removal of small slivers of the biceps or pectorals for microscopic examination, especially the parts near the muscular insertion, (*d*) the myositis with œdema or muscular rheumatism, (*e*) signs of particular muscular insufficiency, as of the ocular or diaphragmatic muscles and (*f*) leukocytosis with eosinophilia.

Prognosis.—This depends on the number of parasites ingested. Choleraic and typhoidal symptoms, dyspnœa and pneumonia are most ominous. In moderately severe early diarrhœa it is favorable. Children die less often than adults. Convalescence is prompt or tardy as the infection is light or severe. According to Stiles, the German mortality averages 5.6 per cent. In the epidemic at Weimar there were no fatalities in 108 cases. In the epidemic at Hedersleben 30 per cent. died. Instances have occurred when as many as 70 per cent. of those who were attacked have died.

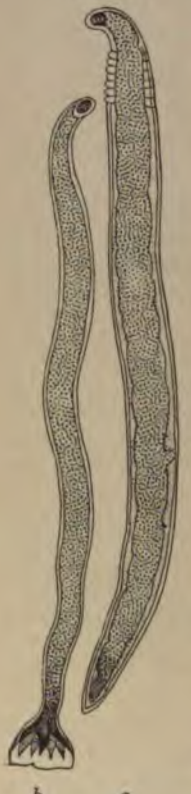
Treatment.—(*a*) *Prevention* comprises the feeding of hogs with clean food, government inspection of meat, and thorough cooking. (*b*) In the *preliminary stage,* active and repeated purgation should be given, for moderate and early spontaneous diarrhœa is prognostically favorable; calomel should be followed by salts and castor oil; male fern, santonin (*v. s.*), thymol gr. v, t. i. d., benzene ♏ ii to v, t. i. d. and oil of turpentine ♏ v to x are recommended; glycerine is now regarded as useless. (*c*) In the stage of *muscular invasion,* the fever is treated as in other infections, the myositis by local applications of ice and by narcotics, and the general nutrition by food and stimulants.

IV. Ankylostoma (Uncinaria).

The uncinaria duodenalis (ankylostoma duodenale) exists in two forms, that of the old and that of the new world. Ankylostomiasis was first well described by Piso (1648) in Brazil, and the parasite was recognized in Milan by Dubini (1838). It occurs chiefly in the tropics and sub-tropics; according to Thornton, it is the most dangerous of all tropical diseases. In Egyptian chlorosis it was found by Griesinger, and is said to be present in nearly every autopsy made there. It is epidemic in Italy ("brick-layers'" anæmia). In India it is found in even 80 per cent. of the healthy coolies and in 300 autopsies in Assam it was present 299 times. Thirty per cent. of Porto Rican deaths result from uncinariasis, and Ashford and King state that 90 per cent. of the rural and 50 per cent. of the urban population have the disease; during six months in 1905, 18,865 cases were treated in Porto Rico of which 33 per cent. died. It prevails in our southern states. Capps in 1903 reported one case, and collected 50 other American cases. More than twenty years ago, Belfield recognized the parasite in cats.

In the Westphalian mines, Tenholt himself treated over 4,000 cases. It was the cause of the "tunnel anæmia," which was observed in building the St. Gothard tunnel. It is disseminated by Italian and Polish workmen. Infection occurs from ingestion of the larvæ and not of the ova. Infection comes from the food, drinking water and earth which are infected by dejections from persons suffering with the disease. Loos has proven that it may enter by the skin, producing the "ground itch" which is common in the Southern States. The exact role of the uncinaria in clay eaters is not yet determined; the parasite certainly exists in some cases, though it is claimed that the dirt eaters instinctively eat clay to remove the parasite. The male is 8 to 10 and the female 10 to 18 mm. in length; the mouth has hooks by which it adheres to the mucosa of the duodenum. (See figs. 97, 98, and 99.) The ova measure 30 to 60 micromillimeters; they are oval, are covered with a translucent shell and differ from oxyuris ova which are flat. They show 1 to 6 yolk segments and may number over four millions. The larvæ develop in moist earth, whence they infect the water or the fingers or enter through the skin.

FIG. 97.

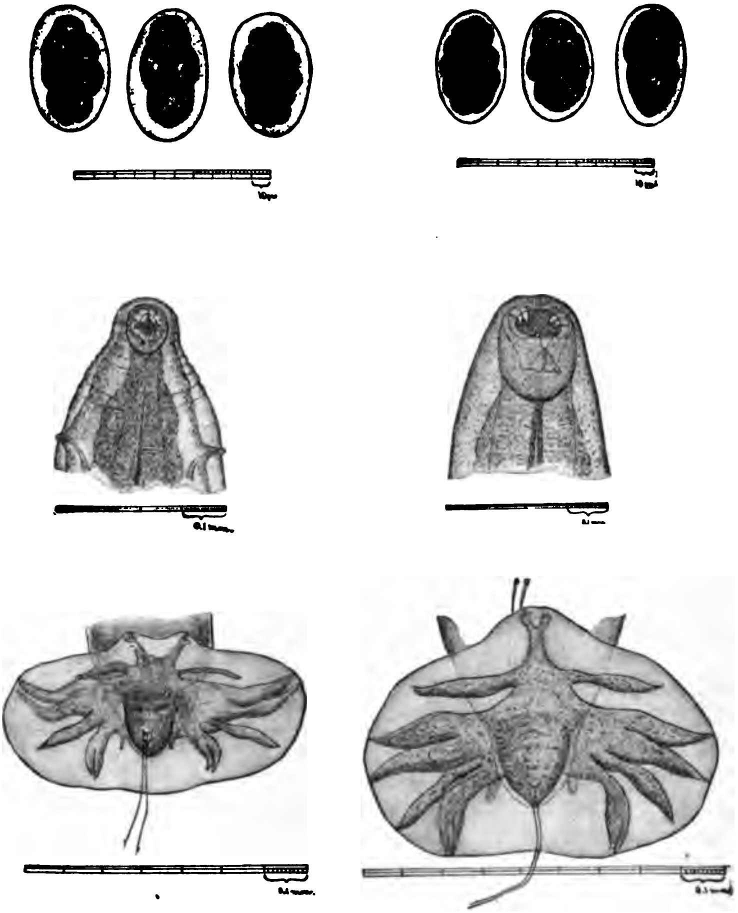


Uncinaria duodenalis, magnified: a, female; b, male. (Bristow.)

Symptoms.—Symptoms may be absent, or of a mildly dyspeptic character. In pronounced infections (*a*) an *anæmia* develops which is first of the secondary type and later of the progressive pernicious type; anæmia results not only from sucking of blood by the parasites, but from hæmolysis due to some toxin which they elaborate (in their salivary glands); normoblasts are much more common than megaloblasts, poikilocytosis is less marked than in the usual pernicious type, and the eosinophiles are increased, and sometimes reach 30 per cent. of the total number of leukocytes. (*b*) *Gastro-intestinal symptoms* are usual, as ink-like patches on the tongue, diarrhœa and colic; in Herrick's Panama case (reported by Capps) which the author saw in the County Hospital, there was great nausea, pain and exquisite abdominal tenderness; the stools contained blood, eosinophile cells, Charcot-Leyden crystals and ova. Stiles suggests a ready test for blood, by which parts of the stool are placed upon blotting paper; after an hour or so a rusty stain develops about the stool. Gastro-intestinal catarrh and small ecchymoses are found at autopsy. (*c*) *Nutrition suffers*; as in pernicious anæmia, œdema develops. Clay eaters are emaciated and apathetic; debility is frequent. In young children puberty is retarded;

development is poor and the abdomen is greatly distended. Cardiac atrophy and dilatation are common. The prognosis is grave, though less serious in the New World than in the Old World type.

FIG. 98.



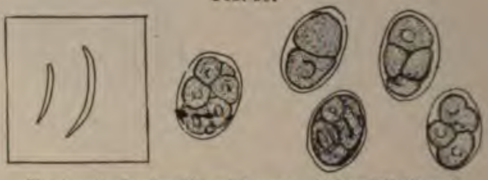
Above; to the left, three ova of *Uncinaria Americana* in different stages of segmentation, from $66\,\mu$ to $68\,\mu$ in long diameter; to the right, three ova of *Uncinaria Americana* in varying stages of segmentation, long diameter from $58\,\mu$ to $62\,\mu$.

Middle: to the left, the head of *Uncinaria Americana* (dorsal view), exhibiting the smaller size of the head and more tapering anterior extremity of the worm in comparison with *U. duodenalis*, and its buccal armature consisting of lips; to the right, the head of *Uncinaria duodenalis* (dorsal view), drawn to same scale, showing its greater size and the hook armature of the buccal border.

Below: to the left the caudal border of male *Uncinaria Americana* (dorsal view, dorsal lobe of bursa thrown forward), showing comparative size of the worm in relation to that of *U. duodenalis* partly divided dorsal lobe and suggestion of a ventral lobe, the dorsal ray divided nearly to base, each of its divisions having a bipartite tip, showing also the tip of tail within bursa, the long barbed sexual spicules, and the arrangement of the rays of the bursa; to the right, caudal bursa of male *U. duodenalis*, showing larger size of bursa, entire dorsal lobe, absence of ventral lobe, division of dorsal ray limited to distal third and tripartite tips of each division, with other features in genera similar to *U. Americana*. (Smith.)

Treatment.—1. **Prevention.**—Drinking water should be boiled, and the hands should be cleansed before eating. The stools should be received in water-tight closets and disinfected; careless defecation in mines has caused wide dissemination of the disease. Mines should be disinfected with the milk of lime.

Fig. 99.



Uncinaria duodenalis, with eggs. (After Blickhau.)

2. **Medication.**—*Thymol* is the most efficacious remedy. After a light diet, ʒ ss should be given, and this should be followed by an equal dose in two hours, and by a saline purge two hours afterward. As toxic symptoms (nervous excitation, or smoky urine) may develop, no solvents of thymol should be given, as alcohol, chloroform, ether or castor oil. This treatment should be repeated once a week, as long as ova or Charcot-Leyden crystals appear in the stools. The most severe cases do not respond to any treatment. *Male fern* is considered the best remedy by Tenholt, and other Westphalian writers.

V. Filaria.

Demarquai (1863) first found the embryos of the filaria; Wücherer (1866) found them in the urine, and Lewis (1872) in the blood.

Filaria Nocturna.—Its distribution is largely tropical and subtropical. It occurs in 10 to 50 per cent. of the inhabitants of the Samoan Islands, and in 32 per cent. of those of the Friendly Islands. In the United States it has been found in the South, and also in New York, Illinois and Pennsylvania. (*a*) *Infection of man* occurs by mosquito bites or possibly by water which the mosquito contaminates with its eggs. In the mosquito, the embryos change to the young adult form. (*b*) The *next stage* is the presence of *adult filaria in the lymph channels*, where they cause lymph stasis and lymphangitis. The female filaria measures 155 x 0.7 mm. and the male 83 x 0.4 mm. (*c*) The *embryos enter the blood current* from their birth place, the lymph channels; they are very numerous, measure ⅛ inch in length, and have the same diameter as a red blood cell. Under a low power lens their active movements are easily seen. They appear in the blood only at night, usually near midnight, and during the day retire to internal organs as the lungs but if the patient sleeps by day, they appear in the day time only.

Symptoms are absent in the majority of cases of human and animal infection, and are due to the parent worm, not to the embryos. (*a*) *Chyluria* or hæmatochyluria is due to plugging by the worm of the lymphatic vessels in the peritoneum, pelvis, vesical and perirenal tissues. Lymph stasis, ectasia and lymphangitis result, with intermittent rupture into some part of the urinary tract, and the passage of milky chylous urine, containing molecular albumin and fat. The amount of the urine varies; it may be normal, or increased. There is usually also some blood. If the blood clots, vesical disturbance results. The embryos are often found in the blood and sometimes in the urine; there is also a non-filarial chyluria (page 806). The affection may last for years without impairment of health. The very rare rupture into the peritoneum causes chylous ascites. (*b*) The *lymph scrotum* is due to lymphatic obstruction by the adult worm. The scrotum may weigh 50 to 200 pounds. Rupture into the tunica vaginalis causes chylocele. The labia may be similarly involved. The embryos are often missing from the blood, being apparently confined to the local involvement by the lymphangitic thickening, whence the difficulty in stating the relative percentage of parasitic and non-parasitic cases of lymph scrotum and elephantiasis. (*c*) *Elephantiasis* usually develops in the legs, scrotum (*v. s.*) and labia, less often in the arms, breasts or trunk. In forms (*b*) and (*c*), there occur intermittent exacerbations with fever and nervous disturbance, which end with critical sweats.

Other Forms of Filaria.—The exact status of the *filaria diurna* is not known; *f. perstans*, which causes pustular eruptions (craw-craw); *f. loa* (the adult stage of filaria perstans) which inhabits the conjunctiva; *f. labialis*; and *f. oris*, and *bronchialis*, are other forms.

Treatment.—(*a*) For *prevention*, mosquito bites and infected water should be avoided. (*b*) No parasiticide is known. (*c*) *Chyluria* is treated expectantly; a dry and fat-free diet is recommended but it is difficult to understand how it can influence lymph stasis. (*d*) *Lymph scrotum* and *elephantiasis* may necessitate castration, amputation or less radical measures to eradicate the obstructing parasite.

VI. Dracunculus (Filaria) Medinensis.

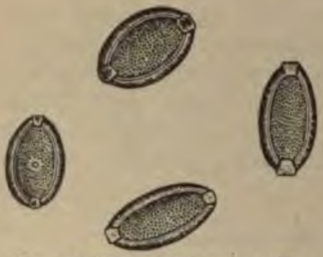
This is found on the West African coast, the Gold Coast, in Abyssinia and South Egypt; on the Caspian Sea and Persian Gulf; in the East Indies, parts of India, and South America. Guinea-worm disease (drachontiasis) has developed in a person who had always lived in Philadelphia. Man is infected by a small water flea (cyclops) which contaminates the water with the larvæ (embryos). These embryos, measuring 0.6 mm., enter the stomach, bowel and probably the mesentery, where they copulate; the male, of which little is known, is either discharged from the bowel or dies in the mesentery. The female is cylindrical in form, measures 50 to 80 x 2 mm., has a triangular mouth, eight papillæ and a blunt tail; its uterus occupies almost its entire length. The female which is usually solitary, wanders downward in the subcutaneous and intermuscular tissues to the thighs, legs and in 66 per cent. of cases reaches the feet where, near its head, vesicles, abscesses and ulcers develop. Its downward course occupies a year, during which it may be felt subcutaneously. When the skin breaks the parasite discharges her embryos and then leaves the body, to die.

Treatment.—Treatment by excision, or by 1 to 1,000 bichloride injections, seems most rational. If the worm is ruptured in efforts to extract it, high temperature, alarming nervous symptoms and sepsis are said to result from migration of the embryos which are liberated in the tissues.

VII. The Trichocephalus Dispar or Whip Worm.

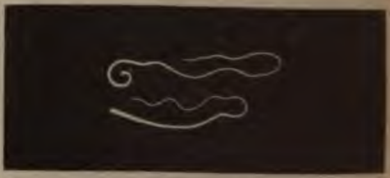
This is a nemotode, which is found most often in Southern Italy and France and but seldom in this country; one case occurred in the County Hospital service. It is also called trichuris trichura. It requires no intermediate host, enters with the food and drink, and is found in the cæcum and large gut. The male is slightly shorter than the female which is 4 to 5 cm. in length. The anterior portion of the worm is thin; the posterior two-fifths is thick, in the male round and in the female pointed. It bores into the mucosa and is nourished by the blood and not by the alimentary contents. The eggs measure 0.05 mm., are oval and are marked by a button-like projection. It causes few symptoms; occasionally there is severe abdominal pain. Three fatal cases are recorded. Male fern and thymol internally and benzene ʒi to Oij of water as an enema are recommended. (See figs. 100 and 101.)

Fig. 100.



Eggs of *Trichuris trichura:* ×275. (After Eichorst.)

Fig. 101.



Trichuris trichura, male and female: natural size. (Eichorst.)

VIII. The Strongyloides Intestinalis.

This includes a number of nematodes, as the anguillula intestinal or stercoralis, rhabdonema, etc. They are found in the endemic diarrhœa which occurs in Cochin China. Strong reports cases from Manila, and W. F. Thayer reported the first case in America. A few parasites cause no symptoms. Thymol is almost specific.

(C). DISEASES CAUSED BY TREMATODES (DISTOMIASIS).

1. *Bilharzia disease* (endemic hæmaturia) is due to the blood fluke (Cystosoma hæmatobium). Bilharz described the disease in 1851. It is an African affection; it has prevailed for many centuries in Egypt, where Bilharz considered that half of the lower classes were affected. It is equally frequent in Uganda. Importation has occurred into India, the Mediterranean Islands, and America. Only six cases have occurred in the United States (Gunn, 1906).

Children are more predisposed than adults to infection, which occurs through the drinking water or possibly through the skin. The embryos enter the stomach, penetrate the alimentary mucosa, and mature in the veins of the pelvis, spleen, mesentery, liver, kidney and bladder. The male worm measures 4 to 15 x 0.6 mm., the female 15 to 20 x 0.3 mm. The male is rolled up like a leaf, forming a groove in which the female lies during copulation. Many ova are found in the urine; they are oval, carry a spine, and contain a ciliated embryo. They die in the urine in a day but live for some time in water, where their life history is still imperfectly understood.

Hæmaturia is the most common, and often the only symptom; it may be macro- or microscopic and passes chiefly at the end of urination; its examination shows many ova and eosinophiles. Cystitis occurs in severe infection. Stones are not uncommon, particularly in cases in Canton, China. Suppuration may develop fistulæ. These clinical symptoms result from massive accumulation of ova in the submucosa, which leads to papillomatous hyperplasia, hemorrhage, suppuration, necrosis, salt deposits (calculi), and rarely to malignant degeneration. Similar changes in the rectum cause pile-like papillomata, dysenteric evacuations and tenesmus. As complications, pyelitis, prostatitis and changes in the uterus, seminal vesicles and renal pelvis, resembling those in the bladder, may develop. The eosinophiles are increased to 10 to 50 per cent.

Treatment.—Change of climate is advisable in order to avoid infection. The parasites usually die, though slowly, and as no local application or systemic remedies modify their life history, treatment must be expectant.

2. *Lung flukes* cause the *endemic hæmoptysis* which is observed chiefly in North China, Corea, Japan and Formosa. The parasite is named Distoma pulmonale (Paragonius Westermanni); it measures 10 x 6 mm. and is found largely in the lungs, but sometimes in the brain and skin. In young males a chronic cough develops with bloody or prune-juice sputum, in which are found blood, dark brown ova, eosinophile cells and Charcot-Leyden crystals. The hemorrhages, which are sometimes profuse, result from small infarcts of ova in the subpleural lung tissue. The course is usually favorable, though chronic, and is not influenced by treatment.

3. *Liver flukes* (Fasciola hepatica, Opisthorchis siensis) enter like the above forms through contaminated water. The fluke is found in the upper small intestine, and the bilary or pancreatic duct. It is found in Japan, and certain provinces show a mortality of 20 to 72 per cent. (Bälz and Inouye). The liver is enlarged and tender; icterus results from biliary stasis, for the parasites lie in the walls of the biliary duct and gall-bladder; diarrhœa develops, and the dejections contain ova and blood; anæmia, ascites, anasarca, adynamia, and emaciation develop and death invariably results. There is no treatment beyond the symptomatic support of the heart. Prophylaxis is valuable; avoidance of drinking or of swimming in canal water, and thorough cooking of fish and mussels, has stamped out the plague in one Japanese province (Inouye).

(D). DISEASES CAUSED BY PROTOZOA.

I. Malaria and Amœbic Dysentery (*v.* ACUTE INFECTIONS).

II. The Trypanosoma.—This is a protozoön found by Gruby (1843) in frogs, Döflein (1845) in rats, and later in many other animals, and in man (Nepvue, 1890–98). There are many species. It is leech-shaped; its body is granular, measures 13 to 25, by 2 to 4μ, and contains a nucleus and micronucleus (centrosome); on one side and attached to an "undulating membrane" is a solitary flagellum by which the parasite moves. In cool and moist hanging-drop perparations they may live for a month and a half. Novy and McNeal cultivated the T. Lewisi and Brucei on agar and defibrinated blood. Infection is carried by the tsetse-fly, flea, louse, mosquito and ticks. In man the two clinical forms (*v. i.*) of trypanosomiasis are conveyed by the tsetse-fly (Glossina palpalis) or possibly by bites from infected rats. Animals may be infected during coitus, by subcutaneous inoculation, or by the stomach.

1. *Trypanosoma fever* was first described by Nepveu. The atrium may be present as an inflamed bite, or the history of a bite may be obtained. Parasites in the *blood* may cause no symptoms; they occur in the plasma and not in the corpuscles; some anæmia is present and the large lymphocytes are increased in number (up to 20 per cent.), but the eosinophiles show no increase. *Fever* is irregular, subcontinuous or remittent; it does not react to quinine; it lasts from three days to three weeks. An erythematous eruption is almost constant. Œdema, particularly of the lower lids, weak and rapid pulse, prostration, splenic tumor and enlargement of the lymph glands which contain the parasite, usually occur. In *treatment*, arsenic is apparently most helpful, while quinine is without effect. Careful feeding and cardiac stimulation are indicated. The patient, as is the case in yellow fever, should be so screened that flies can neither convey fresh infection to him nor bite him, and thus infect others.

2. *Sleeping sickness* or *African lethargy*, which is endemic in Central Africa, is due to localization of the same trypanosoma parasite in the nervous system, causing a diffuse meningo-encephalomyelitis (Mott). Negroes are chiefly affected but Caucasians may contract the disease.

The incubation is long, and may possibly cover years. Incipient symptoms are those of the trypanosoma fever (*v.s.*) or less often there are preliminary psychical phenomena, as epileptiform convulsions, melancholia or mania. Somnolence develops, from which at first the patient can be aroused, but which later develops into profound lethargy. The parasite is found in the blood (in 92 per cent., Bruce) and in 60 per cent. of cases in the cerebrospinal fluid when withdrawn by lumbar puncture (Castellani), or in 100 per cent. (Bruce). Malnutrition, decubitus, and sometimes secondary infections develop, and after the convulsions deepen into coma, the patient dies. The course lasts from months to several years. Treatment is unavailing. More than a quarter of a million people died in Uganda in the last few years from this affection.

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